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About the Cover

The *Schizophrenia Bulletin* features art done by current and former mental hospital patients. The drawing that appears on the cover of this issue was sent to us by Mary E. Moller, MSW, Director, Sycamore Center, Petersburg, VA. Although wishing to remain anonymous, the artist has granted us permission to reproduce her art. Ms. Moller has provided us with the following description of the artist:

Our artist was known to be very shy and withdrawn with frequent periods of depression. Her mental and emotional problems began when she was a child. Although she had been hospitalized several times in the past, she has been out of the hospital for 13 years. She is presently a member of a clubhouse program and now enjoys being with people and having projects to work on such as the clubhouse newsletter. In addition, she motivates others to get involved in the activities of the program and is a great source of encouragement to other members. The artist assists other clubhouse members with their drawings. She feels art is an outlet and an important means of self-expression.

The artist herself has described the drawing as follows:

My drawing is entitled "Happiness" or "Energy." This drawing describes how I would like to feel. The bright vivid colors and exciting designs represent joy, exhilaration, and a great zest for life.

Anosognosia in Tardive Dyskinesia: "Tardive Dysmentia" or "Tardive Dementia"?

by Michael S. Myslobodsky

At Issue

Abstract

Wilson et al. (1983) called attention to the increasing incidence of affective symptomatology in schizophrenic patients on chronic neuroleptic medication. They designated the emerging syndrome as tardive dysmentia in order to emphasize its connection with the pathophysiology of tardive dyskinesia. The present contribution suggests that the emotional indifference or frank anosognosia of abnormal involuntary movements noted in the great majority (95 percent) of patients with tardive dyskinesia is another possible feature of tardive dysmentia. An attempt was made to explain anosognosia, and some difficulties that mar interpretation of data were pointed out. Anosognosia was proposed to represent a symptom related to some sort of cognitive disorder accompanying tardive dyskinesia, at least in the elderly. It was suggested that dementia may be associated with a predominant impairment of dopaminergic circuitry in the right cerebral hemisphere.

Several investigators have noted a significant inverse correlation between the severity of dyskinesia and depressive symptomatology (Vogel 1982; Oyewumi et al. 1983). Wilson et al. (1983) proposed that the insidious intrusion of affective symptomatology in chronic medicated schizophrenics is a behavioral equivalent of tardive dyskinesia, which they provocatively christened "tardive dysmentia."

While interviewing institutionalized patients with tardive dyskinesia (Holden, Sandler, and Myslobodsky 1984; Myslobodsky, Holden, and Sandler 1984), I was struck by the fact that most of them (95 percent) were oblivious to their faciolingual dyskinesia (see table 1 for characteristics of the study sample). I had imagined that at least patients who had Parkinson's signs concurrently with tardive dyskinesia would express concern when asked directly about their involuntary movements. However, they too appeared to be indifferent to their abnormality, even when it was very severe. This lack of awareness may explain the fact that even grotesque dyskinesia does not encourage social withdrawal and, as Owen (1979) has emphasized, is a distressing condition mostly for the patient's family. Our informal observations suggest that minimization or anosognosia adds another feature to the concept of tardive dysmentia inasmuch as our patients on chronic neuroleptic medication manifested other affective symptoms akin to those described in "dysmentia."

Matters are complicated by the fact that the major symptoms of tardive dysmentia outlined by Wilson et al. (1983)—for example, euphoria; emotional incontinence; impaired control of impulses; episodes of petulance, irritability, withdrawal, and apathy; loquaciousness; and verbal stereotypy—may accompany a moderate state of dementia, with or without concomitant abnormal involuntary movements (Blass 1982; Olkinuora 1982; Benson 1984). Moreover, the course of tardive dyskinesia is most likely to be severe in the elderly or in individuals with organic brain disorder (Chouinard et al. 1979). In computed tomography studies, Bartels and Themelis (1983) recently demonstrated structural abnormalities in the basal ganglia of

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Table 1. Characteristics of the study sample

Group	Age		Sex (M/F)	Mean TD score[1]	Mean Parkinson's score[2]	Mean current dose[3] (mg/day)	Years of hospitalization	
	Mean	Range					Mean	Range
Schizophrenics[4] (*n* = 23)	63.3	38–88	12/11	3.7	2.1	872	28.9	4–60
Manic-depressives (*n* = 7)	67.7	55–83	3/4	3.6	1.1	228	228	3–50
Epilepsy (*n* = 5)	60	49–70	2/3	3.6	0.8	360	30.6	15–45
OBS[5] (*n* = 14)	61	37–84	8/6	3.6	1.9	184	13.2	2–60

[1] Mean number of items on Rockland scale (Simpson et al. 1979).
[2] Mean number of items on Simpson and Angus (1970) scale.
[3] Chlorpromazine equivalent units.
[4] All subtypes.
[5] OBS = organic brain syndrome with psychotic symptoms, agitation, and assaultiveness.

patients with tardive dyskinesia. A large number of residents in homes for the elderly without a history of neuroleptic treatment reportedly exhibit abnormal involuntary movements (Bourgeois et al. 1980; Blowers 1981). Finally, there is an intriguing overlap between the neurotransmitter systems implicated in dementia (Gottfries 1982; Reisberg, Ferris, and Gershon 1983) and those assumed to malfunction in tardive dyskinesia (Klawans 1973). Similar to dementias, tardive dyskinesia may be considered a case of overmedication of the elderly (Cahn and Diesfeldt 1973), in which the fragile balance between neurotransmitter systems controlling locomotion and arousal is disrupted. Since aging itself confers a significant risk of both tardive dyskinesia and dementia, it is uncertain whether tardive dysmentia is a distinct syndrome or an unrecognized case of "tardive dementia."

Why patients with tardive dyskinesia should develop signs of emotional indifference is not immediately apparent. A number of investigators—most notably, Alexopoulos (1979), Rosen et al. (1982), Simpson (1975), and Smith et al. (1979)—have written on the subject, but no explanation has yet emerged. In fact, there may be more than a single explanation of this phenomenon. Some of the problems in interpreting anosognosia may also be relevant to an understanding of tardive dysmentia.

Wilson et al. (1983) have proposed that tardive dysmentia, similar to tardive dyskinesia (Klawans 1973), is associated with supersensitive dopamine receptors. If dopamine-mediated activity contributes significantly to reward and emotional behaviors, dopamine hyperactivity might be expected to promote an intrusion of affective symptomatology and enhance the "hedonic tone" of schizophrenia. The major limitation of this explanation is that the hyperproduction of either D_1 or D_2 receptors in tardive dyskinesia has yet to be supported experimentally (Hyttel, Christensen, and Arnt 1983). Also, the dopamine hypothesis of schizophrenia, which postulates an overactivity in the mesolimbic system, does not explain the profound anhedonia associated with this psychosis (Meehl 1962; Rado 1962).

Antiparkinson medication might be perceived as another factor of mood improvement. Unfortunately, in the study by Wilson et al. (1983), no data were presented relating to this variable. However, among patients with tardive dyskinesia examined by us, only a fraction received antiparkinson drugs (Holden, Sandler, and Myslobodsky 1984). Also, in a carefully controlled study, Mindham et al. (1972) failed to support claims of mood elevation in patients on amantadine and orphenadine. Even dysphoric reaction in patients with early akineto-rigid symptomatology were not alleviated by antiparkinson medication (Singh and Kay 1979).

Minimization and indifference might reflect a general disturbance of body image and self-identity in schizophrenia (Fisher and Cleveland 1958). However, tardive dyskinesia is not limited to schizophrenia. Some of our manic and epileptic patients showed most convincing examples of unawareness. Hence, the above variable would not fully account for this feature of tardive dyskinesia.

Minimization and denial of disease are common features in the epilepsies (De Angelis and Vizioli 1983). Schizophrenic patients often have epileptiform abnormalities of the electroencephalogram (EEG) (e.g., Stevens and Livermore 1982) that are not necessarily detectable by scalp electrodes. These seizure potentials may conceivably act as a "mild" chronic electroconvulsive therapy, postulated by De Angelis and Vizioli to improve mood in epileptic patients. While this possibility warrants a special investigation, it should be recalled that the so-called B-mitten pattern remains the only EEG correlate of tardive dyskinesia described so far (Wegner et al. 1979). However, the prevalence of B-mittens was shown to decline from age 40 upward, so that in patients older than 40 (i.e., in the majority of patients with tardive dyskinesia) no mittens are recorded. Also, it is of interest that B-mittens were originally believed to correlate with the Parkinson's syndrome (Winfield and Sparer 1954), which is seldom associated with epilepsy.

Another possibility reinforces the contention that tardive dyskinesia appears upon a background of neuroleptic-induced parkinsonism. It proposes that the symptoms in question are not idiosyncratic ones but originate along the akinesia/hyperkinesia dividing lines of parkinsonism. Parkinson's patients affected by tremor, which is considered a transitional symptom between parkinsonism and hyperkinesia (Chouinard et al. 1979), feel subjectively better than patients who have hypokinesia as a leading sign (Vogel 1982). Similarly, individuals treated with L-dopa are reportedly better able to tolerate severe dystonic and choreiform hyperkinesis than being hypokinetic (Duvoisin 1982; Vogel 1982). Duvoisin has commented that a patient's preference may represent a useful guide to dosage in cases of poor response to long-term L-dopa treatment. We (Holden and Myslobodsky, unpublished) elicited bitter complaints of stiffness and inability to walk from patients with tardive dyskinesia only within 2 weeks of cessation of anticholinergic medication. However, there was no appreciable difference in the degree of minimization among patients with various symptoms of parkinsonism, including rigidity, as far as faciolingual symptomatology was concerned.

Like Wilson et al. (1983), we were struck by the less socially disruptive character of psychotic patients with tardive dyskinesia. It is tempting to liken this perplexing phenomenon to the anosognosia of right-hemisphere-damaged patients. Although there is no indication of asymmetric brain involvement in tardive dyskinesia, such a possibility cannot be ruled out. Some investigators (Chouinard and Jones 1978; Kapit 1978) have hypothesized that the presumed hyperdopaminergia underlying schizophrenia is the result of an initial dopamine deficiency. The fact that extrapyramidal symptomatology occurs in some 40 percent of patients on neuroleptics (Marsden, Tarsy, and Baldessarini 1973) is consistent with this conjecture. Neuroleptic-induced parkinsonism is known to develop against the background of striatal dopamine depletion in a certain population of susceptible individuals (Rajput et al. 1982). It has been proposed that schizophrenia is associated with left hemisphere dysfunction (Flor-Henry 1969, 1983). In keeping with this concept, Waziri (1980) found that peripheral signs of tardive dyskinesia predominate on the right side of the body. While the faciolingual signs of tardive dyskinesia are mostly symmetrical (or, rather, their asymmetries are difficult to establish), certain indications of speech impairment accompany dyskinesia (Darley, Aronson, and Brown 1975). Also, symmetrical faciolingual signs may be tentatively attributed to an abnormality of lateralized mechanisms as the left hemisphere has been implicated in the bilateral control of the facial musculature (Kimura 1982).

The foregoing formed a basis for a neuropsychological study conducted in patients with tardive dyskinesia (Myslobodsky et al. 1985). This study encountered a problem at its inception. Only 18 (about 23 percent) of 79 patients drawn from two hospitals were free of serious cognitive deficits as measured by the Mini-Mental State Examination (Folstein, Folstein, and McHugh 1975). Even among these selected patients, several scored uncomfortably close to 25—the cutoff point between normality and cognitive impairment used by Folstein, Folstein, and McHugh (1975). In the majority of tests, the neuropsychological test performance of the 18 patients with tardive dyskinesia was not inferior to that of similarly screened psychotic controls matched for age, sex, and medication. In the Picture Recall Test, however, patients with tardive dyskinesia performed reliably worse than controls when the target stimuli were positioned in the right hemispatial field. Struve and Willner (1983) have reported impaired performance of patients with tardive dyskinesia on the Conceptual Level Analogy Test, a finding that is in keeping with left hemisphere involvement.

We have already alluded to the suggestive evidence of left hemisphere dysfunction in tardive dyskinesia offered by Waziri (1980). Unfortunately, his findings of right-sided dyskinesias were based on a small sample and were not replicated in a prevalence study (Myslobodsky, Holden, and Sandler 1984). Also, Jeste and Wyatt (1982) argue that unlike L-dopa-induced hyperkinesias, lateralized tardive dyskinesia is a rather uncommon phenomenon. Finally, P. Taylor (personal communication) has recently found asymmetrical extrapyramidal symptoms in 3 of 12 patients with tardive dyskinesia. In agreement with our own study (Myslobodsky, Holden, and Sandler 1984), all had tremor.

Hence, the hypothesis of selective left hemisphere deterioration in tardive dyskinesia does not find consistent support. Nevertheless, there are some heuristic benefits in entertaining the idea of a lateralized brain abnormality underlying emotional indifference. It is conceivable that cognitive deficits in tardive dyskinesia develop secondarily to right hemisphere abnormality.

This alternative conjecture is based on a number of findings suggesting that functions controlled by the right hemisphere deteriorate more noticeably with advancing years (e.g., Lapidot 1983). However, the contribution of the right hemisphere to the cognitive decline may be difficult to demonstrate experimentally because of the bilateral control of the right hemisphere over orienting reaction-electrodermal responsiveness (Heilman, Schwartz, and Watson 1978; Zoccolatti, Scabini, and Violani 1982; Mintz and Myslobodsky 1983), attention (Mesulam et al. 1976), touch exploration of extrapersonal space (Desmedt 1977), affect (Gainotti 1972, 1983), and some paralinguistic aspects of communication (Weintraub, Mesulum, and Kramer 1981). Even disprosody in patients with tardive dyskinesia (Darley, Aronson, and Brown 1975) may be associated with right hemisphere dysfunction. Hence, inconsistency of attention and incomplete monitoring of changes in various segments of extrapersonal space such as breakdowns of the body image and oscillations in cognitive and emotional reactivity (i.e., phenomena underlying various patterns of the dementias) may conceivably follow pharmacologically induced incapacity of the right hemisphere. It is intriguing that tardive dyskinesia assumes a notoriously severe course in patients with affective psychoses (e.g., Wolf, Ryan, and Mosnaim 1982) which are believed to be associated with right hemisphere abnormalities (Flor-Henry 1969, 1983). Therefore, the last and more prudent possibility relates anosognosia in patients with tardive dyskinesia to some form of cognitive decline associated with dementia disorder, apparently owing to some neuroleptic-induced deficiency within the dopaminergic circuitry.

This article cannot provide a confident answer to the question posed by its title. The problem of tardive dysmentia, as Wilson et al. have correctly pointed out, is that of the behavioral equivalents of tardive dyskinesia have yet to be established. Tardive dyskinesia can be encountered in children and adolescents (Gualtieri et al. 1984) who would hardly be expected to show signs of dementia. Whether they also manifest symptoms of tardive dysmentia or indifference and anosognosia remains to be elucidated.

References

Alexopoulos, G.S. Lack of complaints in schizophrenics with tardive dyskinesia. *Journal of Nervous and Mental Disease*, 167:125-127, 1979.

Bartels, M., and Themelis, J. Computerized tomography in tardive dyskinesia: Evidence of structural abnormalities in the basal ganglia system. *Archives of Psychiatry and Neurological Sciences*, 233:371-379, 1983.

Benson, D.F. Parkinsonian dementia: Cortical or subcortical? In: Hassler, R.G., and Christ, J.F., eds. *Advances in Neurology*. Vol. 40. New York: Raven Press, 1984. pp. 235-240.

Blass, J.P. Dementia. *Medical Clinics of North America*, 66:1143-1160, 1982.

Blowers, A.J. Epidemiology of tardive dyskinesia in the elderly. *Neuropharmacology*, 20:1339-1340, 1981.

Bourgeois, M.; Bouilh, P.; Tignol, J.; and Yesavage, J. Spontaneous dyskinesias vs. neuroleptic-induced dyskinesias in 270 elderly subjects. *Journal of Nervous and Mental Disease*, 168:177-178, 1980.

Cahn, L.A., and Diesfeld, H.F.A. The use of neuroleptics in the treatment of dementia. *Psychiatrie, Neurologie und Neurochirurgie*, 76:411-420, 1973.

Chouinard, G.; Annable, L.; Ross-Chouinard, A.; and Nestoros, J.N. Factors related to tardive dyskinesia. *American Journal of Psychiatry*, 136:79-83, 1979.

Chouinard, G., and Jones, B.D. Schizophrenia as dopamine-deficiency disease. *Lancet*, II:99-100, 1978.

Darley, F.; Aronson, A.; and Brown, J. *Motor Speech Disorders*. Philadelphia: W.B. Saunders, 1975.

De Angelis, G., and Vizioli, R. Epilepsy and depression. In:

Parsonage, M.; Grant, R.H.E.; Craig, A.G.; and Ward, A.A., eds. *Advances in Epileptology*. New York: Raven Press, 1983. pp. 203–206.

Desmedt, J.E. Active touch exploration of extrapersonal space elicits specific electrogenesis in the right cerebral hemisphere of intact right-handed man. *Proceedings of the National Academy of Sciences of the United States of America*, 74:4037–4040, 1977.

Duvoisin, R.C. Management of patients who fail to respond to levodopa therapy. *Clinical Neuropharmacology*, 5 (Suppl. 1):s13–s18, 1982.

Fisher, S., and Cleveland, S.E. *Body Image and Personality*. Princeton, NJ: Van Nostrand, 1958.

Flor-Henry, P. Psychosis and temporal lobe epilepsy—A controlled investigation. *Epilepsia*, 10:363–395, 1969.

Flor-Henry, P. Hemisyndrome of temporal lobe epilepsy: Review of evidence relating psychopathologial manifestations in epilepsy to right- and left-sided epilepsy. In: Myslobodsky, M., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 149–174.

Folstein, M.F.; Folstein, S.E.; and McHugh, P.R.. Mini-mental state: A practical method for grading the cognitive state of patients for clinicians. *Journal of Psychiatric Research*, 12:189–198, 1975.

Gainotti, G. Emotional behaviour and hemispheric side of lesion. *Cortex*, 8:41–55, 1972.

Gainotti, G. Laterality of affect: The emotional behavior of right- and left-brain-damaged patients. In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 175–192.

Gottfries, C.G. The metabolism of some neurotransmitters in aging and dementia disorders. *Gerontology*, 28(Suppl. 2):11–19, 1982.

Gualtieri, C.T.; Quade, D.; Hicks, R.E.; Mayo, J.P.; and Schroeder, S.R. Tardive dyskinesia and other clinical consequences of neuroleptic treatment in children and adolescents. *American Journal of Psychiatry*, 141:20–23, 1984.

Heilman, K.M.; Schwartz, H.D.; and Watson, R.T. Hypoarousal in patients with the neglect syndrome and emotional indifference. *Neurology*, 28:229–232, 1978.

Holden, T.; Sandler, R.; and Myslobodsky, M. Tardive dyskinesia: Prevalence and subtypes at Valkenberg Hospital. *South African Medical Journal*, 66:132–134, 1984.

Hyttel, J.; Christensen, A.V.; and Arnt, J. Neuroleptic classification: Implications for tardive dyskinesia. *Modern Problems of Pharmacopsychiatry*, 21:49–64, 1983.

Jeste, D.V., and Wyatt, R.J. *Understanding and Treating Tardive Dyskinesia*. New York: The Guilford Press, 1982.

Kapit, R.M. Schizophrenia and tardive dyskinesia: Is schizophrenia also a "denervation hypersensitivity"? *Medical Hypotheses*, 3:207–210, 1978.

Kimura, D. Left hemisphere control of oral and brachial movements and their relationship to communication. *Philosophical Transactions of Royal Society. Series B: Biological Sciences*, B298:135–149, 1982.

Klawans, H.D., Jr. The pharmacology of tardive dyskinesia. *American Journal of Psychiatry*, 130:82–86, 1973.

Lapidot, M.B. Is there hemi-aging? In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 193–212.

Marsden, C.D.; Tarsy, D.; and Baldessarini, R.J. Spontaneous and drug-induced movement disorders in psychotic patients. In: Benson, D.F., and Bloom, D., eds. *Psychiatric Aspects of Neurologic Disease*. New York: Grune & Stratton, 1973. pp. 219–266.

Meehl, P.E. Schizotaxia, schizotypy, and schizophrenia. *American Psychologist*, 17:827–838, 1962.

Mesulam, M.-M.; Waxman, S.G.; Geschwind, N.; and Sabia, T.D. Acute confusional states with right middle cerebral artery infarctions. *Journal of Neurology, Neurosurgery, and Psychiatry*, 39:84–89, 1976.

Mindham, R.H.S.; Gaind, R.; Antee, B.H.; and Rimmer, L. Comparison of amantadine, orphenadine, and placebo in the control of phenothiazine-induced parkinsonism. *Psychological Medicine*, 2:406–413, 1972.

Mintz, M., and Myslobodsky, M. Two types of hemisphere imbalance in hemi-parkinsonism coded by brain electrical activity and electrodermal activity. In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 213–238.

Myslobodsky, M.S.; Holden, T.; and Sandler, R. Asymmetry of abnormal involuntary movements: A prevalence study. *Biological Psychiatry*, 19:623–628, 1984.

Myslobodsky, M.S.; Tomer, R.; Holden, T.; Kempler, S.; and Sigal, M. Cognitive impairment in patient with tardive dyskinesia. *Journal of*

Nervous and Mental Disease, 173:156–160, 1985.

Okinuora, M. Organic brain syndromes from a psychiatric point of view: Diagnostic and nosologic aspects. *Acta Neurologica Scandinavica*, 66 (Suppl. 92):47–57, 1982.

Owen, R.T. Dyskinesias. *Drugs of Today*, 15:65–80, 1979.

Oyewumi, L.K.; Lapierre, Y.D.; Gray, R.; Batth, S.; and Gelfand, R. Abnormal involuntary movements in patients on long-acting neuroleptics. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 7:719–723, 1983.

Rado, S., ed. *Psychoanalysis of Behavior: Collected Papers*. New York: Grune & Stratton, 1962.

Rajput, A.H.; Rozdilsky, B.; Hornykiewicz, O.; Shennak, K.; Lee, T.; and Seeman, P. Reversible drug-induced parkinsonism: Clinicopathophysiological study of two cases. *Archives of Neurology*, 39:644–647, 1982.

Reisberg, B.; Ferris, S.H.; and Gershon, S. An overview of pharmacological treatment of cognitive decline in the aged. *American Journal of Psychiatry*, 138:539–600, 1983.

Rosen, A.M.; Mukherjee, S.; Olarte, S.; Varia, V.; and Cardenas, C. Perception of tardive dyskinesia in outpatients receiving maintenance neuroleptics. *American Journal of Psychiatry*, 139:372–373, 1982.

Simpson, G.M. CNS effects of neuroleptic agents. *Psychiatric Annals*, 5:53–60, 1975.

Simpson, G.M., and Angus, J.W.S. A rating scale for extrapyramidal side effects. *Acta Psychiatrica Scandinavica*, Suppl. 212:11–19, 1970.

Simpson, G.M.; Lee, J.H.; Zoubok, B.; and Gardos, G. A rating scale for tardive dyskinesia. *Psychopharmacology*, 64:171–179, 1979.

Singh, M.M., and Kay, S.R. Dysphoric response to neuroleptic treatment in schizophrenia: Its relationship to autonomic arousal and prognosis. *Biological Psychiatry*, 14:277–294, 1979.

Smith, J.M.; Kucharski, L.T.; Oswald, W.T.; and Waterman, L.J. A systemic investigation of tardive dyskinesia in inpatients. *American Journal of Psychiatry*, 136:918–922, 1979.

Stevens, J.R., and Livermore, A. Telemetered EEG in schizophrenia: Spectral analysis during abnormal behaviour episodes. *Journal of Neurology, Neurosurgery, and Psychiatry*, 45:385–395, 1982.

Struve, F.A., and Wilner, W.E. Cognitive dysfunction and tardive dyskinesia. *British Journal of Psychiatry*, 143:597–600, 1983.

Vogel, H-P. Symptoms of depression in Parkinson's disease. *Pharmacopsychiatria*, 6:192–196, 1982.

Waziri, R. Lateralization of neuroleptic-induced dyskinesia indicates pharmacologic asymmetry of the brain. *Psychopharmacology*, 68:51–53, 1980.

Wegner, J.T.; Struve, F.A.; Kantor, J.S.; and Kane, J.M. Relationship between the B-mitten EEG pattern and tardive dyskinesia: A pilot control study. *Archives of General Psychiatry*, 36:599–603, 1979.

Weintraub, S.; Mesulam, M.-M.; and Kramer, L. Disturbances in prosody: A right hemisphere contribution to language. *Archives of Neurology*, 38:742–744, 1981.

Wilson, I.C.; Garbutt, J.C.; Lanier, C.F.; Moylan, J.; Nelson, W.; and Prange, A.J., Jr. Is there tardive dysmentia? *Schizophrenia Bulletin*, 9:187–192, 1983.

Winfield, D.L., and Sparer, P.J. The electroencephalogram in paralysis agitans. *Diseases of the Nervous System*, 15:114–117, 1954.

Wolf, M.E.; Ryan, J.; and Mosnaim, A.D. Organicity and tardive dyskinesia. *Psychosomatics*, 23:475–480, 1982.

Zoccolatti, P.; Scabini, D.; and Violani, C. Electrodermal responses in patients with unilateral brain damage. *Journal of Clinical Neuropsychology*, 4:143–150, 1982.

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Past Issues: Comments and Responses

Measurement of Negative Symptoms

Abstract

The issue of the dimensionality of negative symptoms is addressed. In reference to data reported by Lewine, Fogg, and Meltzer (1983), it is suggested that the nonsignificant correlation between a SADS-C negative symptom scale and a NOSIE negative symptom scale, both independently developed using the Rasch model, is evidence for the multidimensionality of negative symptoms. Additional data supporting the multidimensionality of negative symptoms using confirmatory factor analysis is also discussed (Gibbons et al. 1985). A new theory for the structure of negative symptoms is needed, which specifies the number of dimensions, their classification, and their pattern of intercorrelation.

Lewine, Fogg, and Meltzer (1983) addressed the issue of measuring the severity of negative and positive symptoms of schizophrenia. They used the Rasch model, a probabilistic latent trait model, to develop scales for both negative and positive symptoms. Negative symptom items were derived from the Schedule for Affective Disorders and Schizophrenia-Change Version (SADS-C) (Endicott and Spitzer 1978) and the Nurses Observation Scale for Inpatient Evaluation (NOSIE) (Honigfeld, Gillis, and Klett 1966), whereas positive symptom items were derived only from the SADS-C.

In view of the fact that Lewine, Fogg, and Meltzer (1983) derived two scales of negative symptoms (one from the NOSIE and one from the SADS-C), the question must be raised—how should a negative symptoms score be assigned to a patient? Should the two scores be summed? This approach would be unwise since the two scales of negative symptoms are not correlated ($r = .12$ for men; $r = .21$ for women). Should one of the scores be chosen? If so, which is the "true" negative symptoms score? Or should both scores be retained? If so, there would no longer be a single dimension of negative symptoms but, rather, at least two dimensions of negative symptomatology—symptoms assessed by the SADS-C and those assessed by the NOSIE or, alternatively, the "molecular affective-cognitive signs" and the "molar behavioral signs" (p. 374).

The above questions point to a fundamental difficulty in Lewine, Fogg, and Meltzer's application of the Rasch model: the problem of dimensionality. When the Rasch model is applied to psychiatric data, the hypothesis tested is that *all* the symptoms of the syndrome (in this case, negative symptoms) lie along a single continuum and that *all* patients with the syndrome also lie along the same continuum. Important features of the Rasch model are item independence and sample independence. Item independence implies that any group of symptoms selected from the total set of symptoms that constitute the syndrome will assign the same scores (to within a linear transformation) to a particular sample of patients, as will any other group of symptoms of the same syndrome. Conversely, the severities (prevalence) assigned to a set of symptoms by a sample of patients from the specified population will be the same (to within a linear transformation) as the severities assigned by any other sample from the same population. The acceptance of the Rasch model has profound practical and theoretical implications: One may search for a functional mechanism that would generate unidimensional data, such as a single biological substrate or developmental pattern, and one may summarize any patient's negative symptoms with a single number, facilitating comparisons among patients.

Is there evidence that negative symptoms are in fact unidimensional? The goodness-of-fit statistics for the three scales reported by Lewine, Fogg, and Meltzer were not statistically significant, leading the authors to accept the hypothesis of unidimensionality for negative symptoms and for positive symptoms. However, the negative symptoms scale developed from the SADS-C was not correlated with the scale developed from the NOSIE. The lack of correlation is contrary to what one would expect from the item independence of the Rasch model, and indicates that there are *at least* two dimensions of negative symptoms. Lewine, Fogg, and Meltzer tacitly acknowledge this possibility, and hypothesize that the lack of correlation is due either to differences in the content of the SADS-C and the NOSIE (signs of psychopathology vs. personal hygiene and global activity) or to differences in the method of data collection. In any case, the data indicate that negative symptoms cannot be measured on a single dimension, and that the Rasch model should accordingly be rejected for the domain of negative symptoms as a whole.

Although Lewine, Fogg, and Meltzer concluded that their application of the Rasch model to develop reliable and stable unidimensional measures of negative and positive symptoms was successful, the position taken by the authors vis à vis the dimensionality of negative symptoms is unclear. Because the hypothesis of unidimensionality must be rejected, a new theory about the structure of negative symptoms would be useful. How many dimensions of negative symptoms are there? How may they be classified? What is their pattern of intercorrelation? A reasonable first hypothesis is that there are two dimensions of negative symptoms: the personal-hygiene-and-global-activity dimension, and the affective-cognitive dimension. With more subjects and more items, the affective-cognitive dimension might split into two dimensions, as might the personal-hygiene-and-global-activity dimension. And each method of observation might provide a separate kind of dimension. In fact, there may be many dimensions of negative symptoms.

A recently published study provides additional support for the multidimensionality of negative symptoms (Gibbons et al. 1985). In a sample of 416 newly admitted schizophrenic inpatients who participated in a multihospital collaborative study sponsored by the National Institute of Mental Health, confirmatory factor analysis was performed on negative symptoms assessed with the Inpatient Multidimensional Psychiatric Scale (IMPS). The symptoms included in this analysis were selected from the IMPS by Lewine (p. 391) based on two theoretical views of negative symptoms: Kraepelinian items (reflecting an absolute loss of function) and Bleulerian items (reflecting a loss of goal orientation or organization). Contrary to the expectations of Gibbons et al., *three* factors fit the data best, which they named "apathy," "retardation," and "Bleulerian." As we suggested earlier, more subjects and more items would likely yield additional negative symptoms dimensions. Future theories and research should further delineate the multidimensional structure of negative symptoms.

References

Endicott, J., and Spitzer, R.L. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35:837–844, 1978.

Gibbons, R.D.; Lewine, R.R.J.; Davis, J.M.; Schooler, N.R.; and Cole, J.O. An empirical test of a Kraepelinian vs. a Bleulerian view of negative symptoms. *Schizophrenia Bulletin*, 11:390–396, 1985.

Honigfeld, G.; Gillis, R.; and Klett, J. NOSIE-30: A treatment-sensitive ward behavior scale. *Psychological Reports*, 19:180–182, 1966.

Lewine, R.R.J.; Fogg, L.; and Meltzer, H.Y. Assessment of negative and positive symptoms in schizophrenia. *Schizophrenia Bulletin*, 9:368–376, 1983.

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Reply to Grau and Mueser

Abstract

Grau and Mueser (this issue) use the modest correlation between two negative symptom scales (Lewine, Fogg, and Meltzer 1983) to argue for the rejection of the Rasch model upon which the development of the scales was based. Their analysis fails, however, to take into account the conceptual and methodological intricacies of research assessment in clinical settings. Any sweeping generalization at this point is premature and risks the loss of heuristically valuable models.

Grau and Mueser (this issue) point to what at casual glance seems to be an internal inconsistency in the logic of our use of the Rasch model in the development of a negative symptoms rating scale (Lewine, Fogg, and Meltzer 1983). However, this conclusion rests on uncritical assumptions about clinical assessment in general, and the situational and methodological specificity of behavioral ratings in particular.

The authors argue that the failure to obtain a significant positive correlation between the two negative symptom scales—one based on the Schedule for Affective Disorders and Schizophrenia-Change Version (SADS-C) (Endicott and Spitzer 1978) and the other on the Nurses Observation Scale for Inpatient Evaluation (NOSIE) (Honigfeld, Gillis, and Klett 1966)—implies a multidimensionality of negative symptoms. Because this, in turn, contradicts the Rasch model, which assumes unidimensionality and item independence, the Rasch model must "be *rejected* for the domain of negative symptoms as a whole."

The Rasch model was developed within the context of educational assessment, with a focus on skills, attitudes, and traits. The data were composed of "*counts* of observable events . . ." (Wright and Masters 1982, p. 59) and, more specifically, the subjects' responses to a set of questions (or items). The question of interviewer inference or interaction between rater and phenomenon was never at issue; it is implicitly assumed that the subject's direct self-report is an accurate reflection of that subject's experience. In short, the Rasch model, like its numerous test construction counterparts, grew up in a "paper and pencil" environment, whose uncritical reliance on self-report is largely inappropriate to the assessment of psychopathology (Lewine and Sommers 1984). The Rasch model, in its assumption of unidimensionality and item independence, also implicitly assumes constancy of method and phenomenological domain.

Grau and Mueser make the mistake of assuming that the same conditions hold in clinical/applied research as hold in the paper and pencil domain. As a result, they overlook obvious obstacles that inhere in the application of the Rasch model to psychiatric data. There were three reasons for keeping the SADS-C and NOSIE data separate: they represent substantially different methods of data collection, represent vastly different domains of phenomena, and exhibit different psychometric properties. Only the first two are of relevance here. Method variance could easily and parsimoniously account for the modest correlation between the SADS-C and NOSIE negative symptom scales, a question best addressed in a multimethod-multitrait design (Campbell and Fiske 1959). The NOSIE is filled out by nurses, is based strictly on observation, and is longitudinal in nature, whereas the SADS-C is filled out by psychiatrists or psychologists (who differ from nurses in their training and theoretical orientation), derives largely from an interview, and is cross-sectional in nature. Furthermore, there may be differential effects due to making observations of the patient in a dyad away from the unit (SADS-C) or with other patients present on the unit (NOSIE); gender effects may also be pertinent as the NOSIEs collected in our original study were filled out by female staff and the SADS-C were filled out primarily by male staff.

More serious, however, is what appears to be a failure to appreciate the concept of multidomain assessment and the expected relationship between domains at different levels of description, especially when one of those domains can be considered a "final common pathway" and the other domain only one of several antecedent routes (admittedly, only alluded to in the original article). With its roots in 19th century physiology, the concept of "final common pathway" referred to the fact that a given sensation could arise from multiple external stimulus-nerve response combinations. Thus, a visual sensation can be caused by presentation of a light flash, pressure on the eyeball, or a blow to the head. The global experience, "I saw a flash of light," represents a final event having different possible antecedents. Although a correspondence between certain external events and perceptions exists, the correspondence is not perfect because the final phenomenon can also be caused by other environmental events.

Let us now assume that the phenomena measured by the NOSIE represent a final common pathway; these phenomena are quite global and nonspecific, and therefore are likely to have numerous antecedents. Furthermore, let us assume that the phenomena measured by the SADS-C represent one set of many possible antecedents; these phenomena are more narrowly defined and specific. When a nurse observes that a patient fails to speak, the patient's behavior could be preceded by a multitude of psychological events, some of which fall under the "negative symptoms" rubric (such as loss of drive or apathy) and some not (such as paranoid fear of people). In their NOSIE ratings, nurses do not make this sort of distinction, whereas such distinctions are made in the interview-based SADS-C (this reflects one of several problems in using rating scales for something other than their originally intended purpose; Sommers 1985). The phenomena included in the NOSIE are necessarily broader in nature than, and are *not* expected to show substantial overlap with, those of the SADS-C. Convergence of the different domains in a single construct of "negative symptoms" requires repeated demonstrations of modest correlations across measures, all of which are in the theoretically predicted direction (Knight 1983). It may well be that the NOSIE, as currently operationalized and used, is simply too broad in scope to serve as a basis for a negative symptom scale and, to this extent, Grau and Mueser's argument rests on our incorrect labeling of the NOSIE items as "negative symptoms."

There are far more critical and theoretically interesting critiques that can be made about the use of the Rasch model for psychiatric data, not the least of which is the assumption about hierarchical arrangement of the "items." That is, the arrangement of symptoms yielded by the Rasch model is presumed to reflect a dimension of difficulty such that loss of interest is more "difficult" (or severe or less frequent in the psychiatric domain) than is blunted affect among negative symptoms. That is, the model predicts that one is more likely to find blunted affect than loss of interest among schizophrenics manifesting the negative symptom syndrome. As indicated in the original article, there is certainly a strong precedent for the hierarchical organization of psychopathology, although whether such a conceptualization is appropriate to or whether the particular hierarchy of symptoms/signs holds for negative symptoms is an empirical issue. Another question begged in applying the Rasch model to psychiatric phenomena is the metric for scoring the individual items (or symptoms); it is assumed that a dichotomous (present vs. absent) decision adequately reflects the phenomena of interest. While the data from the original study support a simple dichotomous scoring, it is of considerable interest to know how general this is. A great deal of time and effort are expended in training interviewers to make continuous ratings when it may be just as useful and certainly easier to use dichotomous scoring.

We cannot, in concluding, fail to mention that Grau and Mueser point to what is in fact a significant question about the "negative symptoms" construct, even if they come upon it for the wrong reasons. Specifically, are negative symptoms unidimensional or multidimensional? There are several empirical studies suggesting that the symptoms and signs that generally fall under the "negative symptom" umbrella actually comprise independent factors. For example, Fleiss, Gurland, and Cooper (1971), using factor analysis, found separate factors for "retarded speech," "retarded movement," and "flat affect"; Everitt, Gourlay, and Kendall (1971) reported two factors conceptually related to negative symptoms, "schizoid personality-schizophrenic defect state" and "retardation-apathy." Studies such as these suggest that psychomotor retardation, thought disorder (including negative thought disorder), and apathy/withdrawal are independent dimensions rather than a single factor. In contrast, others have suggested that thought disorder, apathy, and retardation are all part of the same syndrome (Manschreck 1983). All of these studies restricted themselves to psychiatric signs and symptoms—in other words, analyzed data that were similar to those obtained from SADS-C and thus are not understood as reflecting differences in assessment domain. In light of the theoretical importance of the dimensionality issue in negative symptoms, a comprehensive synthesis of this literature would be most timely.

One hopes that multiple models will be thoughtfully used, and criticized, in an attempt to converge on some truth about "negative symptoms." Whether the conceptual and methodological transition of the Rasch model from testing data to psychiatric data can be made requires extensive study. To dismiss a potentially heuristic model, on the basis of a single study, especially without considering the methodological and conceptual complexities of adopting test models to clinical problems, is premature.

References

Campbell, D.P., and Fiske, D.W. Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin*, 56:81-105, 1959.

Endicott, J., and Spitzer, R.L. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35:837-844, 1978.

Everitt, B.; Gourlay, A.; and Kendell, R. An attempt at validation of traditional psychiatric syndromes by cluster analysis. *British Journal of Psychiatry*, 119:399-412, 1971.

Fleiss, J.; Gurland, B.; and Cooper, J. Some contributions to the measurement of psychopathology. *British Journal of Psychiatry*, 119:647-656, 1971.

Honigfeld, G.; Gillis, R.; and Klett, J. NOSIE-30: A treatment-sensitive ward behavior scale. *Psychological Reports*, 19:180-182, 1966.

Knight, R. Converging models of cognitive deficit in schizophrenia. *Nebraska Symposium on Motivation*, 31:93-156, 1983.

Lewine, R.R.J.; Fogg, L.; and Meltzer, H.Y. Assessment of negative and positive symptoms in schizophrenia. *Schizophrenia Bulletin*, 9:368-376, 1983.

Lewine, R.R.J., and Sommers, A.A. The clinical definition of negative symptoms. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies*. New York: Guilford Press, 1984. pp. 267-279.

Manschreck, T. Psychopathology of motor behavior in schizophrenia. In: Maher, B., ed. *Progress in Experimental Personality*. New York: Academic Press, 1983. pp. 53-99.

Sommers, A.A. "Negative symptoms": Conceptual and methodological problems. *Schizophrenia Bulletin*, 11:364-379, 1985.

Wright, B., and Masters, G. *Rating Scale Analysis: Rasch Measurement*. Chicago: MESA Press, 1982.

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Schizophrenia as a Semiotic Disorder

Abstract

Lanin-Kettering and Harrow (1985) argue the traditional position that schizophrenia is a thought disorder. Chaika and Lambe (1985) counter that it is a speech disorder at the syntactic-discursive level, and not a thought disorder. On the basis of state-of-the-art research in linguistics, it is suggested that the symptoms of schizophrenia are evidence of neither a thought disorder nor a syntactic-discursive disorder but a semiotic disorder. Semiotic structures have the form of saying something about something to someone and involve speech act, reference, pragmatics, and interpretation. Therefore, it appears that schizophrenic disorder is located in this structure.

In a recent article, Chaika and Lambe (1985) took us a step further in unraveling the nature of schizophrenia. Nevertheless, I find both the thesis of Chaika and Lambe and the counterthesis of Lanin-Kettering and Harrow (1985) inadequate for conceptualizing the precise nature of schizophrenia.

The two arguments may be summarized as follows. Lanin-Kettering and Harrow introduce four symptoms into evidence:
(1) problems in sorting tests;
(2) speech that is hard to follow, has poor topic maintanence, and intermingles personal material out of context to situation; (3) strange postures and gestures; and
(4) delusions. They infer that these symptoms are caused by "strange ideation" and that schizophrenia is a "thought disorder."

Chaika and Lambe introduce somewhat different evidence:
(1) chaining, glossomania, inappropriate rhyming, neologisms, and word salads; (2) errors in speech at the discursive and syntactic levels of organization; and (3) the inability to say what one intends to say, so that although one has a clear idea in mind, deviant speech is produced. From this evidence they infer that schizophrenia is a "speech disorder," a kind of aphasia at the discursive and syntactic level.

Based on the evidence presented by both sides, Chaika and Lambe are fully justified in concluding that schizophrenia is a speech disorder and not a thought disorder. The evidence presented by Chaika and Lambe clearly supports this conclusion. If we may rule out delusions as well as strange postures and gestures as definitive for schizophrenia—a ruling supported by the differential criteria of *DSM-III* and other state-of-the-art texts on schizophrenia—and we set aside the sorting test evidence as ambiguous in interpretation, then the evidence presented by Lanin-Kettering and Harrow is reduced to that of speech disorder symptoms. In effect, Lanin-Kettering and Harrow's evidence further supports Chaika and Lambe's conclusion.

Nevertheless, I must disagree with Chaika and Lambe's hypothesis that schizophrenia is some sort of disorder of discursive-syntactic structures. I believe the evidence points to a more refined conclusion; schizophrenia is not a discursive-syntactic disorder, but a *semiotic disorder.*

State-of-the-art research in linguistics can be seen to cover two broad domains. First, there is the domain of phonological (Jakobson), semantic (Saussure, Griemas), syntactic (Chomsky), and discursive (Griemas, Bremond) structures, which organize language as an instrument for use. We may call this the domain of linguistic composition.

Second, there is the domain of language-in-use. This domain covers types of speech acts (Austin, Searle, Buchler), the act of referring (Pierce, Husserl), the pragmatics of communicating (turn-taking, topic maintenance, speech style, etc.), and, finally, the act of understanding, especially, understanding conflicting interpretations of things (Levi-Strauss, Gadamer, Hirsch). Analogy, metaphor, and literality fall in this domain. I call this domain, after C.S. Peirce (1935-36), the "semiotic" domain.

We may assume that just as the domain of linguistic composition is neurologically based, so is that of semiotics (e.g., Paul and Cohen 1982).

The semiotic domain has the structure of saying something about something to someone. This formula encapsulates the aspects of speech act, reference, pragmatics, and interpretation.

It seems reasonable to conclude from the evidence that it is precisely this semiotic structure that is disordered in schizophrenia, or at least in one major type of schizophrenia.

Lanin-Kettering and Harrow's strongest piece of evidence—the one that pertains to speech problems—falls squarely under the domain of pragmatic semiotics. Chaika and Lambe's evidence, especially the symptom so definitive of schizophrenia, namely, the patient's awareness of producing deviant speech in spite of possessing clarity of mental intent, also points to a disordering of semiotic structure. If so, then errors in discursive and syntactic speech organization would be secondary to the primary semiotic disturbance.

Other symptoms taken to be differential for schizophrenia, such as thought insertion, thought broadcasting, thought withdrawal, and running commentary provide further examples of a disordering of the semiotic structure. Conversely, these symptoms do not reflect some sort of linguistic composition disorder; nor do they reflect some sort of "thought" disorder, if by "thought" one means, e.g., logic, reflection on intentionality, evaluation of social performance, etc.

In addition, other symptoms one encounters in conversing with persons experiencing schizophrenic episodes, such as confusions between metaphorical and literal use of language and inability to understand "what is being talked about," i.e., what the referent of the conversation is, also point to schizophrenia as a semiotic disorder.

In sum, I believe the evidence indicates that schizophrenia, or at least one major type of it, is neither a thought disorder nor a disorder of linguistic composition, but rather a semiotic disorder. I would urge that future research be directed at this refinement of Chaika and Lambe's hypothesis.

References

Chaika, E., and Lambe, R. The locus of dysfunction in schizophrenic speech. *Schizophrenia Bulletin*, 11:8-15, 1985.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorders. *Schizophrenia Bulletin*, 11:1-7, 1985.

Paul, R., and Cohen, D.J. Communication development and its disorders: A psycholinguistic perspective. *Schizophrenia Bulletin*, 8:279-293, 1982.

Peirce, C.S. *Collected Papers*. (8 vols.) Cambridge: Harvard University Press, 1935-36.

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Is Schizophrenia a Semiotic Disorder? Replies to Harrod

Abstract

While in agreement with Harrod's (1986) evaluation of the relevant data, Chaika and Lambe argue that several conclusions he has inferred from these data exceed what they warrant. In particular, Chaika and Lambe *do not* claim that schizophrenia *is* a language (or speech) disorder; nor do they claim that schizophrenic persons lack *cognitive* deficits; finally, in no way do they imply that language (or speech) data are the only appropriate data upon which to base inferences regarding the cognitions of schizophrenic persons.

We are in agreement with Harrod (1986) regarding his evaluation of the evidence presented by Lanin-Kettering and Harrow (1985) and by us (Chaika and Lambe 1985). However, he has made some unjustified inferences about our position. For example, he has missed the essential distinction that forms the logical foundation of our analysis:

> speech [is] an *overt* behavior, and thought [is] a cognitive process *inferred* on the basis of many different overt behaviors including speech. [Chaika and Lambe 1985, p. 8]

At no time did we (Chaika and Lambe 1985) claim that speech data are the only ones to consider in research on schizophrenia. One of us (Chaika 1982*a*) had in fact discussed the relationship of visual eye-tracking dysfunction to speech dysfunction in schizophrenia. The experiment on schizophrenic narration mentioned in Chaika and Lambe (1985) is a study of pragmatics, one which includes discussions of the relationship of intention to meaning (also, Chaika and Lambe, submitted for publication).

In our response to Lanin-Kettering and Harrow (1985), we concluded that some, but not all, schizophrenic speech shows a reliable pattern of deviation from normal speech at the different levels of language, including the pragmatic. Because these reliable patterns of structural deviation occur in schizophrenic speech, we stressed the need to take them into account before making any inferences about processes such as thought or cognition. We also cannot assume that the presence of a paralinguistic phenomenon such as facial expression, kinesics, or eye contact has any status as an inferential base in and of itself.

Note that we did *not* conclude that schizophrenia is *not* a thought disorder. Rather, we assumed the opposite:

> if one is using structural deviation as the basis of dividing schizophrenic patients into TD [thought disordered] vs. NTD [non-thought disordered], then one is in the peculiar position of claiming that some schizophrenic patients, those with structurally normal speech, are not thought disordered. Why, then, do we consider them schizophrenic? One would assume that all psychotic patients suffer an impairment in thinking, but structural deviations in speech . . . are not the proof of that, nor are they proof of the nature of the impairment. [Chaika and Lambe 1985, p. 9]

Moreover, at no time have we, or anyone else with whom we are acquainted, claimed that "schizophrenia is a speech disorder." As Chaika and Lambe (1985) state, many schizophrenics show no structurally deviant speech. Such speech, therefore, need not be a necessary component to a diagnosis of schizophrenia. However, when structurally deviant speech does occur, one must explain what makes it deviant, and what that might mean about schizophrenia. That is the position we take in our response to Lanin-Kettering and Harrow (1985).

Our position does not obviate the consideration of semiotics. Linguists and semioticians are not divided. Indeed, they share the same concerns, and many linguists are semioticians, and vice versa. One of us (Chaika 1981) has discussed pragmatics in the context of schizophrenia research, and *Language: The Social Mirror* (Chaika 1982*b*) contains chapters on kinesics, eye contact, facial expression, postures, utterance pairs, and discourse processes. All of these are of equal concern to semioticians, linguists, psychologists, anthropologists, and sociologists. There can be no hard line between the various disciplines concerned with human messages. The fact that we tackle one level of message giving does not mean that we feel that is the only one to be considered.

References

Chaika, E. How shall a discourse be understood? *Discourse Processes,* 4:71-88, 1981.

Chaika, E. A unified explanation for the diverse structural deviations reported for adult schizophrenics with disrupted speech. *Journal of Communication Disorders,* 15:167-189, 1982*a*.

Chaika, E. *Language: The Social Mirror.* Massachusetts: Newbury House, 1982*b*.

Chaika, E., and Lambe, R. The locus of dysfunction in schizophrenic speech. *Schizophrenia Bulletin,* 11:9-15, 1985.

Chaika, E., and Lambe, R. "Cohesion in Normal and Psychotic Narration." Submitted for publication.

Harrod, J.B. Schizophrenia as a semiotic disorder. *Schizophrenia Bulletin,* 12:12-13, 1986.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorders. *Schizophrenia Bulletin,* 11:1-7, 1985.

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Abstract

Harrod (1986) has proposed that schizophrenia is a "semiotic" disorder. We suggest that there is no evidence that a semiotic disorder is the core impairment underlying the disordered thinking and multiple types of strange and deficit behaviors shown by schizophrenics. Instead, data of ours and others link schizophrenics' strange verbal and nonverbal behavior, and their odd, unrealistic, delusional ideas, to their disordered thinking. All people use words and speech, but when people use words to express bizarre ideas and say strange things, it is not necessarily because of a word disorder, or a speech disorder, or a semiotic disorder. The semiotic domain does not address the level involved in the core deficit underlying the wide range of disturbances in schizophrenia.

Harrod (1986) has proposed that schizophrenia is a semiotic disorder, and has tried to discount our view that disordered thinking is an important factor in schizophrenia (Lanin-Kettering and Harrow 1985). In his position statement, Harrod argues that the best evidence points to schizophrenia as a deficit confined to language. In making his claim, Harrod tries to dismiss evidence about the prominence of strange schizophrenic ideas such as delusions by stating that delusions are not an important phenomenon in schizophrenia. He also has ignored a wide range of other data which support the thesis of disordered thinking as one of several important features of schizophrenia, and this evidence will be outlined later in the current discussion.

Harrod also discounts the view that language is a speech disorder as proposed by Chaika (1982) and Chaika and Lambe (1985). He argues that schizophrenic deficits extend beyond "discursive-syntactic structures" (Harrod 1986).

Harrod's View: Schizophrenia as a Semiotic Disorder

Harrod's alternative proposal is, as noted above, that schizophrenia is essentially a semiotic disorder, a disorder contained within the realm of communication but not limited to speech. Strictly speaking, the term "semiotic" means "semantic" or "of or pertaining to semantics"; semiology is defined as "the science that relates to sign language" as defined in the 1984 edition of Funk and Wagnalls' dictionary. The study of semiotics has, however, assumed a variety of forms, from the study of biological releasing mechanisms and animal sign systems ("zoo semiotics") to the study of medical semiotics (connecting signs and symptoms to an illness) and interpretation of semiotics in the context of current semantic theory (Eco 1976; Sebeok 1977). In introducing semiotics, Harrod makes specific reference to Charles Peirce, who offered a particularly influential theoretical framework for the study of semiotics (Peirce 1935-36). Peirce's contribution included a theory of signs (distinguishing indexical, iconic, and symbolic signs) and a triadic model of the relationship between sign, vehicle, and interpretant. According to Peirce's point of view (see, also, Morris 1938), the sign is that which acts as a sign; the vehicle is that which the sign refers to; and the interpretant is the effect of the sign on the interpreter.

In describing semiotics as "saying something about something to someone," Harrod (1986) closely approximates Morris' view that ". . . a sign refers to something for someone" (Morris 1938, p. 3). Harrod seems to intend "semiotics" to refer to a process that involves conveying meaning by signs or symbols with syntax, semantics, and pragmatics as cooperating mechanisms within that general process.

Thought Pathology as a Central Feature: The Evidence

Harrod's claim that schizophrenia is a semiotic disorder is not justified. To make that claim is to constrain schizophrenia to a disorder in the communication of meaning, a constraint not supported by available empirical evidence. Among the many problems with Harrod's position are that: (1) it ignores a considerable amount of evidence supporting the position that disordered thinking is an important factor in schizophrenia; (2) it ignores a range of other evidence about the nonverbal as well as verbal behavior of both schizophrenic and nonschizophrenic patients; (3) it ignores a wide range of evidence about impaired behavior in other areas for schizophrenic patients; and (4) there is no specific evidence offered by Harrod to support the position that the underlying disorder in schizophrenia is confined to semiotics.

In terms of the first point, the position of our research group and of others is that schizophrenia often involves a thought disorder and not just a disorder in speech or in the use and interpretation of symbolic communication. Our group and a large number of others have cited much empirical evidence indicating that many schizophrenic patients have a thought disorder (Chapman and Chapman 1973; Johnston and Holzman 1979; Harrow and Quinlan 1985). Much of this large array of evidence about disordered schizophrenic thinking would be hard to explain on the basis of speech or communication alone. In addition, viewing schizophrenic patients as having a thought disorder rather than a speech disorder or a semiotic disorder fits into a larger nomological net (Cronbach and Meehl 1950) that links their strange thinking to many other types of strange nonverbal behavior which they show. This approach leads to further productive inquiry, and is a more fruitful approach to advancing knowledge about schizophrenia (Lanin-Kettering and Harrow 1985).

As we have noted, Harrod chooses to ignore a large body of data about schizophrenic patients' strange ideas or delusions. It is hard to dispute the evidence that a very large percent of schizophrenic patients are delusional at the acute phase, and that even after the acute phase a surprisingly high percentage of schizophrenic patients show delusional ideas (Harrow, Carone, and Westermeyer 1985). In addition, there is evidence that *in schizophrenic patients* there is a strong relationship between the presence of delusional beliefs, which often involve *strange, unreal ideas* that are a function of their thinking, and the presence of disordered speech, which we also view as a function of their strange or disordered thinking (e.g., see Harrow, Silverstein, and Marengo 1983, for evidence about the relationship between these two aspects of thinking).

It is important to note that schizophrenic patients do not necessarily have any difficulty communicating their delusional ideas to others; the deficit is in the ideas, not in communicating the meaning of these ideas, i.e., not in the semiotic process. Given that disordered ideas can be clearly expressed, we argue that the deficit is in thought rather than in semiotics (or in speech), and that when disordered speech occurs, it is secondary to the more general thought disorder.

Other evidence that schizophrenic patients' strange behavior is not limited to their use of words, or to symbolic communication, but rather is best understood in the context of a more general tendency toward bizarre and idiosyncratic thinking and strange ideas, is seen in their strange nonverbal behavior, which can be found in both routine life situations and in formal test situations. With respect to routine life situations, one can cite such instances as the suspicious, seclusive behavior of some early and late schizophrenics, and the strange posturing of select others. In a very large number of empirical studies, hospitalized schizophrenics are given pathological ratings by nurses and other professionals on measures of strange behavior.

In addition to their strange behavior in real-life situations, schizophrenic patients behave strangely in controlled test situations which allow one to assess both bizarre nonverbal and verbal behavior. As evidence of this, Harrow and Quinlan (1985), Payne and Hewlett (1960), and many others have used the Object Sorting Test, with their data indicating strange sortings of objects by schizophrenic patients. These consistent findings of strange sortings by schizophrenic patients are often a consequence of their strange concepts and ideas, rather than a result of strange use of words. In general, schizophrenic patients are able to understand the use of symbols by others, and to communicate their strange ideas to others quite adequately.

Schizophrenia as a Semiotic Disorder: The Evidence

In addition to the wide body of evidence supporting the thesis of strange schizophrenic thinking, there are a number of other major problems with Harrod's (1986) conclusion that "it is precisely this semiotic structure that is disordered in schizophrenia." The problems with this conclusion are briefly outlined below:

- There is no evidence presented by Harrod, either from his own work or from that of others, that unique problems with the use of signs and symbols in communicating explain why schizophrenic patients say strange things, have strange and unrealistic ideas (delusions), or show deficits in a wide number of other nonverbal areas.
- A further problem is that Harrod has not described precisely what is meant when he uses the term semiotic. Because of this vague description, he adds to the confusion in the literature. As noted earlier, the term semiotics, in isolation, refers to communication generally; it is a process that involves a triadic relationship between sign, vehicle, and interpreter and one that subsumes semantics, syntax, and pragmatics. Harrod gives no suggestion or evidence about *where* in the semiotic process the "breakdown" may occur for schizophrenic patients, as a guide to further understanding, inquiry, or research in this area.

  The term "semiotic" has been used by a number of different theorists, and has been used so generally and in such a variety of ways that many diverse things can fit under it. Harrod's use of the term adds confusion instead of creating clarity.
- Harrod also has ignored evidence about other types of disordered cognition in schizophrenia which would be difficult to view as only a matter of a speech disorder or a semiotic disorder (e.g., problems in sustained attention, selective attention, and concreteness).
- In his thesis equating schizophrenia and speech disorder, Harrod also has ignored evidence about disordered functioning in other noncognitive areas by schizophrenic patients (e.g., slowed reaction times, psychomotor disturbances, and problems in social and work functioning). Thus, Harrod overlooks the fact that schizophrenia is not just a speech disorder, or even a disorder of thinking alone, but rather involves malfunctioning in a wide number of areas, some of which are only distantly related to semiotics.
- In claiming that schizophrenia is a semiotic disorder, Harrod ignores another large body of evidence showing that patients with other types of disorders (e.g., mania) also show speech and thought disorders (Andreasen and Powers 1974; Andreasen 1979; Harrow and Quinlan 1977; Marengo and Harrow 1985). Are these groups, then, also to be characterized as having a core deficit in "semiotics," despite the range of symptoms that differentiate them from schizophrenic patients? We and other researchers have been working toward finding a common denominator for their common symptoms; semiotics does not seem to offer a useful theoretical framework for guiding that enterprise.
- Harrod appears to believe that chaining is a major feature of schizophrenia. This ignores the evidence that chaining is a rare phenomenon in *modern-day* schizophrenia.
- Harrod also seems to believe that "the symptoms so definitive of schizophrenia, namely, the patient's awareness of producing deviant speech in spite of possessing clarity of mental intent, also points to a disorder of semiotic structure" (Harrod 1986). He cites as evidence for his statement Chaika and Lambe's similar *belief* in this area (Chaika and Lambe 1985). To the contrary, our research team has produced evidence that schizophrenics often are unaware of the strangeness of their disordered speech, thinking, and behavior at the time that they produce such aberrant behavior. Thus, in a series of experiments, our own research team has produced clear data indicating that in most cases schizophrenic and thought-disordered patients—and many other psychotic patients—are not aware, or lack perspective, that their speech and thinking are deviant (Harrow and Miller 1980). We ascribed this lack of awareness to a problem in *perspective*, involving an impairment in their ability to recognize in global fashion and in terms of broad consensual standards which behaviors of their own are inappropriate for a given context. We have proposed that impaired perspective—

which involves an impairment in executive control functions—is one of the key factors in understanding disordered schizophrenic thinking (Harrow and Quinlan 1985), and have collected data indicating that it also is a dimension of importance in schizophrenic delusions (Harrow, Rattenbury, and Stoll, in press).

The Locus of Schizophrenic Impairment

The fundamental question in relation to Harrod's thesis is not whether schizophrenic patients show disturbed speech, or whether their use of signs and symbols results in the communication of ideas which seem strange and bizarre to an observer. Rather, the fundamental question is whether or not a disturbance in that particular function is the core impairment in schizophrenia—i.e., is that the level at which research should be primarily directed, and under whose umbrella all other deficits should be understood?

We have discussed above data from a variety of different sources indicating that an approach based on the semiotic process is not the level of analysis most likely to be fruitful.

If we examine the most basic features used in verbal communication, we can see that everyone, including normals, mildly disturbed people, very disturbed people, and schizophrenic patients, uses signs and symbols to communicate. Such use of signs and symbols is a human characteristic, and the use of signs and symbols can be found at a somewhat less developed stage in lower forms of animal life. The fact that everyone uses signs and symbols in speech does not mean that when someone says strange things the underlying basis of the disorder rests at a semiotic level. The question is not whether signs and symbols are used in schizophrenic patients' disturbed verbalizations. The issue is whether a disturbance in that *particular* function, or in other functions, is the *locus* of the disorder. Thus, for those who characteristically say strange things, the question remains as to the level at which the impairment, deficit, or disturbance exists that underlies the odd verbalizations, thinking, and behavior of schizophrenic patients and of some other disorders as well.

If we put this last point another way, by supporting the thesis of disordered thinking as one of the central features of schizophrenia, we are suggesting that its centrality requires a different level of analysis than that for which Harrod as well as Chaika and Lambe argue. Taking thought versus speech as the specific exemplars, one could characterize the issue as a debate between the selection of a "domain-general" versus a "domain-specific" deficit as core to schizophrenia. Chaika argues that schizophrenic speech represents a transient aphasia, i.e., a disorder confined to the domain of speech. Harrod similarly confines schizophrenia as a disorder in the domain of language. We argue alternatively that the underlying disorder is a domain-general deficit, probably involving executive control mechanisms (i.e., regulation of thought processes, perspective and judgment, attentional mechanisms, etc.; see, e.g., Asarnow 1982; Harrow and Quinlan 1985). Because the deficit is domain general, specific domains such as language, object perception, and spatial orientation might be affected to the extent that they depend on those domain-general functions, but secondarily rather than primarily. To use a specific example, routine or automatic language processes such as lexical access do not require executive control; integration of verbal information over time does require executive control (paying attention, purposefully relating). (See Fodor, 1983, for a general discussion of these issues.) Consequently, individual features, including impaired speech and impairment in several individual areas of behavior, may be symptoms of a higher level, domain-general disorder, and may not themselves be representative of the basic disorder in schizophrenia.

In reviewing the evidence available to date, we can summarize by noting that both Chaika and Lambe (1985), and Harrod (1986) do not come to grips with a large body of data. This includes a number of lines of evidence indicating that schizophrenic patients frequently show disordered thinking, with the data based on both their verbal and their nonverbal behavior, and other data showing that their strange thinking (or positive thought disorder) is frequently related to strange and unrealistic ideas or to delusions and to other strange behavior. It also includes a wide range of data indicating that whether characterized at the level of speech or at the level of thought, this is only *one* of many disturbed characteristics of schizophrenia. Disordered schizophrenic behavior extends to a large number of other areas.

Overall, there is little or no evidence that a semiotic disorder is the *locus* of the core deficit for most schizophrenic patients. While there is little evidence that schizophrenia is, in that sense, a semiotic disorder, there is a large body of evidence indicating that schizophrenia is a multidimensional disorder, involving *several* major features, with severely disordered thinking being one of the major features.

References

Andreasen, N. Thought, language and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry,* 36:1325–1330, 1979.

Andreasen, N.C., and Powers, P.S. Overinclusive thinking in mania and schizophrenia. *British Journal of Psychiatry,* 125:452–456, 1974.

Asarnow, R. Schizophrenia. In: Tarter, R., ed. *The Child at Psychiatric Risk.* London: Oxford University Press, 1983. pp. 150–194.

Chaika, E. Thought disorder or speech disorder in schizophrenia? *Schizophrenia Bulletin,* 8:587–591, 1982.

Chaika, E., and Lambe, R. The locus of dysfunction in schizophrenic speech. *Schizophrenia Bulletin,* 11:8–15, 1985.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia.* New York: Appleton Century Crofts, 1973.

Cronbach, L.J., and Meehl, P.E. Construct validity in psychological tests. *Psychological Bulletin,* 32:281–302, 1956.

Eco, U. *A Theory of Semiotics.* Bloomington: Indiana University Press, 1976.

Fodor, J. *The Modularity of Mind.* London: The Oxford University Press, 1983.

Hardy, W. *Language, Thought, and Experience.* Baltimore: University Park Press, 1978.

Harrod, J.B. Schizophrenia as a semiotic disorder. *Schizophrenia Bulletin,* 12:12–13, 1986.

Harrow, M.; Carone, B.J.; and Westermeyer, J.F. The course of psychosis in early phases of schizophrenia. *American Journal of Psychiatry,* 142:702–707, 1985.

Harrow, M., and Miller, J.G. Schizophrenic thought disorders and impaired perspective. *Journal of Abnormal Psychology,* 89:717–727, 1980.

Harrow, M., and Quinlan, D.M. Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry,* 34:15–21, 1977.

Harrow, M., and Quinlan, D. *Disordered Thinking and Schizophrenic Psychopathology.* New York: Gardner Press, 1985.

Harrow, M.; Rattenbury, F.; and Stoll, F. Schizophrenic delusions: An analysis of their persistence, of related premorbid ideas, and of three major dimensions. In: Oltmanns, T.F., and Maher, B.A., eds. *Delusional Beliefs: Interdisciplinary Perspectives.* New York: John Wiley & Sons, Inc., in press.

Harrow, M.; Silverstein, M.L.; and Marengo, J. Disordered thinking: Does it identify nuclear schizophrenia? *Archives of General Psychiatry,* 40:765–771, 1983.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking.* San Francisco: Jossey-Bass, 1979.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorders. *Schizophrenia Bulletin,* 11:1–7, 1985.

Marengo, J., and Harrow, M. Thought disorder: A function of schizophrenia, mania or psychosis? *Journal of Nervous and Mental Disease,* 173:35–41, 1985.

Morris, C.W. Foundation of the theory of signs. In: Neurath, O., ed. *International Encyclopedia of Unified Science.* Vol. I. Chicago: University of Chicago Press, 1938. pp. 1–59.

Payne, R.W., and Hewlett, J.H.G. Thought disorder in psychotic patients. In: Eysenck, H.J., ed. *Experiments in Personality.* London: Routledge and Kegan Paul, 1960. pp. 3–104.

Peirce, C.S. *Collected Papers.* (8 vols.) Cambridge: Harvard University Press, 1935–36.

Sebeok, T. Zoo semiotic components of human communication. In: Sebeok, T., ed. *How Animals Communicate.* Bloomington: Indiana University Press, 1977. pp. 1055–1077.

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Psychopharmacology of Schizophrenia: A European View

by Nenad Bohaček

In 1980 the Schizophrenia Bulletin *(Vol. 6, No. 4) published a series of articles describing research on schizophrenia in the North European countries, Switzerland, and the United Kingdom. I wrote to the editor suggesting that it would be useful to add other articles so as to present a more comprehensive picture of the European contribution to knowledge about schizophrenia. The editor of the* Bulletin *accepted this suggestion and invited me to guest-edit these contributions. The present issue is thus complementary to the articles published earlier and contains reviews produced by authors from countries that were not included and on topics not covered in the 1980 issue—for example, epidemiology of schizophrenia and schizophrenia in children.*

The two issues are complementary but do not cover all the contributions that European psychiatry has made to the solution of problems related to schizophrenia. It is therefore to be hoped that there will be further issues with contributions from European psychiatry and perhaps from psychiatry in other lands. The problem of schizophrenia is of tremendous proportions, and international and national collaboration in research and in exchange of information is a most promising avenue to its solution.

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Abstract

This article describes how psychopharmacological therapy is applied in Yugoslavia and other countries in Central Europe. The first part of the article summarizes experience and customary use of psychopharmacological substances in relation to clinical syndromes, course of disease, and dominant symptoms. In the second part of the article, the experience obtained in treatment with combinations of drugs and in prolonged pharmacotherapy of schizophrenia is described. Finally, the last section of the article addresses the social implications of psychopharmacological treatment and presents some of the problems and disadvantages that accompany this form of treatment.

This article is neither a theoretical treatise on psychopharmacotherapy nor an attempt to summarize psychopharmacological research in Europe. It describes Yugoslav practice and experience in treating people who suffer from the various forms of schizophrenia, and is thus not a comprehensive description of the practices of European psychiatrists. To aim at such a description would be a vain effort. There are at least twice as many schools of psychiatry as there are countries in Europe; and few of the practicing psychiatrists remain within their school's rules during their working life.

Even Yugoslav practice and teaching has changed considerably over the years. It will undoubtedly continue changing with new discoveries, and more experience. In spite of these limitations, I believe that what is described here would be accepted as the usual practice of treatment of schizophrenia in Europe. This belief is based on contacts with

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colleagues and observations of psychiatrists in different countries of Europe. It is not based on a study of published work for the simple reason that there is no consensus description of the most commendable treatment of schizophrenia or, for that matter, much more than textbook instructions which are usually given in fairly general terms. There is little doubt that much of the healing art and practice in Europe is still transmitted in apprenticeship instead of through written instruction.

Psychopharmacological treatment has assumed the central place in the treatment of schizophrenia in all of the European countries (Shepherd, Lader, and Rodnight 1968). Psychotherapy and social measures are recognized as important and used in conjunction with medication. There are clear variations in the social and psychotherapeutic methods employed, as well as in the degree of importance attached to them.

Medications used in the psychopharmacotherapy of schizophrenia are usually divided into two major groups (Angst 1969; Benkert and Hippius 1974; Heinrich 1976): The first are basic (or nonspecific) neuroleptics. They are given in high doses, and have a global influence on the majority of symptoms seen in schizophrenia, including agitation, insomnia, restlessness, hallucinations, and delusions. They tend to have fewer extrapyramidal side effects and a stronger sedative action than drugs in the second group. The second group comprises drugs that are given in relatively small doses and appear to be more *specific* in their action.

The following characteristics of schizophrenia are generally considered in decisions about pharmacotherapy:

- Clinical form of schizophrenia (simple, hebephrenic, catatonic, or paranoid).
- Course of disease and phase in which treatment is prescribed (acute or chronic).
- Dominant symptoms (agitation, stupor, apathy, or abulia).
- Balance between productive (positive) and nonproductive (negative) symptoms.

Psychopharmacotherapy in Relation to Illness Characteristics

Clinical Form.[1] In simple and hebephrenic schizophrenia, basic neuroleptics are used first, and specific neuroleptics may not be used at all. If the latter are used, they will be administered after treatment with basic neuroleptics has been tried for some time. In catatonic excitement, both basic and specific drugs are used, whereas in catatonic stupor, specific drugs are prescribed. In paranoid, hallucinatory forms of schizophrenia, treatment begins immediately with specific drugs; later, and only occasionally, some of the nonspecific symptoms (such as insomnia) will be treated by basic drugs.

The principle of using as few drugs as possible is observed in most settings. In addition to neuroleptic treatment, a patient is usually also given antiparkinsonian drugs and sometimes antidepressants. As a rule, these combinations are used with less reluctance than the combination of several neuroleptics, although there are some clinics in which a large number of neuroleptics are used at the same time.

Course of Illness. The acuteness of the disease plays a major role in decisions concerning drug type and dosage. In very acute cases, basic neuroleptics such as chlorpromazine, levomepromazine, and promazine (reported to be well tolerated in the elderly) are prescribed. Shortly thereafter, specific neuroleptics (such as fluphenazine, trifluoperazine, thioproperazine, clozapine, and haloperidol) are added depending on the clinical syndrome that dominates the picture. In acute cases, the doses are usually high—as much as 1,200 mg of chlorpromazine, 600 mg of levomepromazine, or 600 mg (or even more) of promazine. Fluphenazine, trifluoperazine, and thioproperazine are given in doses ⩾ 30–60 mg/day. In chronic forms of schizophrenia (and for the maintenance of remission), thioridazine and pimozide, as well as depot preparations (see below), are used. Doses of thioridazine range from 200–600 mg/day; doses of pimotide, 4–12 mg/day.

Dominant Symptoms. A patient with a dominant picture of psychomotor excitement is given basic neuroleptics, whereas in a patient with psychomotor stupor or retardation, specific neuroleptics are preferred. In a patient with paranoid or paranoid/hallucinatory symptomatology, specific neuroleptics are used from the beginning. In the instance of apathic or apathic/abulic syndromes, most of the basic neuroleptics have little effect. Specific neuroleptics do seem to have some effect in such cases and are therefore preferred—for example, pipothiazine, thioproperazine, and trifluoperazine. Clozapine has also been reported to have a good effect in the treatment of this syndrome.

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[1] In describing clinical syndromes and assessing change of the patient's condition in the course of treatment, a variety of instruments have been used. The Present State Examination (World Health Organization 1973, 1979) and the AMP System (Association for Methodology and Documentation in Psychiatry) (Angst et al. 1969; Bobon 1978; Bohaček 1978) are examples of some of the instruments used in a number of countries.

Presence of Productive Symptoms. The balance of productive (positive) and nonproductive (negative) symptoms of schizophrenia is also a factor in decisions concerning the type and dosage of neuroleptics to be prescribed. Most neuroleptics are efficacious in treating positive symptoms (such as hallucinations or excitement), but their effects in treating negative symptoms (such as apathia-abulia) are far less dramatic. There have been attempts to use psychostimulants (amphetamines or sidnocarb) (Peršić et al. 1972) and even hallucinogens (LSD-25 or Deanol) (Fouks et al. 1966) to deal with negative symptoms. It is hoped that such agents will precipitate positive symptoms, which will be more amenable to standard treatment with neuroleptics.

Drug Combinations

Occasionally a single medication can be sufficient to achieve a full therapeutic effect. In most instances, however, psychiatrists prescribe several medications simultaneously. For example, neuroleptics are used to treat the main symptoms, hypnotics to treat subsidiary symptoms, antiparkinsonian drugs to correct side effects, and so on.

Even in dealing with the main symptoms of schizophrenia, the use of only one neuroleptic is more the exception than the rule. Basic neuroleptics such as levomepromazine are often combined with a specific "target" neuroleptic such as fluphenazine even though it is widely recognized that such combinations can potentiate side effects (e.g., extrapyramidal or hypotensive effects or transient delirious states). Occasionally a combination of neuroleptics with drugs such as diazepam is prescribed. The latter is usually given in high doses. Accessory symptoms (such as insomnia) sometimes warrant additional therapy with hypnotic drugs or levomepromazine. Benzodiazepines (e.g., nitrazepam and flurazepam) are also added in these cases (although this type of combination has been shown to cause tiredness and slowness) because of possible interactions of barbiturates with neuroleptics (e.g., the induction of microsomal liver enzymes, which in turn can lead to a decrease of the effectiveness or elimination of effects of psychopharmacological drugs). Combined treatment with several neuroleptics is also sometimes used to avoid side effects.

Management of Side Effects

Early extrapyramidal side effects (e.g., tremor, rigidity, akathisia, and dyskinesia) are treated with antiparkinsonian drugs such as biperidine, trihexyphenidyl, and ethybenztropine. Their administration is often stopped after a short time without a reappearance of extrapyramidal side effects, which may have been intense earlier in treatment.

In general, however, it is no longer considered necessary to continue giving antiparkinsonian drugs indefinitely, and they are not universally used to prevent extrapyramidal side effects. The earlier practice of providing antiparkinsonian drugs to all patients treated with neuroleptics was abandoned when it became clear that no more than half the patients treated with neuroleptics would experience extrapyramidal side effects, and when it was reported that antiparkinsonian drugs increased the risk of tardive dyskinesia (Gerlach 1979). There is also a possibility that combined treatment with neuroleptics and antiparkinsonian drugs decreases the concentration of neuroleptics in plasma and thus decreases their antipsychotic activity. Moreover, anticholinergic drugs (thus, also antiparkinsonian drugs), when combined with a phenothiazine, can lead to a disturbance of thermoregulatory centers and result in hyperpyrexia. More recently, reports of the serious nature of anticholinergic effects of antiparkinsonian drugs (e.g., dryness of mouth, disturbances of visual accommodation, and micturition) and the fact that the use of some anticholinergic drugs (such as trihexyphenidyl) can lead to dependence further discouraged the routine use of antiparkinsonian drugs as preventive medicine (Vinar 1973; Cattabeni et al. 1980).

The most serious and treatment-refractory example of late extrapyramidal side effects—tardive dyskinesia—has been reported in all European countries. In hospitalized patients, the reported incidence of tardive dyskinesia varies between 0.5 and 40 percent of all patients treated; most reports give a figure of about 15 percent. In chronic patients, the reported range (5 to 20 percent) is smaller. The reasons for these differences in the reported incidence of tardive dyskinesia are unclear and insufficiently investigated. Most reports state that dyskinesia will occur after long-term treatment with neuroleptic drugs (usually after 2 to 5 years), but cases occurring after 6 months of therapy have been described.

A variety of treatments have been tried but without much success. Since there is no effective therapy for tardive dyskinesia, attempts are being made to prevent its occurrence by following these principles: (1) Every attempt should be made to achieve and maintain optimal effects with the smallest possible dose. (2) Very high doses should be avoided at all times. (3) Wherever possible, treatment with antiparkinsonian (anticholinergic) drugs should be avoided; if these drugs are considered necessary, their use should be interrupted at regular intervals. (4) "Drug holidays" should be employed when the patient's condition permits. (5) An intensive effort should be made to train practitioners to recognize tardive dyskinesia in its incipient stages.

Various psychological side effects of neuroleptic treatment have been described and linked to the dose of neuroleptics, combination of drugs used, and the premorbid personality structure and age of the patient. One such effect which requires immediate cessation of all treatment is the transient delirious state that is most often seen when neuroleptics are used together with antidepressant and/or antiparkinsonian drugs. Another side effect described in protracted treatment with neuroleptics is the "depressive shift": the occurrence of a depressive syndrome in the course of treatment with neuroleptic drugs, usually after the acute symptoms (especially in paranoid/hallucinatory forms of schizophrenia) have disappeared (Bohaček 1965; Müller 1981). Such a depressive shift (or postpsychotic depression) has been found in patients treated with all types of neuroleptics, from phenothiazines to butyrophenones. It can be seen in 15–20 percent of all those treated. If left unattended, this depressive syndrome can lead to suicide and attempted suicide. The condition responds well to tricyclic and tetracyclic antidepressants (e.g., imipramine, amitriptyline, and maprotiline), which can be given in combination with neuroleptic drugs. Some authors have argued that the syndrome of postpsychotic depression does not exist. If a depressive syndrome occurs in the course of treating schizophrenia, these authors believe that a mixed state in which schizophrenic and depressive syndromes were present must have existed before treatment began. Others believe that the depressive shift is, in fact, a syndrome of exhaustion that occurs after remission from schizophrenia (der "post remissive Erschoepfungszustand"; Heinrich 1967).

When physical disease occurs in patients with schizophrenia, it is generally agreed that it is essential to provide additional therapy for the concurrent diseases while maintaining neuroleptic treatment. Such combined therapy can lead, however, to untoward interaction between drugs and side effects. Among these, the following are the most frequently reported: (1) The combination of oral antidiabetic drugs (of sulfonyleurea type) with neuroleptics of the phenothiazine type can decrease the effectiveness of phenothiazines. (2) Diuretics (thiazide and other saluretics), when given with phenothiazines, can potentiate hypotension. (3) Antibiotics such as cephalosporin, chloramphenicol, penicillin G, and tetracyclines can be inactivated or have their effects diminished. (4) Alcohol can potentiate the effect of chlorpromazine. (5) Neuroleptics increase the action of sedatives, alcohol, narcotics, and β-blockers, and decrease the effectiveness of anticonvulsants.

Duration of Neuroleptic Therapy

Neuroleptics are generally seen as a symptomatic treatment. To be effective, neuroleptic treatment must coincide with the presence of manifest symptoms of the disease: in this sense, by pharmacological parallel, neuroleptic treatment could be called "psychostatic."

Up to 80 percent of schizophrenic psychoses in our experience follow a chronic course. In these patients permanent pharmacotherapy is necessary. Because it is impossible to predict with certainty whether recovery from an acute episode of schizophrenia will be followed by relapses, and whether schizophrenia will follow a chronic course, in our practice pharmacotherapy is continued for approximately 1 year after the first attack. At that point, if the patient's condition permits, neuroleptic treatment is stopped. If symptoms of the initial episode are still present, treatment is continued. If a patient has a relapse (i.e., a second episode of the disease), treatment is continued for 3 years from the time of the second episode. If a third attack occurs, it is generally considered that pharmacotherapy will have to be lifelong. These guidelines provide a general framework within which adjustments for the individual patient are necessary (Palmović and Bohaček 1970).

Maintenance Therapy

After recovery from the acute phase of schizophrenia has occurred, drug dosages are gradually decreased to a minimum necessary to maintain an asymptomatic state. This decreased dose will be $\frac{1}{4}$, $\frac{1}{5}$, or even $\frac{1}{10}$ of the dose that was necessary during the acute phase.

Prolonged pharmacotherapy once required patients to take the neuroleptic in three or four daily doses—a difficult regimen to follow. Today, however, a variety of depot neuroleptics is used: in the group of tricyclic phenothiazines, fluphenazine enanthate and decanoate, perphenazine enanthate, flupenthixol decanoate, pipotiazine undecilatester, and pipotiazine palmitinester (which can maintain the blood levels constant from 7 to 28 days); among butyrophenones, haloperidol decanoate (supposed to maintain the therapeutic action of haloperidol for a period of approximately 4 weeks); among diphenylbutyrylpiperidenes, pimozide (taken once every day or every 2 days), fluspiriline (once weekly), and penfluridol (once weekly when taken orally) (Doongaji et al. 1981).

There is general agreement about the advantages of treatment with depot preparations: they are easy to apply, there is a good local and general tolerance, and the patient feels less marked as a mentally disordered person when he can receive treatment in a general health facility. There are also numerous reports that this form of treatment leads to a better relationship and transference between the patient and the doctor, greater possibilities for the use of other treatment methods (such as psychotherapy and sociotherapy), fewer side effects (in particular, extrapyramidal side effects), and a reduced danger of suicide, which was a particular risk in countries where the scarcity of services made it necessary to prescribe medication in sufficient quantities to last a month and to give it to the patient in a health facility.

Social Implications of Psychopharmacological Treatment

Psychopharmacotherapy is undoubtedly an effective treatment for schizophrenia. It by far surpasses the effectiveness of previous biological treatments and poses considerably fewer risks. The improved treatment of schizophrenia has had numerous social consequences, ranging from changes in attitudes to changes in legislation and organization of health services. Reports from various parts of Europe suggest that the attitudes of the patient, his family, lay personnel, and professionals toward the disease and its treatment are significantly more positive. Confidence in the efficacy of pharmacotherapy is constantly increasing.

The main social implication of the introduction of pharmacotherapy has been the possibility of outpatient treatment of a large number of schizophrenic patients who previously would have been institutionalized.

Although empirical observations were very important in establishing the efficacy of psychopharmacotherapy—the clinician's eye was the instrument that discovered the antipsychotic and antidepressive action of pharmacological drugs—psychopharmacological treatment led to the development of objective measurement techniques in psychiatry. These methodological advances have contributed to the scientific evidence about the value of different pharmacological drugs, so that results no longer rest only on clinical impressions. They have also made a contribution to the technology of assessment of complex human behavior. The pharmacotherapy of schizophrenic psychoses has also made it feasible to apply social and psychological therapeutic measures and to provide help for the schizophrenic and his family (Bohaček 1973).

There is much more agreement among psychiatric schools (regardless of their social, economic, political, and other differences) about principles of pharmacotherapy than there is similarity in the provision of treatment in different European countries. This is so for a variety of reasons. Pharmacotherapy, for example, is dependent on the social system, the philosophy of health care, and the organization of health services in general. In some European countries, health care is free and comprehensive; in others, health care is only free in part; and in others, much depends on the capacity of the patient and his family to pay the often exorbitant fees.

Conclusion

Much hope, and even more money and other resources are being invested in the search for new neuroleptics that will (1) be more effective and therefore applicable in smaller doses; (2) have an even more prolonged effect; (3) be more effective in dealing with nonproductive symptoms; and (4) have few or no side effects.

A question that psychopharmacology must resolve in the process of searching for new drugs is that of specificity of psychopharmacological substances. True specificity would mean that drugs used for the treatment of depression and schizophrenia were, in fact, specific for these diseases—which cannot today be affirmed for any one of the substances used.

In addition to these improvements in the psychopharmacological arsenal, most European countries are trying to improve the organization of services and other components of care. In this process the potential of different modes of treatment—with psychopharmacological, social, and biological means—has to be reexamined, and ways to ensure the provision of treatment that will satisfy the patient, his family, and the community must be found.

References

Angst, J. *Die somatische Therapie der Schizophrenie.* Stuttgart: Georg Thieme, 1969.

Angst, J.; Battegay, R.; Bente, D.; Berger, P.; Broeren, W.; Cornu, F.; Dick, P.; Engelmeier, M.P.; Heimann, H.; Heinrich, K.; Hippius, H.; Poldinger, W.; Schmidlin, P.; and Schmitt, W. Das Dokumentations-System der Arbeitsgemeinschaft fur Methodik und Dokumentation in der Psychiatrie (AMP). *Arzneimittel Forschung,* 19:399, 1969.

Benkert, O., and Hippius, H. *Psychiatrische Pharmacotherapie.* New York: Springer-Verlag, 1974.

Bobon, D.P. *Le Manuel AMDP.* Liège: Presse Universitaire, 1978.

Bohaček, N. Discussion working group 4. In: Bente, B., and Bradley, P.B., eds. *Pharmacological and Clinical Action of Neuroleptic Drugs. Proceedings of the IVth CINP Congress.* Amsterdam: Elsevier, 1965. pp. 175–178.

Bohaček, N. Social aspects of psychopharmacology. In: Ban, T.A.; Boissier, G.R.; Gessa, G.J.; Heimann, H.; Hollister, L.; Lehmann, H.E.; Munkvard, I.; Steinberg, H.; Sulser, F.; Sundwall, A.; and Vinar, O., eds. *Psychopharmacology, Sexual Disorders and Drug Abuse: Proceedings of the VIIIth CINP Congress.* Amsterdam: North-Holland Publishing Company, 1973. pp. 75–82.

Bohaček, N. First translation of the AMP System: The Croation one. In: Deniker, P.; Radouco-Thomas, C.; and Villneuve, A., eds. *Neuropsychopharmacology: Proceedings of the Xth CINP Congress.* Vol. 2. New York: Pergamon Press, Inc., 1978. pp. 1565–1568.

Cattabeni, F.; Racagni, G.; Spano, P.F.; and Costa, E. Long-term effects of neuroleptics. In: Costa, E., and Greengard, P., eds. *Advances in Biochemical Psychopharmacology.* Vol. 24. New York: Raven Press, Inc., 1980.

Doongaji, D.R.; Sheth, A.S.; Apte, J.S.; Datta, M.R.; Parikh, M.D.; Lakdawalla, P.D.; Ratnaparkhi, S.; and Sharma, U. Penfluridol in chronic schizophrenia. *Acta Psychiatrica Belgica,* 81:407–415, 1981.

Fouks; Lerno; Périvier; and Gilbert. The clinical use of Deanol Acetylglutamate (DMAEG). *Annales Médico-Psychologiques,* 124:272–280, 1966.

Gerlach, J. *Tardive Dyskinesia.* Copenhagen: Legeforeningens Forlag, 1979.

Heinrich, K. Ueber psychotische Gestimmtheiten. In: Kranz, H., and Heinrich, K., eds. *Pharmakopsychiatrie und Psychopharmacologie.* Stuttgart: Georg Thieme, 1967. pp. 22–32.

Heinrich, K. *Psychopharmaka in Klinik und Praxis.* Stuttgart: Georg Thieme, 1976.

Müller, P. *Depressive Syndrome im Verlauf Schizophrener Psychosen.* Stuttgart: P. Enke Verlag, 1981.

Palmović, R., and Bohaček, N. Preliminary analysis of a discharged group of schizophrenics treated with psychotropic drugs. *Psihofarmakologija, Medicinska Naklada,* 2:145–152, 1970.

Peršić, N.; Hrčko, N.; Muačević, V.; and Vinek, Z. Primjena Sidnokarba u terapijskom aktiviranju psihotičnog procesa uz istodobno praćenje bioelektrične aktivosti mozga. In: Bohaček, N., ed. *Neki Problemi Eksperimentalne i Kliničke Psihofarmakologije.* Zagreb: Zbor Lijecnika, Hrvatske, 1972. pp. 97–105.

Shepherd, M.; Lader, M.; and Rodnight, R. *Clinical Psychopharmacology.* London: The English Universities Press, Ltd., 1968.

Vinar, O. Whom do neuroleptic drugs help? In: Ban, T.A.; Boissier, G.R.; Gessa, G.J.; Heimann, H.; Hollister, L.; Lehmann, H.E.; Munkvard, I.; Steinberg, H.; Sulser, F.; Sundwall, A.; and Vinar, O., eds. *Psychopharmacology, Sexual Disorders and Drug Abuse: Proceedings of the VIIIth CINP Congress.* Amsterdam: North-Holland Publishing Company, 1973. pp. 247–253.

World Health Organization. *Report of the International Pilot Study of Schizophrenia.* Vol. 1. Geneva: The Organization, 1973.

World Health Organization. *Schizophrenia: An International Follow-up Study.* New York: John Wiley & Sons, 1979.

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The Contribution of European Case Registers to Research on Schizophrenia

by Heinz Häfner and Wolfram an der Heiden

Abstract

Psychiatric case registers have become important instruments for epidemiological research and for the evaluation of care provided for persons suffering from schizophrenia. Under the condition of a sufficient provision of care, case registers permit relatively reliable estimates of "treated" incidence and prevalence, and enable the investigation of associations between morbidity risks, disease courses, and variables such as ecological, social, and occupational factors. The linkage with twin, adoption, and birth registers provides new opportunities for the investigation of the impact of genetic versus environmental factors on the probability of becoming ill with schizophrenia. On the basis of long-term utilization figures, case registers enable a valid measure for controlling utilization, effectiveness, quality, and costs of care. Because case registers take demographic and regional factors into account, they enable better planning of mental health services—a task that grows more important with the shift from hospital-based to community-based treatment, especially for the chronic patients.

Cumulative Psychiatric Case Registers: Composition and Organization

Cumulative psychiatric case registers are health information systems of a geographically delimited region. They register all contacts of their population with psychiatric services over long periods of time. Case registers were preceded by hospital statistics, which in general are based on aggregate data and therefore are of minor value to research. They were also preceded by disease registers to which hospitals and physicians reported all cases of a certain diagnosis. Disease registers were mostly used to collect large enough numbers of cases with rare diseases to enable clinical and genetic studies to be carried out. Only relatively recently have more and more disease registers, such as cancer registers, been run as population-related case registers, so that findings can be generalized, and changes in morbidity and environmental impact can be assessed. Cumulative psychiatric case registers collect and compile their data in relation to cases and exclude multiple counting. Contrary to disease registers, they cover the entire spectrum of diagnoses given to inhabitants of the catchment area when they contact a psychiatric service.

The functions of psychiatric case registers are determined by the extent to which they can be used. According to Wing, Wing, and Hailey (1970), there are four possible uses, namely correlation and comparison with other population-related registers and statistics, follow-up of utilization cohorts and of long-term utilization trends in an entire health care region, and finally drawing of defined, representative patient samples.

Case Identification. The procedure of case identification leading to registration in a case register is based on a diagnosis given by a psychiatrist who contributes to the provision of mental health care. Therefore, case registers could only be started in those areas where all psychiatrists had learned to diagnose according to common, sufficiently precise definitions—for example, in Norway after

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the adoption of Kraepelin's scheme of diagnoses in 1926 (Ødegård 1971*b*) or after the introduction of the ICD in various European countries about 1960 (Kendell 1975). The use of case registers for epidemiological research depends on the reliability of diagnoses and on the proportion of cases recorded in the register in relation to the total number of individuals of a population who suffer from the same disease.

The possibilities of epidemiological and service-related evaluation improve when, in addition to the main diagnosis, secondary diagnoses are also registered (e.g., three additional diagnoses are recorded in the Danish register (Dupont 1979)) or when diagnoses are based on a multiaxial classification.

In diseases that have a long course, the reliability of attribution to a diagnostic category is increased in a case register, because successive diagnoses by several services, as well as observation of the course, can be used to validate the first diagnostic judgment. Because the reliability of the diagnosis of schizophrenia has been satisfactory since the 7th and 8th revisions of the ICD (Klug 1983), the course of schizophrenia proves to be long term in about 70 percent of first onsets, and nearly all schizophrenics contact a psychiatric service in their lifetimes (Ødegård 1971*b*; Nielsen and Nielsen 1977), case registers are important instruments of research on schizophrenia. For psychiatric disturbances which have low diagnostic reliability, or of which only a minor proportion receive care from psychiatric services—for instance, neuroses—the use of psychiatric case registers is rather limited. Because of the increasing period over which disease courses can be observed and the growing amount of information to be evaluated, the scientific value of a case register increases with the length of its existence (Baldwin 1970; Helgason 1979).

Size of Catchment Area and Population. Registers are either run on a national level, as is done in Norway and Denmark (Ødegård 1952, 1971*a*, 1971*b*; Kiselev 1972; Dupont, Videbech, and Weeke 1974; Weeke, Kastrup, and Dupont 1979), or on a regional or local basis. *National* registers cover large population figures (about 5 million in Denmark, about 250 million in the U.S.S.R.). Thus, they comprise relatively large case figures of a specific diagnosis. National registers allow epidemiologists to investigate the relation of morbidity risk and course of schizophrenia to factors showing an uneven regional distribution, such as occupational status or ecological variables.

Regional or *local* registers usually comprise the catchment area of a community mental health service. Therefore, the population at risk included in these registers is comparatively small: Lomest, Italy—88,000 (Torre, Marinoni, and Allegri 1982); Salford, U.K.—130,000–140,000 (Wing and Fryers 1976); Iceland—about 110,000 (Helgason 1977); Mannheim, F.R.G.—about 300,000 (Häfner and Klug 1982). The non-European case registers are organized on a regional level, but most of them cover a larger population; e.g., Maryland, U.S.A.—about 3.5 million; Monroe County, U.S.A.—about 700,000; Victoria, Australia—about 3.5 million.

Since their catchment areas are easy to survey, most of the regional case registers cover all contacts with psychiatric services. They are able to furnish additional information about services and context variables of utilization or course of illness. Also, on the basis of regional registers, it is convenient to carry out followup studies in neighboring medical or social facilities. Regional case registers can contribute to research on schizophrenia, especially in the evaluation of mental health care systems and services, and in the investigation of epidemiological samples—for instance, prospective cohorts which allow the analysis of changes over long periods of time.

Information systems based on individual data—similar to case registers—originated in centralized national health care systems (e.g., in Finland). These information systems have the advantage of compiling utilization data from all branches of the health care system. Even though this approach reduces the reliability of relevant psychiatric data, general health registers can be used for making important epidemiological evaluations.

Services and Facilities Affiliated with a Register. When interpreting epidemiological data from case registers, one should take into consideration how extensive the network of services providing data is. The Norwegian register records admissions to all psychiatric hospitals and departments run in Norway (Ødegård 1971*a*, 1971*b*). The Danish register additionally includes data on all admissions to psychotherapeutic sanatoriums and psychiatric day and night clinics. In general, regional registers also receive information about contacts with psychiatric outpatient services; this information is nearly complete in centralized health care systems (e.g., the United Kingdom and the U.S.S.R.). In countries where a larger proportion of outpatient care is provided by psychiatrists in private practice (Federal Republic of Germany, Netherlands, Italy), outpatient utilization is often registered incompletely.

If a register also covers neighboring fields of the social or health care system, it improves the possibilities of evaluating interdisciplinary care provided to some patient groups. Thus, the Camberwell Register also includes data on homeless persons who contact a psychiatric service or are provided social care in homes of the catchment area (Wing and Hailey 1972; Wing and Fryers 1976). While the majority of the registers compile only data on mentally ill adults, a few are affiliated with mental health services for children and adolescents and with facilities for the mentally retarded (e.g., the Camberwell Register of Psychiatric Illness and Mental Retardation and the Register of Essex Hospital Region in the United Kingdom). The Mannheim Register comprises a subregister recording all parasuicidal acts seen in general hospitals of the region. The inclusion of these acts, which can be identified rather reliably, makes possible epidemiological studies on the risk of suicide and attempted suicide. Furthermore, the Mannheim Register comprises all psychiatric homes, sheltered apartments, and sheltered workshops. Case identification is made by a consultant psychiatrist. The inclusion of these facilities allows studies to be carried out of the utilization of alternative and complementary services by schizophrenic patients, and changes in the mental health care system to be evaluated (Häfner and an der Heiden 1982, 1983).

Diagnostic Groups Recorded. The Norwegian Case Register records only psychoses (ICD no. 295-299), whereas the majority of case registers cover all psychiatric diagnoses and contacts with mental health services.

Number of Items Registered. The number of registered items varies between 12 in the U.S.S.R. (Kiselev 1972) and a maximum of 126 in Aberdeen, Scotland (Baldwin 1971). Mean values are between 20 and 40 items (Camberwell, 20; Denmark, 26; Iceland, 29; Mannheim, 42). The restriction to a smaller number of items is generally dictated by the need to keep the proportion of missing information as small as possible. Although detailed social, health, and treatment data are of value to epidemiological research, a register that provides a minimum of information about each patient in a community is more important than one that provides more detailed information only from specific facilities, as pointed out by Murphy (1975).

Different levels of information—e.g., between inpatient and outpatient facilities—are bridged by block systems of data registration (Jakubaschk et al. 1978).

One exception is the Icelandic Case Register. Since 1960, it has registered only first contacts of all persons who utilize a psychiatric service, a general hospital, or a nursing home and receive a psychiatric diagnosis. This register is therefore exclusively used for epidemiological research (Mazer 1966; Helgason 1975, 1979) (see table 1).

Access to and Crossing With Public Statistics. In principle, disease registers need aggregate data from population statistics in order to get population-related, age-corrected rates and to be able to compute on the basis of parallelized control groups. Additionally, the local authorities (registrar's offices, public health offices, residents' registration offices) transmit case-related information about social and demographic indicators, deaths and causes of death, to most of the registers. This makes it possible to undertake investigations on mortality (e.g., the mortality of suicide) and to eliminate the deceased. But it also improves the completeness and quality of the registered items.

The crossing of psychiatric case registers with population registers, such as twin and adoption registers, is of great importance to epidemiology, especially for hypothesis testing. The best prerequisites for crossing registers are to be found in the Northern countries, which have relatively stable populations, excellent population registers, and psychiatric registers containing large case numbers without essential regional distortions.

Epidemiological Data

Indicators of True Morbidity. Under the conditions of a sufficient provision of care for the population and high utilization rates for the investigated disease group, as apply in schizophrenia, first contacts compiled by a case register can be regarded as *indicators of true morbidity*. Investigating the relations among morbidity risks, disease courses, and environmental variables is only appropriate if a diagnostic group is sufficiently large and representative. The analysis of such relations or trends over longer periods of time on the basis of case register cohorts is more valid than cross-sectional comparisons or longitudinal studies among hospital populations. Besides, long-term collection of data on representative patient groups—starting with first admissions—is an excellent instrument for observing the manifestation of diseases over a long period and for assessing changes that may occur due to therapeutic measures

Table 1. Some psychiatric case registers in Europe

Registers	Year of starting	Information going back to	Catchment area	Population at risk (approximate)	Registered population (case definition and affiliated services)
Norway/Oslo	1936	1916	National	8 million	Psychoses; psychiatric inpatient services
Denmark/Aarhus	1937	1905	National	5 million	All mental disorders; psychiatric inpatient services & partial hospitalization
Iceland/ Reykjavic[1]	1968	1908	Regional	40,000	All mental disorders; psychiatric inpatients & outpatients; general hospitals
U.S.S.R./Moscow	1968	1968	National	250 million	All mental disorders; mental hospitals & mental health dispensaries
U.K./Nottingham	1958	1958	Regional/ local	390,000	All mental disorders; mental health services
U.K./Salford	1959	1959	Regional/ local	135,000	All mental disorders; mental health services
U.K./Aberdeen	1962	1962	Regional/ local	200,000	All mental disorders; mental health services
U.K./Camberwell	1964	1964	Regional/ local	155,000	All mental disorders; mental retardation; mental health services; homeless people
F.R.G./Mannheim	1973	1973	Regional/ local	300,000	All mental disorders; mental health services & psychiatric components of social services
Netherlands/ Groningen	1973	1973	Regional/ local	43,000	All mental disorders; mental health services
Ireland/Three County Psychiatric Case Register	1973	1973	Regional	150,000	All mental disorders; mental health services
Italy/Lomest	1975	1975	Regional/ local	81,000	All mental disorders; mental health services
West Dublin County Register	1973		Regional	230,500	Inpatient & outpatient services; public & private

Note.—Further psychiatric case registers are run in the Netherlands on a national level and in Maastricht, in Geneva (Switzerland), Cardiff, Oxford, Southampton, Worcester (U.K.), Verona (Italy), and Basque Country (Spain).

[1] Not cumulative; first ever contacts only.

and health care provisions. The investigation of groups at high risk and the comparison with the rest of the population with regard to morbidity patterns, environmental conditions, and long-term changes may provide information about distribution processes of morbidity risks, connections with possible stress factors, and special needs for care.

Exact comparisons of "administrative" annual incidence rates for schizophrenia from several case registers cannot be made because of differences in definitions (e.g., diagnosis, first contact with a certain facility or with the register) and in denominators (e.g., total population or population aged 15 and over). The influence of differing denominators (total population versus population aged 15 and over) is shown by a comparison of incidence rates from the Mannheim Case Register calcu-

Table 2. Annual incidence rates for schizophrenia in Mannheim—1974-80

Year	First onsets (n)	Rate of first onsets related to: Total population per 1,000	Rate of first onsets related to: Population aged ≥ 15 per 1,000
1974	159	.48	.58
1975	176	.56	.67
1976	210	.67	.81
1977	195	.63	.75
1978	193	.63	.75
1979	174	.57	.67
1980	190	.62	.72
		.59	.71

Source: Psychiatric Case Register, Mannheim.

lated according to both methods for the period 1974–1980 (see table 2).

In spite of these differences, the incidence rates of various case registers range rather precisely about two mean values: the rates from British registers are about a mean of 15–20/100,000/year; those from the U.S.A., Ireland, and the F.R.G. amount to 50–60/100,000/year (see table 3). With respect to confidence intervals, this is a minor difference, but it is impossible to conclude whether it reflects a true difference in morbidity. It might stem, for example, from a wide or narrow definition of schizophrenia, the proportion of age groups at risk (especially the proportion of persons aged 20–40 years) in the total population, and the density of mental health services affiliated to a case register.

The point or period prevalence, i.e., the rate of manifestly ill persons in a population who are in contact with mental health services, has a greater probability of being complete and consistent. However, these rates are influenced by regional differences in morbidity (e.g., higher prevalence of chronically ill in large cities or in populations with a higher life expectancy in general). Furthermore, with long-lasting diseases in particular, they are also influenced by the care system, and by high proportions of long-term hospital patients (e.g., in Ireland) (Häfner and Klug 1982; Häfner and an der Heiden 1983) (see table 4).

Long-term Epidemiology. The Scandinavian case registers collect large numbers of first admissions over long periods, and thus may serve as a basis for investigating correlations and differential processes of distribution for relevant social variables. While first admission rates remained stable over several decades in Iceland (Helgason 1975), in Denmark (Dupont and Weeke 1977; Weeke and Strömgren 1978), and in Norway (Ødegård 1971*b*) (the same is true for the admission rates of persons aged up to 35 (Weeke and Strömgren 1978) and of persons up to 30 years (Ugelstad 1978)), point prevalence rates and length of hospital stays of schizophrenic patients varied greatly. In Norway, the sharpest decline took place in the period 1936–59 (Ødegård 1964). In Denmark, the number of schizophrenic patients in mental hospitals dramatically decreased by about 30 percent between 1957 and 1962 (Dupont and Weeke 1977; Weeke and Strömgren 1978). By 1971, there had been only a slight further reduction, which has remained constant since then. Preceded by a steady increase, in England and Wales, the number of hospitalized schizophrenics continuously declined by over 50 percent in the period 1956–73. These trends reflect systematic efforts to curtail the length of hospital stays of the chronically mentally ill and to accommodate them in complementary facilities.

Following Ødegård's model, Astrup (1982) studied first admissions for schizophrenia recorded in the Norwegian Register from 1926 to 1978. He calculated the lifetime risk of ten 5-year cohorts and one 3-year cohort in a period of investigation covering 53 years. He took the life expectancy of the same age classes of the total population as a basis (timetable method). The lifetime risk for schizophrenia declined in males from 194/100,000 in 1926–30 to 42/100,000 in 1977–78; in females it decreased from 179/100,000 in 1926–30 to 35/100,000 in 1977–78. To the same extent, the "lifetime risk for the diagnostic group of reactive psychoses and paranoid syndromes" increased from 43/100,000 to 167/100,000 in males and from 52/100,000 to 220/100,000 in females. In the more comprehensive diagnostic category of "functional psychoses," the lifetime risk corrected for age and age group remained stable over a period of 53 years. According to Astrup (1982), the decrease in first admission rates for schizophrenia can be explained by the increasing tendency of Norwegian psychiatrists to diagnose schizophrenic disorders that are precipitated by life events, and show severe

Table 3. Incidence rates for schizophrenia from psychiatric case registers

Place	Author	Year	Rate/1,000
Norway	Astrup (1982)	1926–30	.23[1]
Norway	Astrup (1982)	1977/78	.24[1]
Iceland	Helgason (1977)	1966/67	.27
Camberwell, U.K.	Hailey et al. (1974)	1971	.13
Salford, U.K.	Fryers et al. (1974)	1971	.11
Denmark	Dupont and Weeke (1977)	1972/73	.12[2]/.06[3]
Victoria, Australia	Krupinski (1984)[9]	1978	.16
Netherlands	Giel et al. (1980)	1978/79	.11[4]
South-Verona, Italy	Tansella et al. (1985)	1983	.08[5,6]
Maryland, U.S.A.	Warthen et al. (1967)	1963	.50
Mannheim, F.R.G.	Häfner and Reimann (1970)	1965[7]	.54
Mannheim, F.R.G.	Häfner	1974–80[8]	.59
Rochester, U.S.A.	Babigian (1975)	1970	.69
Oberbayern, F.R.G.	Dilling and Weyerer (1975)	1974/75[7]	.48

Note.—Includes data from case registers in Europe, United States (survey based on comparable sources of data collection), and Australia. Incidence rate = first contacts with a psychiatric case register per year and per 1,000 total population.

[1] "Lifetime expectancy rate," hospitalized cases only, schizophrenia, and "reactive psychoses."

[2] Rate separately for men (hospitalized cases of schizophrenia without "reactive psychoses").

[3] Rate separately for women (hospitalized cases of schizophrenia without "reactive psychoses").

[4] Including ICD 297.0–297.9; 298.3–298.9.

[5] Rate for first-ever contacts.

[6] Rate for schizophrenia and other functional psychoses.

[7] Survey based on comparable sources of data collection.

[8] Data from the Cumulative Psychiatric Case Register of the Central Institute of Mental Health, Mannheim, F.R.G.

[9] Personal communication.

Table 4. Prevalence rates for schizophrenia from psychiatric case registers

Place	Author	Year	Rate/1,000
Baltimore, U.S.A.	Wing et al. (1967)	1963	5.1[1]
Camberwell, U.K.	Wing et al. (1967)	1965	3.4[2]
Rochester, U.S.A.	Babigian (1975)	1970	4.7[2]
Ireland	Walsh et al. (1980)	1973	8.3[1]
Mannheim, F.R.G.	Häfner[3]	1974–80	2.3[2]

[1] Census day prevalence.

[2] Period prevalence.

[3] Data from the Cumulative Psychiatric Case Register of the Central Institute of Mental Health, Mannheim, F.R.G.

affective symptoms or comprehensible delusional ideas, as a "reactive psychosis" or a "paranoid syndrome" (Ødegård 1967; Astrup 1982). This assumption was confirmed by cohort studies of Achté (1961), Noreik et al. (1967), and Welner and Strömgren (1958) (see, also, Kringlen 1980).

Although the provision of complementary services and outpatient care received much greater emphasis in recent years, the long-term study carried out in Iceland among a much smaller register population (Helgason 1975, 1977) showed stable rates of first contacts with mental health services for schizophrenia when three periods were compared: 1926–27, 1946–47, and 1966–67. In the registers of Camberwell and Salford, Wing and Fryers (1976) found stable rates of first contacts for schizophrenia in the period 1964–74. The fact that the admission rate for schizophrenic patients aged under 30 years had remained stable in the Danish Case Register from 1957 to 1976 (Ugelstad 1978) also supported Ødegård's assumption: *The astonishing stability of first admission rates over half a century indicates that in the countries investigated the true incidence of schizophrenia remained essentially unchanged.* The findings also tallied with Goldhamer and Marshall's (1953) studies in Massachusetts. Due to non-age-corrected data from the Norwegian register, Astrup had assumed in 1956 that the frequency of schizophrenia would particularly increase in the period of rapid industrialization as a consequence of the capitalist society. However, Astrup corrected his results and their interpretation in his 1982 publication. As Astrup (1982) stated, his data are consistent with the hypothesis that functional psychoses are biological reaction types that are relatively independent of the social environment.

Ecological Factors. In the industrial City of Mannheim, which then counted 300,000 inhabitants, Häfner and Reimann (1970) once again found a concentric distribution pattern of first admission for schizophrenia, similar to the one found in Chicago by Faris and Dunham (1939). The analysis of deviating accumulation—e.g., in subcenters on the outskirts of the city—indicated that mobility steered by sociopolitical measures (such as housing projects) influences the distribution of morbidity. Ødegård (1972) could not furnish proof of a concentric distribution pattern of first admissions in Norwegian towns, although the first admission rates in the City of Oslo, like those from Copenhagen (Dupont and Weeke 1977), were higher than the national average. Levy and Rowitz (1970, 1973), in their replication of the study by Faris and Dunham (1939), were able to demonstrate in Chicago a zonal distribution pattern of the origin of patients readmitted for schizophrenia, but not of first admissions. Ødegård took this finding to be a confirmation of his assumption: Within the complex structure of modern cities, the migration processes of individuals at risk for schizophrenia cannot be described by unidimensional ecological models. Although the greatest concentration of domiciles of persons already suffering from schizophrenia is in cheap dwelling areas as a consequence of downward mobility, Ødegård's findings based on case register data were confirmed by Hare (1956) in Bristol and Stein (1957) in London. They showed that first onsets have a stronger connection with social isolation than with poverty.

Civil Status. At the time of first admission, schizophrenics are significantly more often unmarried than the population of the same age (Ødegård 1946; Dupont and Weeke 1977). A comparison of two time periods (1931-45 and 1956-65) showed that in Norway the proportion of unmarried males among first admissions for schizophrenia rose from 4.3 to 7.7 and that of unmarried females from 3.7 to 4.5 times the amount of the same age group in the general population. Ødegård attributed these differences to prepsychotic character traits. Social disability preceding the psychosis affects men more strongly than women, since in European culture men have to play a more active role in matchmaking. It is true that the rate of unmarried persons among schizophrenics rose in a period in which the frequency of marriage declined and the average age at marriage increased (1931-65). But the disproportionately higher increase in unmarried schizophrenic males reflects their disproportionately larger difficulties in active matchmaking due to prepsychotic disturbances in the ability to establish interpersonal relationships (Ødegård 1971*a*). Widowed persons have a considerably higher first admission rate compared to married ones, but a lower rate than unmarried persons. Ødegård (1971*a*) attributes this to the missing protective factor of matrimony, which at least in cases of less severe illness, diminishes the probability of hospital admission.

Social Class. The uneven distribution of schizophrenia over social classes or socioeconomic strata, being maximal in the lower class, is a consistent finding over nations and continents, with rare exceptions. Because schizophrenia frequently entails social disability, the uneven distribution of prevalence rates over social classes is much more obvious than the inequality of first admission or true incidence rates (Adelstein et al. 1968; Goldberg and Morrison 1963). However, the construct of "socioeconomic class" is too imprecise to be used to assess detailed hypotheses proposed to account for the distribution processes of individuals at risk for schizophrenia (Ødegård 1975).

Employment. Various factors unevenly distributed over time may have contributed to the long-term trend toward shorter hospital stays and extramural care of schizophrenic patients in industrial countries (Brown 1960; Häfner and Klug 1982). At first, the decrease in hospitalized schizophrenic patients resulted exclusively from the decline of long-term hospitalized patients (Wing and Fryers 1976; Fryers 1979; Häfner and Klug 1982; Häfner and an der Heiden 1983). As was earlier stated by Brenner (1967) in his study covering a 60-year period of hospitalization in New York (1900-60), Ødegård (1971) and Astrup (1982) pointed to the correlation of time between changes in the length of hospital stays of schizophrenic patients and the employment situation. The reversed trend observed by Fryers in Salford since 1973 and by Weeke and Strömgren on the basis of the Danish Case Register since 1971—a new increase in the accumulation rate for long-term hospitalized schizophrenic patients—is clearly connected with declining economic activity and increased unemployment in both countries. In the Federal Republic of Germany, where unemployment increased more recently, the proportion of long-term hospitalized patients decreased until 1980. In principle, the entry of psychosocially disabled persons into the free labor market must be understood as a threshold phenomenon: Whenever unemployment rates and demands on the applicants for a position are high, the number of schizophrenics who, due to their psychosocial disability, cannot find employment on the free labor market grows disproportionately in relation to the total population.

Therapeutic Measures. Ødegård attached only partial importance to the introduction of psychotropic drugs, since in Norway the greatest changes had already occurred in the 1936-50 period following the introduction of electroshock therapy. In some European countries, the provision of complementary services and their influence on the length of hospital stays of schizophrenic patients came to the forefront between 1960 and 1980 (Wing and Fryers 1976; Weeke and Strömgren 1978; Fryers 1979; Häfner and an der Heiden 1982, 1983), but this influence was counterbalanced by the unfavorable effects of the high unemployment rate (Wing and Fryers 1976; Weeke and Strömgren 1978; Weeke, Kastrup, and Dupont 1979; Dupont 1979). An increased provision of complementary services, especially of sheltered homes, apartments, and workshops, can keep the proportion of schizophrenic long-stay patients low even in times of economic recession and make it possible for the majority (about 75 percent) of schizophrenic patients needing care for over 1 year to live in the community (Häfner and an der Heiden 1983).

Occupation and Social Mobility. From an analysis of the age-corrected distribution of first admissions for schizophrenia over various occupational groups in the period 1926-65, Ødegård (1971*a*, 1971*b*) deduced that the largest proportion was to be found in occupational groups with little practical and theoretical training and low social prestige. He found high rates in sailors and farm laborers, and considerably lower rates in first mates of the merchant marine and farmers, although their situation in life and work is comparable. Since the morbidity risk of Norwegian seamen was about twice as high as that of industrial workers belonging to the same social class, Ødegård concluded that the economic status had no direct effect on the morbidity risk.

Long-term changes in the characteristic features of occupational groups gave further opportunities to compare two competing hypotheses. The "selection" hypothesis attributes the low socioeconomic status of schizophrenics to their preexisting disorder or to their predisposition to develop disorder. By contrast, the sociogenetic or "breeder" hypothesis links the development of schizophrenia to preexisting social precipitants or stressors. In Norway, the occupational groups with unusually high first admission rates for schizophrenia (seamen and farm workers), which continued to increase in the period of observation, showed a sharp decline in employment. Ødegård demonstrated this process by the example of female domestic servants, an occupation that nowadays has almost died out in Norway: The exceptionally high first admission rates for schizophrenia in this occupational group contrasted with significantly lower rates for single housewives without occupation, a group which differed very little from the domestics with respect to their life and work situation. From this, Ødegård concluded that occupations which are dying out and offer little chance of success are the domain of individuals who, because of their social disability, avoid changing to more promising occupations. They are joined by newly disabled individuals from other occupational groups, for whom the lower threshold of qualification makes it possible to find employment. These data do not support the sociogenetic hypothesis that schizophrenia is caused or precipitated by social strain. However, the selection hypothesis is supported by the substance and dimension of the described processes of distribution over social situations. Thus, Ødegård's findings in case register studies point in the same direction as the findings of Goldberg and Morrison (1963) on intragenerational mobility of individuals suffering from schizophrenia. Eaton (1980) recently used a stochastic model to demonstrate that social intergenerational and intragenerational mobility in male schizophrenics, compared to normal males, fully explains the quantitative differences in social class distribution.

Selective Effects of Premorbid Behavioral Patterns. Since the processes leading to uneven distribution in marriage, in the choice of an occupation, and in migration were in effect before first admission, they must have been determined by premorbid traits of those who later would develop schizophrenia. Noreik and Ødegård (1966) retrospectively examined the school files of a sample of schizophrenic patients and compared them with a matched control group. They found that those pupils who later developed schizophrenia, on the average, had obtained lower grades and had had fewer social relations than their classmates. These findings, which have since been verified by prospective studies of individuals at high risk for schizophrenia (Garmezy 1981), support Ødegård's (1972) and Astrup's (1982) assumption that the social processes which lead to an uneven distribution of the morbidity risk are initiated by the preexisting personality traits of individuals who later develop schizophrenia.

Schizothymic personality traits, lack of social contact, rigidity, and lack of self-assertiveness can also be found more frequently in the lower classes (Ødegård 1975). From this apparent evidence of social selection, Ødegård (1975) deduced that schizophrenia is deeply rooted in the patient's past and that its causes should be sought, not at the time of onset, but in the premorbid period. Ødegård considers it possible that the earliest manifested disturbances are not character traits, but initial symptoms of a psychosis that is taking a very slow course. At this early stage, the personality change can barely be recognized as pathological. Ødegård's conclusion has implications for theories about the productive (positive) symptoms that characterize schizophrenia. The fact that the productive symptoms are not powerful predictors of long-term outcome (Strauss and Carpenter 1977; Tsuang, Woolson, and Fleming 1979; Schubart et al. 1982) could be explained as follows: The real process of schizophrenia, which is accompanied by the development of social disability and is of greatest prognostic value, in general precedes and is largely independent of, episodes of productive symptoms, which may be precipitated by additional factors.

Geographic Mobility (Migration). The first comprehensive empirical study of the relation between morbidity rates for schizophrenia and migration was published by Ødegård in 1932. It was based on data from the Norwegian Case Register and on hospital admission data from Minnesota. The studies demonstrated elevated first admission rates in Norwegians who had emigrated to the United States in comparison to the native population in the United States and in Norway. Even in Norwegians who had reemigrated from the United States, the first admission rates for schizophrenia were elevated (Ødegård 1973). Malzberg (1969) demonstrated that the differences diminished when analyses controlled for the variables of age and sex. The tendency, however, remained.

Jointly with members of his staff, Ødegård then carried out several systematic studies on the influence of migration processes on the morbidity rate for schizophrenia. Within the country, Dalgard (1971), Astrup and Ødegård (1960), and Ødegård (1975) found significantly lower first admission rates for schizophrenia in migrants from rural to urban areas and from large cities to suburbs. However, the frequency of first admission was increased in migrants from urban to rural areas. In connection with his findings on processes of labor migration, Ødegård favored an explanation suggested by the selective hypothesis: Premorbid personality traits, especially the lack of social relationships and occupational success, make it easier to decide to leave home and occupation. This explains the fact that in migration streams connected with social ascent there are relatively few, whereas in migration streams of individuals with little social success there are relatively many, individuals with a disposition to schizophrenia.

The morbidity rate for schizophrenia in Hungarian emigrants to Norway, who had left their home country mainly for political reasons, was investigated by Eitinger and Grünfeld (1966) on the basis of the Norwegian register. In the period from 1956 to 1963, hospital admission rates for schizophrenia were slightly elevated in emigrants compared with the Norwegian population and showed a trend to assimilate after a longer stay in Norway.

On the basis of hospital statistics for England and Wales, Cochrane (1977, 1979) found significantly higher first admission rates for schizophrenia in Irish women (but not men) and in immigrants from the West Indies and Pakistan. Bebbington et al. (1981) compared the morbidity rates of West Indian immigrants with those of Irish immigrants and of the native population. Their study used data from the Camberwell Register and from an additional population study covering the catchment area of the register. The purpose of the study was to test the following three alternative hypotheses: (1) The morbidity hypothesis—the morbidity of schizophrenia is dependent on racial or national differences. (2) The selection hypothesis—previous abnormalities reflecting a predisposition to schizophrenia influence the decision to emigrate. (3) The stress hypothesis—stress due to emigration and confrontation with a foreign culture increases morbidity.

A comparison between immigrants and the population born in England corrected for age showed significantly higher true incidence rates for schizophrenia in immigrants from the West Indies, in females more markedly than in males. Admission rates and rates for treated prevalence were also elevated in this group. In immigrants born in Ireland, Bebbington et al. (1981) could only partially verify Cochrane's findings, which had not been corrected for age: Incidence rates for schizophrenia were clearly lower in Irish men and clearly, although not considerably, higher in Irish women than in the local population. Bebbington et al. points out that the three hypotheses are not exclusive, but they attribute the greatest importance to the selection hypothesis.

The case register study on psychiatric morbidity of foreign workers in Mannheim done by Häfner (1980) underlines the extraordinary importance of the selection hypothesis. In the period of investigation—1974-78—about 13 percent of the Mannheim population were foreigners. The age composition of foreign workers showed an overrepresentation of younger age groups and thus differed markedly from the German population. Consequently, the rates of psychiatric morbidity among foreign workers were correspondingly lower. When corrected for age, the rates of treated schizophrenic episodes (a comparison of first admission rates was not appropriate because of the low number) were still significantly lower than those of the German population. The lowest rates were found among Turks and slightly reduced rates were found among Italians, whereas Yugoslavs and the group of other foreigners in which refugees prevailed did not differ significantly from the native population. The distribution of the rates for alcohol-related diseases and organic brain syndromes was analogous; however, the rates for affective psychoses and for neurotic and psychosomatic disorders showed no marked differences between the foreign groups and the German population. Häfner (1980) assumed that both the decision to go abroad to work and the chances of passing a medical screening before emigration are influenced by an early onset of chronic disease courses and by premorbid abnormalities of personality. Medical examination by the German labor administration is most severe for Turkish workers. Higher morbidity rates among women of migratory workers may be explained by the fact that they contribute less to the decision to emigrate and are examined less thoroughly. In contrast to the earlier mentioned emigrant groups, in Germany foreign workers represent an example of positive selection (reduced disposition to schizophrenia and other long-term mental disorders). As a consequence, in comparably poorer regions in which large proportions of the healthy population emigrate, the remaining individuals tend to be those who are old and at risk for chronic mental disorders. This may result not only in an unhealthy population structure, but also in a relative increase in the morbidity rates for schizophrenia, as was found in Istria, a region in Yugoslavia that was affected by several waves of emigration (Lemkau et al. 1980), and in Ireland (Scheper-Hughes 1979).

Morbidity Risk for Schizophrenia After Extreme Stress. In several studies, Eitinger (1959, 1960, 1967) used data from the Norwegian Case Register to investigate the morbidity rate for schizophrenia in individuals who had spent several years in German displaced persons (DP) and concentration camps by means of data from the Norwegian Case Register. In a first study, he found 14 persons suffering from schizophrenia among a total of 19,000 persons returned from German concentration and DP camps. Thus, in this group, the rate for the lifetime risk is markedly higher than in Norwegians. However, the group could not be compared for age, social class, or other factors.

In a later study, Eitinger and Strøm (1973) compared a total of 4,447 Norwegians who had returned from German concentration camps with a control group matched for age, sex, and socioeconomic status. The investigators aimed at a more precise determination of mortality and morbidity following severe stress. A representative sample of 480 males and 18 females out of this group were interviewed. The prevalence rate for schizophrenia among survivors of concentration camps was identical with that found in the control group of the Norwegian population. In each of the two interview samples, three cases of schizophrenia had occurred since the war. In contradiction to his former assumption and to the assumption of other authors (e.g., Baeyer and Binder 1982), which were not based on epidemiological results, Eitinger thus could not find any indication that the risk of schizophrenia increases after the severe stress of a stay in a concentration camp. These findings, as well as the unchanged incidence rates for schizophrenia in belligerent countries, are of importance for the hypothesis that any sort of mental stress precipitates psychoses.

Risk for Subgroups of Schizophrenia. As long as clinical diagnosis is not based on standardized diagnostic instruments and as long as detailed findings are not recorded, case register data do not allow inferences to be drawn about changes in the incidence of certain symptoms, syndromes, or subgroups of schizophrenia. Only syndromes that are reliably identified, such as febrile catatonia, are an exception. On the basis of hospital statistics and catamnestic samples, Pauleikoff (1969), Huber (1969) and M. Bleuler (1972) had assumed that the syndrome of febrile catatonia had ceased to occur since 1960 as a result of improved therapy. By means of case register data and data on the utilization of a unit for intensive psychiatric care in Mannheim, Hafner and Kasper (1982) showed that in the period 1974–81, acute life-threatening catatonia occurred with an almost unchanged frequency of episodes per year, about 1/300,000 total population.

The fact that this syndrome disappeared from the psychiatric hospitals in the F.R.G. may be explained by changes in the system of medical care: After the opening of intensive care units at hospitals or departments for internal medicine, neurology, and anesthesiology from about 1960 on, the treatment of life-threatening syndromes was shifted from the psychiatric hospitals, most of which are insufficiently equipped, to these units.

Special Population Groups at Risk for Schizophrenia. Noreik and Ødegård (1966) compared the admission rates of a cohort consisting of about 13,000 Norwegians who in the period 1916 to 1925 had finished secondary education, with those of Gypsies and homeless persons and those of neglected and criminal adolescents in Norway. While the academic cohort showed a slightly lower prevalence rate for schizophrenia in both sexes compared to the total population corrected for age, the rate of schizophrenia was significantly higher than expected among individuals without fixed abode and dissocial adolescents. Ødegård's cohort studies allow statements on the morbidity risk of the investigated population group, whereas the increased prevalence rates for schizophrenics among homeless persons found in Camberwell (Wing et al. 1972) point to an augmented risk of social deterioration and social disintegration in persons suffering from schizophrenia. The followup investigation of representative samples, drawn from the Camberwell Case Register, in the community (Wing and Olsen 1979) confirmed this tendency: Many of them lived in poor circumstances, were out of work, and not able to accomplish their tasks at home. Nevertheless, few members of this socially neglected group wanted to accept offers made by mental health or welfare services. Apparently in contradiction to this, Wing and Brown (1970) had found that most schizophrenic patients who had been living in mental hospitals for many years preferred to stay there. Chronic schizophrenic patients seem to be more afraid of a change in their present circumstances than of the low quality of life often characterizing conditions in the hospital or in the community.

Genetic Studies.[1] After Luxenburger (1936) had used twin registers to get undistorted concordance rates for schizophrenia, Tienari first examined a larger series in 1963: 903 out of 1,053 pairs of male twins from the Finnish Twin Register. Among 15 monozygotic pairs with a clearly schizophrenic index twin, he found no cotwin who met strict criteria for schizophrenia. When a wider definition was applied, three cotwins could be identified as cases: one as reactive psychosis and two as borderline schizophrenia. Later Tienari (1975) crossed the original twin population with the register of all psychiatric hospitals and psychiatric outpatient departments in Finland. The result was essentially unchanged. Only the cotwin first diagnosed with a reactive psychosis later received a diagnosis of schizophrenia.

The most important study is the one carried out by Kringlen (1964). For the period 1901–30, he drew a total of 25,588 twin pairs out of the Norwegian national birth statistics and matched them with the Central Register of Psychoses. The concordance rate of monozygotic twins was 25 percent when narrow, and 38 percent when broad, definitions of schizophrenia were applied. The corresponding rates for dizygotic twins were 4 percent and 10 percent, respectively. Kringlen also found that 38 percent of monozygotic cotwins having schizophrenic partners were given the diagnosis of neurosis or "borderline" disease and that about 30 percent were clinically healthy. Similar to Slater (1953) and Tienari (1963), Kringlen showed that the more dependent, submissive twin is at greater risk to develop schizophrenia in the course of his life than the dominant one.

Fischer, Harvald, and Hauge (1969) and Fischer (1973) crossed all same-sexed twin pairs listed in the Danish Twin Register as being born in the period 1870–1920 with the National Psychiatric Register in Aarhus. Among the 21 monozygotic and 41 dizygotic twins of the same sex who had a schizophrenic partner, they found concordance rates of schizophrenia of 24 percent in monozygotic and 10 percent in dizygotic twins. Furthermore, Fischer examined the children of healthy monozygotic cotwins. The morbidity rate of these children almost equals the rate of monozygotic partners of schizophrenic twins. The number is still too small, however, for these findings to be generalized. The difference between the consistently lower concordance rates from studies using case register data and the mostly much higher and more strongly varying risk figures from nonrepresentative twin populations is mainly due to different sampling methods. The introduction of register strategies greatly contributed to progress in psychiatric genetics (Rosenthal 1962; Kringlen 1967; Shields 1975).

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[1] E. Kringlen (1980) described most of the results of the epidemiological studies carried out among twins and adopted persons in his report on the contribution of the Scandinavian countries to research on schizophrenia. We therefore confine ourselves to a short survey.

The Danish adoption studies were designed to test the alternative hypotheses of a genetic transmission of schizophrenia versus a family/environmental transmission of schizophrenia. They were based on crossing an adoption register with the Danish Case Register. A sample of 5,483 children from the Province of Copenhagen who had been legally adopted by nonrelatives in the period 1924-47 formed the basis of a two-part investigation: (1) a comparison between adopted-away children of schizophrenic patients and adopted children of nonschizophrenic parents (Rosenthal et al. 1968) and (2) a comparison between the biological and adoptive relatives of schizophrenic adoptees and those of nonschizophrenic control adoptees. For case identification in the parents, the register data were supplemented by reports on the parents taken from the adoption files (Kety et al. 1968).

The adopted-away children of schizophrenic and control parents underwent a blind interview and a large battery of tests. Three out of 76 index cases were given a diagnosis of schizophrenia by at least one rater, whereas none of the control cases had developed schizophrenia. As the findings were not significant because of the small sample size, the investigators extended the case definition to the so-called "schizophrenia spectrum disorder," a rather imprecise diagnostic group. They then found 24 (31.6 percent) positive cases among 76 index cases and 12 (17.9 percent) among 67 control cases, a difference that was significant on the 0.5 percent level. The same case definition and the same method of data collection were applied in the second phase of the study, which took as its point of departure adopted persons who developed schizophrenia. In this phase, the biological parents were compared with the adoptive parents.

Meanwhile repeated secondary analyses were made on the basis of these data (Haier, Rosenthal, and Wender 1978; Kendler, Gruenberg, and Strauss 1981*a*, 1981*b*, 1981*c*; Lidz, Blatt, and Cook 1981). The findings, partially processed according to the criteria of *DMS-III* (Kendler, Gruenberg, and Strauss 1981*a*, 1981*b*, 1981*c*), generally affirmed that neither the number of biological relatives who received a reliable diagnosis of schizophrenia nor the number of index cases among the adoptees were sufficiently large for definite conclusions to be drawn. There remains a clear trend toward a greater probability of developing schizophrenia in genetic as compared to adoptive parent and child relationships. This trend needs to be confirmed on the basis of larger case numbers and of precise criteria of case definition and identification. As a next step, the adoption registers could be crossed with a psychiatric case register on a national level.

Quite a few of the Scandinavian register studies were carried out in populations at risk. Mednick and Schulsinger (1971) undertook a prospective longitudinal study among children of schizophrenic mothers in Denmark. Out of 173 children, who were re-examined at an average age of 28, eight (4.6 percent) compared to one (1.1 percent) out of 91 controls had developed schizophrenia. Tienari et al. (1979) identified all females who had been given the diagnosis of schizophrenia and had been recorded in the Finnish Case Register between 1960 and 1970. By crossing these data with the birth register, he formed a sample of those 120 children who had been separated from their genetic mothers by adoption. Kringlen (1978) investigated a sample of 48 married couples in which both partners had a functional psychosis recorded in the Norwegian Register and had been hospitalized. The average age of the parents was 38 years. Five out of 25 children (20 percent) developed schizophrenia; seven (28 percent) developed a reactive psychosis. When corrected for age, the lifetime expectancy rate of schizophrenia was relatively low, at 29 percent. Kringlen emphasized that 28 percent of the children of two schizophrenic parents had remained clinically normal.

Evaluative and Services Research

Assessment of Needs, Planning, and Evaluation of Services. Case registers cover only treated morbidity. A precise assessment of changes in morbidity, especially in the morbidity of those mental disorders in which only a minority receive mental health services, requires additional field studies or data collection in primary health services, particularly in general practice (Ødegård 1972; Fryers 1979). A large amount of work is required in such studies. L. Wing et al. (1968) pointed out that about 100,000 persons would have to be examined per year in order to find 15 new (first onset) cases of schizophrenia. Therefore, field studies are carried out only in rare cases in needs assessment and, if so, with a precise formulation of the research questions to be addressed.

Although they cannot contribute much to the direct assessment of unserved needs of a population, registers are of great importance in evaluating the adequacy of services and care systems. They allow us to ascertain which group of patients utilized which elements of available services at what time and to what extent. They enable us to identify patients who do not utilize offers of care recommended to them, and facilitate the assessment of specific needs of patient groups, e.g., for alternative offers of care, which are still uncovered (Fryers 1979). This presupposes the use of existing knowledge of specific needs of certain patient groups, which—in case they are not covered—lead to misplacement or lack of care.

Demand and Utilization. The utilization of mental health services cannot be equated with a region's actual needs. Data on utilization also do not permit direct conclusions on the effectiveness of services to be drawn. To determine the uncovered proportion of need (see Matthew 1971; Alderson 1976; Häfner 1979; Jakubaschk et al. 1978; Klug, an der Heiden, and Scheel 1980), it would be necessary to do field studies as described above. The field studies would include persons who reject psychiatric care although it is clearly indicated or turn to other medical disciplines (see Weyerer et al. 1982). With only a few exceptions, psychiatric case registers are restricted to utilization data from psychiatric services. The amount of missing information resulting from this restriction lessens the generalizability of findings, but this problem applies much less to the group of schizophrenic patients than to those with milder conditions. When data are also collected from alternative and complementary services, as is done by the Mannheim Case Register (Häfner and Klug 1980, 1981, 1982), changes of care provided to chronic schizophrenic patients—following the recommendations laid down under the concept of "comprehensive community mental health care" (World Health Organization 1973)—can be described continuously.

A crude comparison of census data on treated prevalence collected by eight case registers for all diagnostic groups shows considerable differences at the first glance (see table 5). The data partially reflect different stages of the development of outpatient and complementary services in the respective regions. Besides, they reveal differences in the proportion of long-term hospitalized patients and the completeness of data collection by the institutions contributing to mental health care (see, also, Walsh et al. 1980).

Planning. In the past, changes in the field of mental health care were often made without taking into consideration the scientific knowledge about factors that influence the extent of disease and impairment in a population and also the provision of optimal care (Wing and Hailey 1972). For this reason—often to an unusual degree and unacceptable to the persons affected—political ideologies found expression in laws or measures alleged to improve mental health care.

The prediction of future need on the basis of utilization trends, and demographic and social developments, is important for planning services. Its reliability increases when the factors influencing a large part of the variation are analyzed and the trends are observed over a longer period of time (Ødegard 1972).

There are numerous examples of valuable contributions by case registers to the planning of psychiatric services. Thus, in 1976, the need for psychiatric hospital beds in the Danish provinces was assessed on the basis of the Danish Case Register following a request of the government. The findings were used to prepare the transfer of responsibility for psychiatric hospitals from the state government to the provincial governments (Strømgren et al. 1979). In the United Kingdom, case register studies have long been used to interpret the misleading

Table 5. Point prevalence rates of some European case registers [1]

Register	Rate	Point prevalence	Source
Camberwell, U.K.	769	Dec. 12, 1974	Wing & Fryers (1976)
Salford, U.K.	538	Dec. 31, 1974	Wing & Fryers (1976)
Mannheim, F.R.G.	681	May 15, 1974	Häfner & Klug 1981
Randers, Denmark	822 [2]	March 1, 1977	Kastrup & Bille (1980)
Three County, Ireland	1770	March 31, 1973	Walsh et al. (1980)
Groningen, Netherlands	834	Dec. 31, 1975	Giel & ten Horn (1981)
Lomest, Italy	268	Dec. 31, 1975	Marinoni et al. (1983)
Aberdeen, U.K.	682	Dec. 31, 1966	Baldwin (1971)

[1] Inpatient and outpatient care; rates per 100,000 total population.

[2] Census data added.

aggregate data of hospital statistics and of the National Health Service, and also to correct prognoses of need for hospital beds and alternative facilities (Department of Health and Social Security 1975).

Evaluation of Utilization. In the last decades, a transition from hospital-centered to community mental health care, including outpatient and complementary services, took place in many European countries (World Health Organization 1973; Department of Health and Social Security 1975; Report of the Expert Commission on Psychiatry, F.R.G., 1975). This process of marked change in the system of care, which was accompanied by a considerable increase in treatment prevalence, has been evaluated in various countries on the basis of case register data (Wing and Bransby 1970; Wing and Fryers 1976; Hafner and Klug 1980, 1981, 1982; Giel and ten Horn 1982).

It appears that the different levels of psychiatric care have contributed to the increase in treatment prevalence to a varying extent. The rate for contacts with complementary and outpatient services rose drastically, whereas the population-related rate of occupied beds declined in the regions analyzed or, in case of lower initial values, remained stable (Wing and Fryers 1976; Hafner and Klug 1982; Hafner and an der Heiden 1983).

In Mannheim, for example, the total number of treated episodes per year of schizophrenic patients increased from 485 to 756 in the period 1974–79; in the same period, the number of occupied beds decreased from 243 to 209 (Hafner and Klug 1981). There are two reasons for this trend: (1) the increased provision of outpatient and complementary services, accompanied by changes in the help-seeking behavior of schizophrenic patients (an der Heiden and Klug 1980; Hafner and an der Heiden 1982); (2) the drastic shortening of stays in inpatient services, as reported from other case registers (Gam 1980; Wing and Bransby 1970; Fryers 1974). The reduction of long-term hospital stays (over 1 year) is followed by a rise in short- and medium-term stays so that in spite of unchanged or decreasing rates of occupied beds, an equal number of schizophrenic patients has to be cared for at more frequent intervals during the same period of time.

This historical trend in the care of schizophrenic patients is clarified when the respective rates for the years 1974–79 from the Mannheim Case Register (Hafner and an der Heiden 1982) are added to a review of eight studies on discharge rates for schizophrenic patients 1–5 years after their first admission carried out between 1900 and 1950 (Brown 1960). The proportion of schizophrenics who, after first admission, stay in the hospital for more than 1 year has consistently declined since the turn of the century, from about 70 percent to about 5 percent in 1980 (see figure 1).

Simultaneously with the reduction of the average length of stay, the case/episode ratio has increased. In 1974, one hospitalized schizophrenic patient in Mannheim accounted for 1.16 inpatient treatment episodes per year, but this value increased to 1.28 by 1979 (Hafner and an der Heiden 1982), including long-term patients.

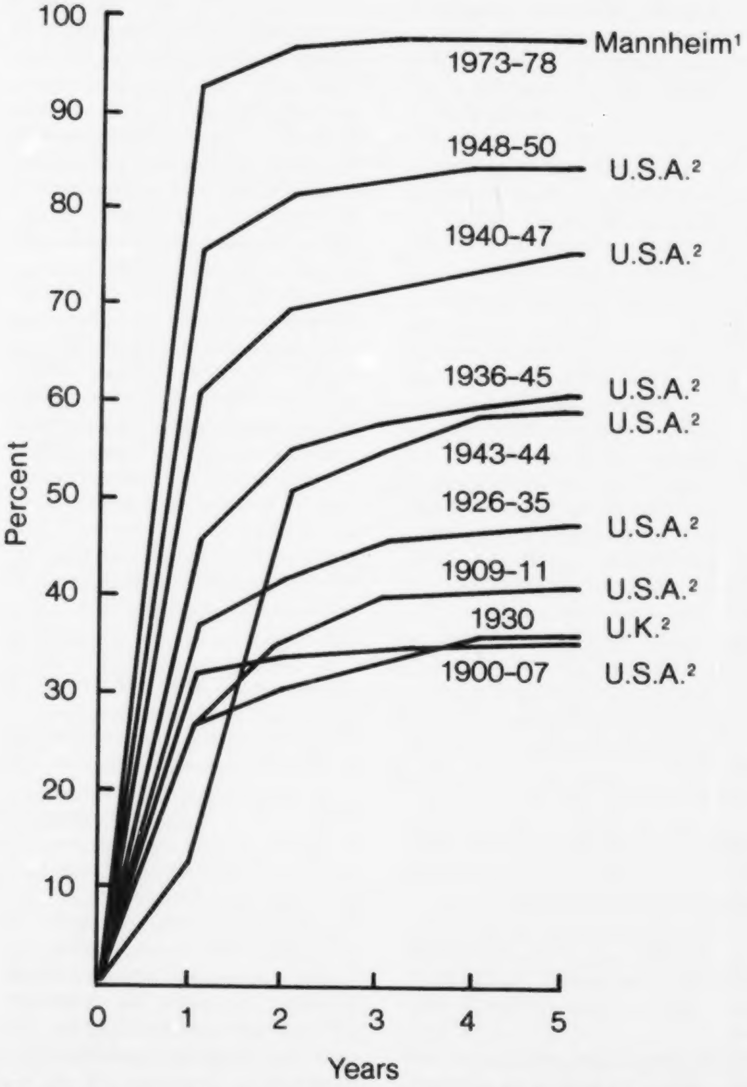
Long-term Patients. In the past, schizophrenics constituted by far the largest proportion of long-term hospitalized patients. On December 31, 1972, for example, 60.3 percent of all long-term hospital patients in Camberwell had been given a diagnosis of schizophrenia; in Salford they amounted to 66.8 percent in the same period (Wing and Fryers 1976). A 1-day census carried out in Mannheim on May 30, 1973, ascertained a proportion of 53 percent (Hafner and Klug 1982).

When the composition of inpatients over several census days is examined on the basis of case register data, the following trend can be discerned: In all diagnostic groups, the proportion of long-term patients in the total number of occupied beds is continuously diminishing; however, the rate of decrease greatly differs in the various areas of care (Wing and Bransby 1970; Wing and Fryers 1976; Hafner and Klug 1980, 1981, 1982; Giel and ten Horn 1982). An example from England and Wales reported by Wing and Fryers (1976) may illustrate this: In the period 1964–74, the rate of schizophrenic patients fell from 72.9 percent to 59.9 percent in Camberwell, whereas in Salford it remained stable between 1967 and 1973 at a high mean of 82 percent.

Comparison of "Old" and "New" Long-stay Cohorts. To show the changes in the need for long-term hospital admission in the context of a community mental health service, the total cohort of long-term patients can be divided into the following two subcohorts: (1) Patients who on a given census day had been hospitalized for over 1 year; to a large part, these are the inheritance of a past hospital policy. (2) Patients who from this census day on become new long-stay patients under the conditions afforded by modern treatment.

In all case register areas, a corresponding accumulation of "new" long-stay patients can be observed, and does not seem to have yet reached its peak (Wing and Bransby 1970; Hailey 1971, 1973, 1974; Wing

Figure 1. Percentage of schizophrenic patients discharged within 5 years after admission—1900-1950



Note.—Studies included seven from the United States, one study from the United Kingdom, and one study from Mannheim.

[1] From Cumulative Psychiatric Case Register, Central Institute of Mental Health, Mannheim.
[2] From Brown (1960).

Reprinted from Häfner, H., and an der Heiden, W. Evaluation gemeindenaher Versorgung psychisch Kranker. *Archiv für Psychiatrie und Nervenkrankheiten*, 232:71-95, 1982.

and Hailey 1972; Fryers 1974, 1979; Wing and Fryers 1976; Eason and Grimes 1977; Weeke, Kastrup, and Dupont 1979; Häfner and Klug 1980, 1981, 1982; Torre, Marinoni, and Allegri 1982).

In several publications, Hailey (1971, 1973, 1974) thoroughly dealt with the problem of long-stay hospital patients living in the catchment area of the Camberwell Register. In 1964, 54 percent of the patients with schizophrenia had been hospitalized for more than 20 years. Out of 95 male patients who had stayed in the hospital for more than 15 years, 83 were diagnosed schizophrenic. In Camberwell the rate for all long-stay patients per 100,000 population calculated on 5 census days between 1964 and 1969 consistently fell from 240.5 to 209.9[2]: Out of 410 patients who on the census day of December 31, 1964, had been admitted for over 1 year, 60.9 percent were still provided inpatient treatment on December 31, 1969.[3]

The reduction of the group of "old" long-stay patients and the accumulation of "new" ones proceeded markedly faster in Camberwell than in Salford. In addition to the differences in the structure of the care system, the different age composition of long-term patients (on December 31, 1972, about 60 percent of all patients recorded in the Camberwell Register were aged 65 and over, whereas this age group accounted for only about 43 percent in the Salford Register) played a role (Wing and Fryers 1976).

A comparison between both cohorts ("old" versus "new" long-term patients) made in Mannheim (census day: May 30, 1973; Häfner and Klug 1982) showed that, under a community mental health service, fewer schizophrenics needed long-term hospitalization than before: their rate decreased from 53 percent of the old to 35 percent of the new long-stay patients (Häfner and Klug 1982). Moreover, schizophrenics became long-stay patients at a considerably later time—mostly only after several preceding hospital stays. Parallels to the findings from Mannheim are to be found in the United Kingdom (Fryers 1979).

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[2] One-day prevalence on December 31, 1972: 173 (Wing and Fryers 1976).

[3] Percentage on December 31, 1970: 56.3 percent (Hailey 1973).

Beds Needed for Long-stay Patients. Before case register data were available, estimations of future needs for hospital beds were based on extrapolations from the change in bed occupancy over a defined period (e.g., Tooth and Brooke 1961) or on a retrospective analysis of the probability of discharge in relation to different lengths of stay in psychiatric hospitals (e.g., Baldwin and Hall 1967). Only the case-related inclusion of accumulating new long-stay patients in case registers allowed planners to make a differentiated analysis of changes in bed needs and residential placements resulting from changes in the mental health care system (Eason and Grimes 1977; Weeke, Kastrup, and Dupont 1979; Fryers 1974, 1979; Häfner and Klug 1982). Thus, case register data also permit a better interpretation of the trends observed in hospital statistics.

Complementary Services for Schizophrenics in the Community. Many chronically ill patients, who formerly would have been consigned to long-term residence in a psychiatric hospital, can now live in the community due to the extensive network of complementary services available. Studies carried out in the United Kingdom (Mann and Sproule 1972; Mann and Cree 1976; Magnus 1967) showed that, according to clinical judgment, even a large percentage of the new long-term hospitalized patients did not require treatment in a psychiatric hospital.

Wing (1982) investigated the specific needs of patients from the Camberwell Register who on June 30, 1979, had had contact with a halfway or complementary facility for at least 1 year. The study comprised institutions providing 24-hour care (hospital departments, homes) and also day clinics, day centers, and sheltered workshops. Patients with one episode of treatment of less than 1 year and patients who had been hospitalized for more than 1 year at the census day were excluded. Out of the 181 patients aged over 18—corresponding to a rate of 139 per 100,000 population—who met the criteria, almost half had received a diagnosis of schizophrenia. Regarding the distribution of sociodemographic variables, the composition was similar to that found in long-term hospital patients. In three quarters of the patients, the first contact with a psychiatric institution was more than 10 years in the past; only four of them had never been hospitalized before. Although the findings of this investigation primarily reflect the specific aspects of the mental health service in Camberwell, some general statements can be formulated. Thus, Wing (1982) emphasizes the need to examine each patient regularly in order to adjust the patient's placement to his needs and to improve his abilities in an environment that optimally encourages his independence.

The provision of support and advice to relatives as part of complementary health care for schizophrenic patients is of special importance. Over 40 percent of the sample drawn from the Camberwell Register had relatives who more or less regularly looked after them. The relatives often had to meet the same requirements as the staff of homes and nursing homes without being provided comparable institutional support.

A study based on data from the Mannheim Case Register (Häfner and an der Heiden 1983) showed to what extent the focus of care for chronic schizophrenic patients can be transferred from the psychiatric hospital to alternative services. In the period between the two census days, December 15, 1974, and December 15, 1979, the proportion of patients who remained in the hospital for more than 1 year among the total number of schizophrenic patients treated in psychiatric hospitals and homes (about 200-220) decreased from 85 percent (= 164) to 68 percent (= 148). In the same period, admissions of "new" long-stay patients from Mannheim, a city of about 310,000 inhabitants, to psychiatric homes increased from 7 in 1974 to 30 in 1979. Thus, in 1979, about three quarters of the annual admissions of all new long-stay patients with schizophrenia were made to psychiatric homes, whereas only about one quarter went to psychiatric hospitals. This distribution has remained stable to date.

Further Research on the Basis of Case Register Data. The necessity of continuous psychiatric treatment and of comprehensive care for chronic schizophrenic patients, including the fields of housing, occupation, leisure time, and social contacts, led to the implementation of a network of services. The evaluation of these complex patterns of care, their effectiveness, and the functions of every single service therefore gained significance (see Wing and Bransby 1970;

ten Horn 1980; Giel and ten Horn 1982; an der Heiden and Klug 1980).

Research on utilization of the network of community mental health services. Ten Horn (1980) demonstrated the multiple utilizations of outpatient facilities by 1,594 patients with different diagnoses who had been recorded in the Groningen Case Register (Netherlands) in 1974-75. The complex pattern of use of various services was even more striking in a cohort of 148 schizophrenic patients from Mannheim who had been admitted to inpatient services between October 1, 1977, and September 30, 1978 (Häfner and an der Heiden 1982, 1983). After hospital discharge, 116 (78.4 percent) patients contacted three to seven facilities of extramural care either simultaneously or successively. In most of the patients, it was a suitable combination of visits to a physician, home care, and attendance at a sheltered workshop and/or patients' club. These results demonstrate the interrelatedness of components of comprehensive community mental health care, a factor that should be considered in planning, coordinating, and analyzing the costs of such an extensive service. The effectiveness of intensive extramural care was demonstrated by the fact that the intensity of utilization considerably reduced the probability of rehospitalization.

Leff and Vaughn (1972) explored disease variables and social indicators that differentiated patients who used extramural care facilities from those who did not. A comparison between two samples of schizophrenic or manic-depressive patients drawn from the Camberwell Register (50 patients "in contact" and 40 patients "out of contact") revealed the following: Patients "in contact" had more frequently been treated in a hospital and to a larger extent received medication. This may indicate that the extent of care is flexibly adapted to the severity of the disease. Patients with more severe disabilities are provided more frequent and more intensive care. An investigation by Häfner and an der Heiden (1982) also showed a significant relation between the number of preceding hospital admissions and the intensity of use of outpatient and complementary services. In the study by Leff and Vaughn (1972), patients "in contact" had more frequent and also a larger number of symptoms, as reported by their relatives, and both family and occupational ties were less stable. Furthermore, the study indicated that among the patients who had no outpatient contacts, those receiving medication showed a better social adjustment (see Leff and Wing 1971).

Experimental research on the basis of case register data. Methodological problems in the retrospective analysis and interpretation of changes and of factors influencing them often reduce the generalizability of epidemiological research findings. This also applies to intervention studies carried out in a nonrepresentative utilization population, such as patients of a specific hospital. It is an inherent deficiency of many clinical and treatment studies. The combination of epidemiological and experimental designs is an excellent way of solving this problem.

Wing et al. (1972) made an interesting attempt to combine an epidemiological with a quasi-experimental approach. Out of a group of 380 schizophrenic patients recorded in the Camberwell Register in 1968, 75 were found who had been living at home for at least 1 year without occupation. Twenty-eight of these patients were divided into two equal groups. In order to evaluate the effectiveness of preparatory training for occupational rehabilitation, the experimental group was assigned to a day clinic program. All of the patients had severe secondary handicaps. Even though a followup examination after 2 years indicated no differences between the groups with respect to occupational reintegration (four patients from each group were employed) or social adjustment, the study nevertheless had some intriguing results: Observations during the phase of social activation indicated, for example, that intensive methods of social treatment, as well as pressure by the family, may lead to deterioration or acute relapses (Vaughn and Leff 1976). In addition, five patients from the original group were found to have committed suicide. So the suicide rate was 11 times as high (132/100,000) as that of the Camberwell population (12/100,000). From this study, it was concluded that the appropriate accommodation for the majority of chronic schizophrenics would be an environment in which permanent handicaps are accepted and excessive activation is avoided.

Methodology

Stability of Diagnoses. The cumulative case-related registration of successive contacts drew attention to the diagnoses attributed to a patient who had been repeatedly recorded in the register. Quite a few publications deal with the search for explanations of changes in the diagnoses recorded. In addition to the possibility of a patient's developing two independent psychiatric disorders, or expressing different states characteristic of the same basic disorder over the course of time, the influence of external, disease-related variables was examined. In one of the first studies based on case register data, Babigian et al. (1965) investigated the stability of diagnoses in 1,215 patients in the Monroe County Psychiatric Case Register who had contacted a psychiatric institution at least twice during 1961-62. When patients were assigned to four diagnostic categories (schizophrenia, affective psychoses, organic brain syndromes, and miscellaneous), the concordance of first and second diagnoses varied between 69 percent and 88 percent. The patient's setting and social class had no discernible influence, but the institution giving a first diagnosis seemed to play an important role, especially with respect to a change in diagnosis: In three quarters of all cases, the diagnosis "worsened," in the direction of a psychosis, if the first contact was with a physician in practice.

In the study of Babigian et al. the period between each contact did not influence diagnostic consistency, but Jakubaschk and Hurry (1977), using data from the Camberwell Register, found that concordance diminished in parallel with the length of the interval between two diagnoses. Concordance was assessed with respect to the 13 diagnostic categories of the register (not identical with the ICD classification) in a random sample of 144 patients entered in 1970 who had had at least one subsequent contact. In 50.5 percent of the cases, the diagnoses given at the first and last contact were identical; there was no significant connection between diagnostic changes and changes of the attending physicians or the institution.

A central register containing all data on admissions to and discharges from psychiatric hospitals in England and Wales was used by Cooper (1967) to investigate the reliability and quality of the recorded diagnoses. In a sample of 293 patients who were registered as first admissions in the second half of 1955 and had three readmissions in the following 2 years, the diagnoses were identical (four-figure ICD classification) in only 20 percent of cases. Unlike Jakubaschk and Hurry (1977), Cooper found an important connection between change of physician and change of diagnosis: A change of physician increased the probability of being given a different diagnosis. Cooper concluded that most diagnostic changes are not based on changes in the clinical picture, but on inadequate diagnostic criteria and classificatory systems.

In a study using the Mannheim Psychiatric Case Register, Klug (1983) included data for 2,028 persons who had had 2-12 contacts with psychiatric facilities between May 31, 1973, and July 31, 1976. For the 17 diagnostic categories investigated (ICD, 8th revision), diagnostic concordance between the first two dates of utilization was about 67 percent when both primary and secondary diagnoses were taken into account. The average time difference between the two diagnoses was 244 days. The comparatively high level of diagnostic agreement found by Klug (1983) may reflect improvements in the classificatory system and effects of training among the diagnosing psychiatrists. Among individual diagnostic groups, lifelong disturbances (chronic and recurrent diseases such as mental retardation and schizophrenia) showed the highest levels of stability.

In Klug's study, the interval between two treated episodes had an unexpected influence: If time differences were small, concordance rates were much lower than with larger differences of time. This result may reflect a tendency to stabilize a diagnosis when the amount of information, particularly on indicators of course, is growing. A further aspect points into the same direction: In case of a change of institution, the stability of diagnoses grew continuously, from 55 percent to 63 percent, between the first and the third change.

Although their findings on the effects of a change of physician and institution or on diagnostic habits differ, all investigators concluded that the type of psychiatric disorder is an important factor. The concordance rate for schizophrenia was about 70 percent throughout (see, also, Kaelbling and Volpe 1963) and was relatively insensitive to influences of time and institution. The effects of external factors, particularly of multimorbidity, on the stability of diagnoses may be diminished by giving primary and secondary diagnoses and by attaching different weights to them (Dupont 1980). The reduction and standardization of diagnostic categories (Babigian et al. 1965; Cooper 1967; Jakubaschk and Hurry 1977; Klug 1983) may also lead to an increase in stability, albeit at the price of a loss of information. The introduction of a multi-axial classification makes it possible to classify important variables on different levels of stability. Diagnostic reliability can be improved if standardized diagnoses are made by specially trained staff of the case register (Cooper 1967; see, also, Walsh et al. 1980).

Data Processing and Evaluation. The amount and quality (in the sense of measurement theory) of information delivered to a case register require increasingly differentiated methods of data processing and analysis. By developing a special documentation system, an der Heiden and Klug

(1980) illustrated the use of intramural and extramural institutions by schizophrenic patients in Mannheim over an 18-month period on the basis of 14-day intervals. The system made it possible to calculate the probability of transition between various stages of care over the course of time. With a time series analysis (Markov chain), they showed that after about 6 months, roughly 63 percent of a cohort utilized aftercare services, 17 percent got inpatient care, and 20 percent were not provided any care. These proportions remained stable at every further cross-section within the 18-month period (Häfner and an der Heiden 1982). This method could also be useful in cost-effectiveness analyses.

Summary

Because schizophrenia often takes a long-term course and is linked with socially conspicuous disturbances, the probability that a schizophrenic will contact a mental health service at least once in his lifetime approximates 100 percent if a sufficiently developed network of care is provided. Case registers therefore are important instruments of research on schizophrenia. In particular, their value to epidemiological and services research increases with the length of period they cover.

The first cumulative psychiatric case register was established in Oslo in 1936. The data of this national register of psychoses go back to 1916. Most local and regional registers in European countries were started in the 1960's in connection with the introduction of the ICD classification. These registers cover smaller populations, but record all contacts with psychiatric inpatient services, and a portion of contacts with complementary and outpatient facilities, irrespective of the diagnosis given.

The validity of cross-sectional comparisons of rates for first admissions and treated prevalence is limited because of the varying registration-density, differing definitions, and—reflecting the age-dependent morbidity risk—different denominators. The development of first admission rates, which can be observed over a period of 53 years in the Norwegian register and over 30 years in the Icelandic and Danish registers, proved to be an indicator of the long-term trend in morbidity: In schizophrenia, the morbidity risk, controlled for age, seems not to have changed in these countries for about half a century. The correlations between the rates for first admissions and demographic, ecological, and social factors—primarily investigated by Ødegård et al. on the basis of the Norwegian Case Register—strongly suggest the validity of selection hypotheses.

In investigations of the frequency of schizophrenia among migrating population groups, the results were inconsistent: In Norwegian immigrants to the United States and in Southeast Asian immigrants to Great Britain, the frequency of schizophrenia was higher, whereas the age-corrected rates for foreign workers in the F.R.G. were lower. These findings can be interpreted on the basis of different selective processes: Those selective factors leading to an unequal distribution at the onset of the disease seem to reflect certain traits of the premorbid personality of preschizophrenic patterns of social behavior. Long-term epidemiology and the analysis of geographic migration and social mobility could not verify the hypothesis that environmental factors—such as social systems, social context variables, or severe stress—might heighten the risk of becoming schizophrenic. Some of the findings tend to refute the assumption that the risk of illness is causally dependent on environmental factors.

By crossing data from twin and case registers, genetic research on schizophrenia for the first time obtained valid empirical data on hereditary factors in schizophrenia. The hypothesis of a genetic transmission of the predisposition to develop schizophrenia was confirmed, although some portion of the risk appeared to depend on environmental factors. Adoption studies based on Danish population registers have made an important contribution to the assessment of genetic versus familial/environmental factors in the transmission of schizophrenia. However, the results of the adoption studies need to be confirmed in larger samples.

Long-term trends in utilization of mental health services can be discerned in analyses of case register data, which permit relatively reliable determinations of future needs. Because they take demographic and regional factors into account, case registers contribute to a better interpretation of aggregate data from hospital statistics and to more valid planning of mental health services. The evaluation of mental health care systems and services on the basis of case registers has enhanced significance because of recent fundamental changes in mental health care. Schizophrenics are the main target group of the reform in the mental health care system, and they also provide the largest group of patients for whom care has been transferred from hospital to complementary services. In this context, case registers are an indispensable instrument for assessing utilization,

effectiveness, quality, and costs of interrelated components of an increasingly complex system of care.

In addition, case registers have been used as a basis for combining epidemiological and quasi-experimental methods of research on schizophrenia. This approach has contributed findings (e.g., on the specific vulnerability of chronic schizophrenics) that can be generalized. The possibilities of using case register data to follow the course of a disease and its dependence on intervening variables, to study the stability of syndromes and diagnoses, and to develop new methodologies have not yet been fully exhausted.

References

Achté, K.A. Der Verlauf der Schizophrenien und der schizophreniformen Psychosen. Eine vergleichende Untersuchung der Veränderungen in den Krankheitsbildern, der Prognosen und des Verhältnisses zwischen den Kranken und dem Arzt in den Jahren 1933–1935 und 1953–1955. *Acta Psychiatrica Scandinavica*, Suppl. 155, 1961.

Adelstein, A.M.; Downham, D.Y.; Stein, Z.; and Susser, M.W. The epidemiology of mental illness in an English city: Inceptions recognized by Salford psychiatric services. *Social Psychiatry*, 3:47–59, 1968.

Alderson, M.R. Indicators of need, demand, and use. In: Acheson, R.M.; Hall, D.J.; and Aird, L., eds. *Seminars in Community Medicine: Health Information, Planning, and Monitoring*. Vol. 2. London: Oxford University Press, 1976. pp. 29–40.

Astrup, C. *Nervöse Erkrankungen und soziale Verhältnisse*. Berlin: Volk und Gesundheit, 1956.

Astrup, C. "The Increase of Mental Disorders." Unpublished manuscript from the National Case Register of Mental Disorder, Gaustad Hospital, Oslo, Norway, 1982.

Astrup, C., and Ødegård, Ø. Internal migration and mental disease in Norway. *Psychiatric Quarterly*, Suppl. 34:116–130, 1980.

Babigian, H.M.; Gardner, E.A.; Miles, H.C.; and Romano, J. Diagnostic consistency and change in a follow-up study of 1215 patients. *American Journal of Psychiatry*, 121:895–901, 1965.

Babigian, H.M. Schizophrenia: Epidemiology. In: Freedman, A.M.; Kaplan, H.I.; and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry*. Vol. II. Baltimore: Williams and Wilkins, 1975. pp. 860–866.

Baeyer, W. von, and Binder, W. *Endomorphe Psychosen bei Verfolgten*. Berlin-Heidelberg-New York: Springer, 1982.

Baldwin, J.A. The register as a mental health service information system. In: Wing, J.K., and Bransby, E.R., eds. *Psychiatric Case Registers*. DHSS, Statistical Report Series No 8. London: Her Majesty's Stationery Office, 1970. pp. 9–13.

Baldwin, J.A. *The Mental Hospital in the Psychiatric Service: A Case-Register Study*. London-New York-Toronto: Oxford University Press, 1971.

Baldwin, J.A., and Hall, I.J. Estimation of the outcome of a standing mental hospital population. *British Journal of Preventive and Social Medicine*, 21:56–65, 1967.

Bebbington, P.B.; Hurry, J.; Tennant, C.; and Wing, J.K. The epidemiology of mental disorders in the Camberwell community. *Psychological Medicine*, 11:561–579, 1981.

Bleuler, M. *Die schizophrenen Geistesstörungen: im Lichte langjähriger Kranken- und Familiengeschichten*. Stuttgart: Thieme, 1972.

Brenner, M.H. Economic change and mental hospitalization: New York State, 1910–1960. *Social Psychiatry*, 2:180–188, 1967.

Brown, G.W. Length of hospital stay and schizophrenia: A review of statistical studies. *Acta Psychiatrica et Neurologica Scandinavica*, 35:414–430, 1960.

Cochrane, R. Mental illness in immigrants to England and Wales: An analysis of mental hospital admissions. *Social Psychiatry*, 12:25–35, 1977.

Cochrane, R. Psychological and behavioural disturbance in West Indians, Indians and Pakistanis in Britain: A comparison of rates among children and adults. *British Journal of Psychiatry*, 134:201–210, 1979.

Cooper, J.E. Diagnostic change in a longitudinal study of psychiatric patients. *British Journal of Psychiatry*, 113:129–142, 1967.

Dalgard, O.S. *Migration and Functional Psychoses in Oslo*. Oslo: Universitetsforlaget, 1971.

Department of Health and Social Security. *Better Services for the Mentally Ill*. London: Her Majesty's Stationery Office, Oct., 1975.

Dilling, H., and Weyerer, S. Zur Behandlungsinzidenz und -prävalenz in drei Kreisen Oberbayerns. In: *Bericht uber die Lage der Psychiatrie in der Bundesrepublik Deutschland—Zur Psychiatrischen und psychotherapeutisch/psychosomatischen Versorgung der Bevölkerung*. Drs. 7/4201. Bonn: Deutscher Bundestag, 1975.

Dupont, A. Psychiatric case registers. In: Häfner, H., ed. *Estimating Needs* *for Mental Health Care*. Berlin-Heidelberg-New York: Springer, 1979. pp. 43-51.

Dupont, A. "Entry in a Case Register as a Definition of a 'Case.' " Paper presented at the WPA Symposium "What is a Case." London, May 14-16, 1980.

Dupont, A.; Videbech, T.; and Weeke, A. A cumulative national psychiatric register: Its structure and application. *Acta Psychiatrica Scandinavica*, 50:161-175, 1974.

Dupont, A., and Weeke, A. Antalet af psykiatriske indlaggelser i Danmark. *Ugeskrift for Lager*, 139:1241-1242, 1977.

Eason, R.J., and Grimes, J.A. Inpatient care of the mentally ill: A statistical study of future provision. *Health Trends*, 8:13-18, 1977.

Eaton, W.W. A formal theory of selection for schizophrenia. *American Journal of Sociology*, 86:149-158, 1980.

Eitinger, L. Incidence of mental disease among refugees in Norway. *Journal of Mental Science*, 105:326-338, 1959.

Eitinger, L. The symptomatology of mental disease among refugees in Norway. *Journal of Mental Science*, 106:947-966, 1960.

Eitinger, L. Schizophrenia among concentration camp survivors. *International Journal of Psychiatry*, 3:403-406, 1967.

Eitinger, L., and Grunfeld, B. Psychoses among refugees in Norway. *Acta Psychiatrica Scandinavica*, 42:315-328, 1966.

Eitinger, L., and Strøm, A. *Mortality and Morbidity After Excessive Stress*. New York: Humanities Press, 1973.

Faris, R.E.L., and Dunham, H.W. *Mental Disorders in Urban Areas: An Ecological Study of Schizophrenia and Other Psychoses*. Chicago: University of Chicago Press, 1939.

Fischer, M. Genetic and environmental factors in schizophrenia. *Acta Psychiatrica Scandinavica*, Suppl. 238, 1973.

Fischer, M.; Harvald, B.; and Hauge, M. A Danish twin study of schizophrenia. *British Journal of Psychiatry*, 115:981-990, 1969.

Fryers, T. Psychiatric in-patients in 1982—how many beds? *Psychological Medicine*, 4:196-211, 1974.

Fryers, T. Estimation of need on the basis of case register studies: British case register data. In: Hafner, H., ed. *Estimating Needs for Mental Health Care*. Berlin-Heidelberg-New York: Springer, 1979. pp. 52-63.

Fryers, T.; Newton, P.; Sousa, R.; and Wooff, K. *Psychiatric Services in Camberwell and Salford: Statistics From the Camberwell and Salford Register: 1964-1973*. London: MRC Social Psychiatric Unit, 1974.

Gam, J. A 5-year retrospective investigation of all first admitted schizophrenics to Danish psychiatric institutions in the fiscal year from April 1st, 1970, to April 1st, 1971. In: Stromgren, E.; Dupont, A.; and Achton-Nielsen, J., eds. *Epidemiological Research as a Basis for the Organization of Extramural Psychiatry*. Copenhagen: Munksgaard, 1980. pp. 332-336.

Garmezy, N. The current status of research with children at risk for schizophrenia and other forms of psychopathology. In: Regier, D.A., and Allen, G.A., eds. *Risk Factor Research in the Major Mental Disorders*. (Publication No. ADM 81-1068) Washington, DC: Superintendent of Documents, U.S. Government Printing Office, 1981. pp. 23-39.

Giel, R.; Sauer, H.C.; Slooff, C.J.; and Wiersma, D. Epidemiological observations on schizophrenia and disability in the Netherlands. *Tijdschrift voor Psychiatrie*, 11-12:710-722, 1980.

Giel, R., and ten Horn, G.H.M.M. "Mental Health in Pilot Study Areas: The Dutch Experience—Introduction to the Participation of the Department of Social Psychiatry." Unpublished manuscript, The Netherlands, August, 1981.

Giel, R., and ten Horn, G.H.M.M. Patterns of mental health care in a Dutch register area. *Social Psychiatry*, 17:117-123, 1982.

Goldberg, E.M., and Morrison, S.L. Schizophrenia and social class. *British Journal of Psychiatry*, 109:785-802, 1963.

Goldhamer, H., and Marshall, A. *Psychosis and Civilization*. Glencoe, IL: Free Press, 1953.

Hafner, H., ed. *Estimating Needs for Mental Health Care*. Berlin-Heidelberg-New York: Springer, 1979.

Hafner, H. Psychiatrische Morbiditat von Gastarbeitern in Mannheim. Epidemiologische Analyse einer Inanspruchnahmepopulation. *Nervenarzt*, 51:672-683, 1980.

Hafner, H., and an der Heiden, W. Evaluation gemeindenaher Versorgung psychisch Kranker. Ergebnisse von 4 Jahren wissenschaftlicher Begleitung der Aufbauphase des Mannheimer Modells. *Archiv fur Psychiatrie und Nervenkrankheiten*, 232:71-95, 1982.

Hafner, H., and an der Heiden, W. The impact of a changing system of care on patterns of utilization by schizophrenics. *Social Psychiatry*, 18:153-160, 1983.

Häfner, H., and Kasper, S. Akute lebensbedrohliche Katatonie. Epidemiologie und klinische Befunde. *Nervenarzt*, 53:385-394, 1982.

Hafner, H., and Klug, J. First evaluation of the Mannheim community mental health service. In: Stromgren, E., ed. "Epidemiological Research as Basis for the Organization of Extramural Psychiatry." *Acta Psychiatrica Scandinavica*, 62 (Suppl. 285):68–78, 1980.

Hafner, H., and Klug, J. Wissenschaftliche Begleitung der Entwicklung einer gemeindenahen psychiatrischen Versorgung in Mannheim. In: Haase, H.J., ed. *Burgernahe Psychiatrie im Wirkungsbereich des psychiatrischen Krankenhauses*. Erlangen: Perimed Fachbuch-Verlagsgesellschaft, 1981. pp. 23–58.

Hafner, H., and Klug, J. The impact of an expanding community mental health service on patterns of bed usage: Evaluation of a four-year period of implementation. *Psychological Medicine*, 12:177–190, 1982.

Hafner, H., and Reimann, H. Spatial distribution of mental disorders in Mannheim 1965. In: Hare, E.H., and Wing, J.K., eds. *Psychiatric Epidemiology*. London-New York-Toronto: Oxford University Press, 1970. pp. 341–354.

Haier, R.J.; Rosenthal, D.; and Wender, P.H. MMPI assessment of psychopathology in the adopted-away offspring of schizophrenics. *Archives of General Psychiatry*, 35:171–175, 1978.

Hailey, A.M. Long-stay psychiatric inpatients: A study based on the Camberwell register. *Psychological Medicine*, 1:128–142, 1971.

Hailey, A.M. The chronic mental hospital population: A six-year follow-up study. *British Journal of Preventive and Social Medicine*, 27:255–260, 1973.

Hailey, A.M. The new chronic psychiatric population. *British Journal of Preventive and Social Medicine*, 28:180–186, 1974.

Hailey, A.M.; Hurry, L.J.; Powell, M.; Shannon, B.; Wing, J.K.; and Wing, L. *Psychiatric Services in Camberwell and Salford: Statistics From the Camberwell and Salford Register: 1964–1973*. London: MRC Social Psychiatric Unit, 1974.

Hare, E.H. Family setting and the urban distribution of schizophrenia. *Journal of Mental Science*, 102:753–760, 1956.

an der Heiden, W., and Klug, J. An integrated record system for the observation of the demand for medical and social aftercare as basis for organizing extramural services. *Acta Psychiatrica Scandinavica*, 62 (Suppl. 285):54–59, 1980.

Helgason, L. Psychiatric services and mental illness in Iceland. *Acta Psychiatrica Scandinavica*, Suppl. 268, 1977.

Helgason, T. Studies on prevalence and incidence of mental disorders in Iceland with a health questionnaire and a psychiatric case register. In: Astrup, C., ed. *Tromso Seminar in Medicine*. Tromso: University of Tromso, 1975. pp. 172–183.

Helgason, T. Estimation of need on the basis of case register studies: Discussion. In: Hafner, H., ed. *Estimating Needs for Mental Health Care*. Berlin-Heidelberg-New York: Springer, 1979. pp. 64–68.

ten Horn, G.H.M.M. Register study: A small cohort of multiple-service-users from a geographically circumscribed area. *Acta Psychiatrica Scandinavica*, Suppl. 285:305–314, 1980.

Huber, G. Psychiatrische Syndrome bei endogenen Intoxikationen und lebensbedrohlichen Katatonien. *Verhandlungen der deutschen Gessellschaft fur innere Medizin*. Vol. 75. München: Bergmann, 1969.

Jakubaschk, J., and Hurry, J. Die Diagnosen im Camberwell-Register, einem kumulativen psychiatrischen Fallregister. *Schweizer Archiv fur Neurologie, Neurochirurgie und Psychiatrie*, 120:243–255, 1970.

Jakubaschk, J.; Klug, J.; Weyerer, S.; and Dilling, H. Behandlungsbedurftigkeit—Uberlegungen zur psychiatrischen Versorgung. *Psychiatrische Praxis*, 5:203–211, 1978.

Kaelbling, R., and Volpe, P.A. Constancy of psychiatric diagnosis in re-admissions. *Comprehensive Psychiatry*, 4:29–38, 1963.

Kastrup, M., and Bille, M. A census study with special regard to long-stay psychiatric patients. In: Stromgren, E.; Dupont, A.; and Achton-Nielsen, J., eds. *Epidemiological Research as Basis for Organization of Extramural Psychiatry*. Copenhagen: Munksgaard, 1980. pp. 270–278.

Kastrup, M.; Nakane, A.; Dupont, A.; and Bille, M. Psychiatric treatment in a delimited population—With particular reference to outpatients. *Acta Psychiatrica Scandinavica*, 53:35–50, 1976.

Kendell, R.E. *The Role of Diagnosis in Psychiatry*. Oxford-London-Edinburgh-Melbourne: Blackwell Scientific Publications, 1975.

Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia. I. The relationship between anxiety disorder and schizophrenia. *Archives of General Psychiatry*, 38:973–977, 1981*a*.

Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia. II. The relationship between schizotypal personality disorder and schizophrenia. *Archives of General Psychiatry*, 38:982–984, 1981*b*.

Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia. III. The relationship between paranoid psychosis (delusional disorder) and the schizophrenia spectrum disorders. *Archives of General Psychiatry*, 38:985–987, 1981c.

Kety, S.S.; Rosenthal, D.; Wender, P.H.; and Schulsinger, F. The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In: Rosenthal, D., and Kety, S.S., eds. *The Transmission of Schizophrenia*. Oxford: Pergamon Press, Ltd., 1968. pp. 345–362.

Kiselev, A.S. Centralized system for collection and analysis of data on patients in the U.S.S.R. *American Journal of Psychiatry*, 128:1019–1022, 1972.

Klug, J. *Psychiatrische Diagnosen. Einflussfaktoren auf die Stabilitat im zeitlichen Verlauf*. Weinheim: Beltz, 1983.

Klug, J.; an der Heiden, W.; and Scheel, R. Psychiatrische Versorgung im Wandel—Determinanten der Bedarfsplanung. In: Biefang, S., ed. *Evaluationsforschung in der Psychiatrie: Fragestellungen und Methoden*. Stuttgart: Enke, 1980. pp. 250–285.

Kringlen, E. Schizophrenia in male monozygotic twins. *Acta Psychiatrica Scandinavica*, Suppl. 178, 1964.

Kringlen, E. Twins—Still our best method. *Schizophrenia Bulletin*, 2:429–433, 1967.

Kringlen, E. Adult offspring of two psychotic parents, with special reference to schizophrenia. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment*. New York: John Wiley & Sons, Inc., 1978. pp. 9–24.

Kringlen, E. Schizophrenia in the Nordic countries. *Schizophrenia Bulletin*, 6:566–578, 1980.

Leff, J., and Vaughn, C. Psychiatric patients in contact and out of contact with services: A clinical and social assessment. In: Wing, J.K., and Hailey, A.M., eds. *Evaluating a Community Psychiatric Service*. London: Oxford University Press, 1972. pp. 259–274.

Leff, J., and Wing, J. Trial of maintenance therapy in schizophrenia. *British Medical Journal*, 3:599–608, 1971.

Lemkau, P.V.; Kulcar, Z.; Kesic, B.; and Kovacic, L. Selected aspects of the epidemiology of psychoses in Croatia, Yugoslavia. IV. Representative sample and results of the survey. *American Journal of Epidemiology*, 112:661–674, 1980.

Levy, L., and Rowitz, L. The spatial distribution of mental disorders in Chicago. *Social Psychiatry*, 5:1–11, 1970.

Levy, L., and Rowitz, L. *The Ecology of Mental Disorders in Chicago*. New York: Behavioral Publications, 1973.

Lidz, T.; Blatt, S.; and Cook, B. Critique of the Danish-American studies of the adopted-away offspring of schizophrenic parents. *American Journal of Psychiatry*, 138:1063–1068, 1981.

Luxenburger, H. Untersuchungen an schizophrenen Zwillingen und ihren Geschwistern zur Prüfung der Realität von Manifestationsschwankungen. *Zentralblatt für die gesamte Neurologie und Psychiatrie*, 154:351–394, 1936.

Magnus, R.V. The new chronics. *British Journal of Psychiatry*, 113:555–556, 1967.

Malzberg, B. Important statistical data about mental illness. In: Arieti, S., ed. *American Handbook of Psychiatry*. Vol. I. New York: Basic Books, Inc., 1969. pp. 161–174.

Mann, S.A., and Cree, W. "New" long-stay psychiatric patients: A national sample survey of fifteen mental hospitals in England and Wales 1972/73. *Psychological Medicine*, 6:603–616, 1976.

Mann, S.A., and Sproule, J. Reasons for six months stay. In: Wing, J.K., and Hailey, A.M., eds. *Evaluating a Community Psychiatric Service*. London: Oxford University Press, 1972. pp. 233–248.

Marinoni, A.; Torre, E.; Allegri, G.; and Comelli, M. Lomest psychiatric case register: The statistical context required for planning. *Acta Psychiatrica Scandinavica*, 67:109–117, 1983.

Matthew, G.K. Measuring need and evaluating services. In: McLachlan, G., ed. *Portfolio for Health: Problems and Progress in Medical Care*. 6th series. London-New York-Toronto: Oxford University Press, 1971. pp. 27–46.

Mazer, M. A psychiatric and parapsychiatric register for an island community. *Archives of General Psychiatry*, 14:366–371, 1966.

Mednick, S., and Schulsinger, F. Breakdown in individuals at high risk for schizophrenia: Possible predispositional perinatal factors. *Mental Hygiene*, 54:50–63, 1971.

Murphy, H.B.M. Evaluation of community psychiatric programmes. In: *Psychiatrie der Gegenwart. Bd. 3: Soziale und angewandte Psychiatrie*. 2. Aufl. Berlin-Heidelberg-New York: Springer, 1975. pp. 361–427.

Nielsen, J., and Nielsen, J.A. A census study of mental illness in Samsø. *Psychological Medicine*, 7:491–503, 1977.

Noreik, K.; Astrup, C.; Dalgard, O.S.; and Holmboe, R.A. A prolonged follow-up of acute schizophrenic and schizophreniform psychoses. *Acta Psychiatrica Scandinavica,* 43:432–443, 1967.

Noreik, K., and Ødegård, Ø. Psychosis in Norwegians with a background of higher education. *British Journal of Psychiatry,* 112:43–55, 1966.

Ødegård, Ø. Immigration and insanity: A study of mental disease among the Norwegian-born population in Minnesota. *Acta Psychiatrica et Neurologica Scandinavica,* Suppl. 4:1–206, 1932.

Ødegård, Ø. Marriage and mental disease. *Journal of Mental Science,* 92:35–49, 1946.

Ødegård, Ø. The incidence of mental diseases as measured by census investigations versus admission statistics. *Psychiatric Quarterly,* 26:212–218, 1952.

Ødegård, Ø. Pattern of discharge from Norwegian psychiatric hospitals before and after the introduction of psychotropic drugs. *American Journal of Psychiatry,* 120:772–778, 1964.

Ødegård, Ø. Reactive psychoses. *Acta Psychiatrica Scandinavica,* Suppl. 203, 1967.

Ødegård, Ø. Epidemiologie der Psychosen. *Nervenarzt,* 42:569–575, 1971*a*.

Ødegård, Ø. Hospitalized psychoses in Norway: Time trends 1926–1965. *Social Psychiatry,* 6:53–58, 1971*b*.

Ødegård, Ø. Epidemiology of the psychoses. In: Kisker, K.P.; Meyer, J.E.; Muller, C.; and Strömgren, E., eds. *Psychiatrie der Gegenwart.* Berlin-Heidelberg-New York: Springer, 1972. pp. 213–258.

Ødegård, Ø. Norwegian emigration, re-emigration and internal migration. In: Zwingmann, C., and Pfister-Ammende, M., eds. *Uprooting and After. . . .* Berlin-Heidelberg-New York: Springer, 1973. pp. 161–177.

Ødegård, Ø. Social and ecological factors in the etiology, outcome, treatment and prevention of mental disorders. In: Kisker, K.P.; Meyer, J.E.; Müller, C.; and Strömgren, E., eds. *Psychiatrie der Gegenwart.* Vol. II, 2nd ed. Berlin-Heidelberg-New York: Springer, 1975. pp. 151–198.

Pauleikoff, B. Die Katatonie (1868–1968). *Fortschritte der Neurologie, Psychiatrie und Ihrer Grenzgebiete,* 37:461–496, 1969.

Report of the Expert Commission on Psychiatry, F.R.G. [*Bericht uber die Lage der Psychiatrie in der Bundesrepublik Deutschland—Zur psychiatrischen und psychotherapeutisch/psychosomatischen Versorgung der Bevölkerung.*] Drs. 7/4200, 7/4201. Bonn: Deutscher Bundestag, 1975.

Rosenthal, D. Problems of sampling and diagnosis in the major twin studies of schizophrenic twins. *Journal of Psychiatric Research,* 2:116–134, 1962.

Rosenthal, D.; Wender, P.H.; Kety, S.S.; Schulsinger, F.; Welner, J.; and Østergard, L. Schizophrenics' offspring reared in adoptive homes. In: Rosenthal, D., and Kety, S.S., eds. *The Transmission of Schizophrenia.* Oxford: Pergamon Press Ltd., 1968. pp. 377–391.

Scheper-Hughes, N. *Saints, Scholars, and Schizophrenics: Mental Illness in Rural Ireland.* Berkeley: University of California Press, 1979.

Schubart, C.; Krumm, B.; Biehl, H.; and Schwarz, R. "The Measurement of Disability in a Schizophrenic Patient Group—Definition, Assessment and Outcomes Over Two Years." Presented at the 1982 Symposium of the Section of Epidemiology and Community Psychiatry of the World Psychiatric Association on the Chronically Mentally Ill, Baltimore, May 10–13, 1982.

Shields, J. Some recent developments in psychiatric genetics. *Archiv fur Psychiatrie und Nervenkrankheiten,* 220:347–360, 1975.

Slater, E. *Psychotic and Neurotic Illness in Twins.* Medical Research Council, Special Reports Series, No. 278. London: Her Majesty's Stationery Office, 1953.

Stein, L. "Social class" gradient in schizophrenia. *British Journal of Preventive and Social Medicine,* 11:181–195, 1957.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia. *Archives of General Psychiatry,* 34:159–163, 1977.

Tansella, M.; Faccincani, C.; Mignolli, G.; Balestrieri, M.; and Zimmermann-Tansella, C. Il registro psichiatrico di Verona-Sud. Epidemiologia per la valutazione dei nuovi servizi territoriali. In: Tansella, M., ed. *L'Approcio Epidemiologico in Psychiatria.* Torino: Boringhieri, 1985. pp. 225–259.

Tienari, P. Psychiatric illnesses in identical twins. *Acta Psychiatrica Scandinavica,* Suppl. 171, 1963.

Tienari, P. Schizophrenia in Finnish male twins. In: Lader, M.H., ed. *Studies of Schizophrenia.* (British Journal of Psychiatry, Special Publication No. 10) Ashford, Kent, England: Headley Brothers Ltd., 1975. pp. 29–35.

Tienari, P.; Lahti, I.; Naarala, M.; Sorri, A.; and Väisänen, E. Schizophrenics' offspring reared in adoptive homes: A family-dynamic study. In: Obiols, J.; Ballus, C.; Gonzalez Monclus, E.; and Pujol, J., eds. *Biological Psychiatry Today.* Amsterdam: Elsevier/North-Holland Biomedical Press, 1979. pp. 27–32.

Tooth, G.C., and Brooke, E.M. Trends in the mental hospital population. *Lancet*, I:710-713, 1961.

Torre, E.; Marinoni, A.; and Allegri, G. Lomest psychiatric case register: Old and new long-stay patients. *Social Psychiatry*, 17:125-131, 1982.

Tsuang, M.T., and Dempsey, G.M. Long-term outcome of major psychoses: II. Schizoaffective disorder compared with schizophrenia, affective disorders, and a surgical control group. *Archives of General Psychiatry*, 36:1302-1304, 1979.

Tsuang, M.T.; Woolson, R.F.; and Fleming, J.A. Long-term outcome of major psychoses: I. Schizophrenia and affective disorders compared with psychiatrically symptom-free surgical conditions. *Archives of General Psychiatry*, 36:1295-1301, 1979.

Ugelstad, E. *Psykiatriske Langtidspasienter i Psykiatriske Sykehus—Nye Behandlingsforsøk*. Oslo: Universitetsforlaget, 1978.

Vaughn, C.E., and Leff, J. The influence of family and social factors on the course of psychiatric illness: A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129:125-137, 1976.

Walsh, D.; O'Hare, A.; Blake, B.; Holpenny, J.V.; and O'Brian, P.F. The treated prevalence of mental illness in the Republic of Ireland—The three county case register study. *Psychological Medicine*, 10:465-470, 1980.

Warthen, F.J.; Klee, G.C., Bahn, A.K.; and Gorwitz, K. *Diagnosed Schizophrenia in Maryland. Psychiatric Research Report 22*. Washington, DC: American Psychiatric Association, 1967.

Weeke, A.; Kastrup, M.; and Dupont, A. Long-stay patients in Danish psychiatric hospitals. *Psychological Medicine*, 9:551-556, 1979.

Weeke, A., and Stromgren, E. Fifteen years later: A comparison of patients in Danish psychiatric institutions in 1957, 1962, 1967, and 1972. *Acta Psychiatrica Scandinavica*, 57:129-144, 1978.

Welner, J., and Stromgren, E. Clinical and genetic studies on benign schizophreniform psychoses based on a follow-up. *Acta Psychiatrica Scandinavica*, 57:129-144, 1958.

Weyerer, S.; Dilling, H.; Kohl, R.; and Martens, H. Social class and mental disorders. *Social Psychiatry*, 17:133-141, 1982.

Wing, J.K., ed. Long-term community care: Experience in a London borough. *Psychological Medicine*, Suppl. 2, 1982.

Wing, J.K., and Bransby, E.R. *Psychiatric Case Register*. (Department of Health and Social Security, Statistical Report Series, No. 8) London: Her Majesty's Stationery Office, 1970.

Wing, J.K., and Brown, G.W. *Institutionalism and Schizophrenia: A Comparative Study of Three Mental Hospitals 1960-1968*. Cambridge: University Press, 1970.

Wing, J.K., and Fryers, T., eds. *Psychiatric Services in Camberwell and Salford: Statistics From the Camberwell and Salford Psychiatric Registers, 1964-1974*. Medical Research Council Social Psychiatry Unit, London, and Department of Community Medicine, University of Manchester, 1976.

Wing, J.K., and Hailey, A.M. *Evaluating a Community Psychiatric Service*. London: Oxford University Press, 1972.

Wing, J.K., and Olsen, R., eds. *Community Care for the Mentally Disabled*. Oxford: Oxford University Press, 1979.

Wing, J.K.; Wing, L.; and Hailey, A. The use of case registers in evaluating and planning psychiatric services. In: Wing, J.K., and Bransby, R., eds. *Psychiatric Case Registers*. (Department of Health and Social Security, Statistical Report Series No. 8) London: Her Majesty's Stationery Office, 1970. pp. 3-8.

Wing, L.; Bramley, C.; Hailey, A.; and Wing, J.K. Camberwell cumulative psychiatric case register: Part I. Aims and methods. *Social Psychiatry*, 3:116-123, 1968.

Wing, L.; Wing, J.K.; Hailey, A.; Bahn, A.K.; Smith, H.E.; and Baldwin, J.A. The use of psychiatric services in three urban areas: An international case register study. *Social Psychiatry*, 2:158-167, 1967.

Wing, L.; Wing, J.K.; Stevens, B.; and Griffiths, D. An epidemiological and experimental evaluation of industrial rehabilitation of chronic psychotic patients in the community. In: Wing, J.K., and Hailey, A.M., eds. *Evaluating a Community Psychiatric Service: The Camberwell Register*. London: Oxford University Press, 1972. pp. 283-308.

World Health Organization, Regional Office for Europe. *The Development of Comprehensive Mental Health Services in the Community*. Report on a conference, Peebles, Scotland, May 24-30, 1972. Copenhagen: EURO 5414 I, 1973.

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Epidemiology of Schizophrenia: A European Perspective

by Assen Jablensky

Abstract

Since its inception, the concept of dementia praecox and, later, of schizophrenia has been one of the most disputed entities in modern medicine. Schizophrenia was, and still is, defined by its clinical symptoms and their characteristic evolution over time. No external validating criteria for the diagnosis have been established, in spite of a host of suggestive biological findings, among which the genetic data carry most weight. This absence of clear-cut substrate markers and indicators underscores the importance of the epidemiological perspective in the study of the disorder. The European contributions to the epidemiological description and understanding of schizophrenic morbidity are numerous. They range from community surveys and studies of pedigrees to case-control designs for assessment of risk factors and long-term followup investigations of course and outcome. This review focuses on epidemiological approaches to schizophrenia that have attempted to highlight the essential attributes of a disease: its incidence and prevalence, ecology, and associated features. It is difficult to generalize about European epidemiological research in schizophrenia, because of the coexistence of a variety of "schools," traditions, and approaches. It is nevertheless possible to discern several clear trends in European epidemiological investigations of schizophrenia that can, to some extent, be contrasted to North American developments.

Both the application of the epidemiologist's main tool—the survey method—to the study of psychiatric morbidity, and the concept of schizophrenia as a group of conditions significantly contributing to morbidity and disability, have their historical origins in Europe (more precisely, in the Swiss canton of Zurich). In 1895, Jenny Koller investigated the occurrence of mental disorders among the relatives of two random samples of subjects (370 persons in each), one consisting of "sane" people drawn from the community, and the other including "mentally ill" individuals who had been hospitalized. Presumably, a large proportion of the probands in her "mentally ill" sample were schizophrenics, but the diagnostic concept of the condition did not appear in its modern format until the publication of Eugen Bleuler's famous monograph in 1911.

In presenting the diagnostic features of the "group of schizophrenias," Bleuler (1911) did not claim to have done more than propose a new name for a condition that had been described earlier by Kraepelin as "dementia praecox." Although Bleuler certainly did far more than just re-name the disorder, the scientific study of schizophrenia began with Emil Kraepelin (1899) who brought together into a single nosological entity three syndromes that had been identified earlier: hebephrenia, catatonia, and dementia paranoides.

Since its inception, the concept of dementia praecox and, later, schizophrenia, has been one of the most disputed diagnostic entities in modern medicine. The controversies that have accompanied it over nearly a century reflect, as a microcosm, the conceptual difficulties that psychiatry encountered in its evolution as a medical discipline and, in particular, the limits of the clinical method on

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which the definition of many of the diagnostic entities in psychiatry still almost exclusively depends.

Schizophrenia was, and still is, defined by its clinical symptoms and their characteristic evolution over time. No external validating criteria for the diagnosis have been established, in spite of a host of suggestive biological findings, among which the genetic data carry most weight. This absence of clear-cut substrate markers and indicators underscores the importance of the epidemiological perspective in the study of the disorder, which alone can provide a bird's eye view of the total "behavior" of the phenomena described as schizophrenic, and thus help in the search for causes, risk factors, and reliable diagnostic tests.

The European contributions to the epidemiological description and understanding of schizophrenic morbidity are numerous. They range from community surveys and studies of pedigrees to case-control designs for assessment of risk factors and long-term followup investigations of course and outcome. A comprehensive review of all these approaches is beyond the scope and purpose of the present article, especially since certain aspects of research have already been covered in a previous issue of *Schizophrenia Bulletin* (Schulsinger 1980), devoted to schizophrenia in Europe. Thus, we leave aside the longitudinal studies of the disorder, the twin, adoptive, and other family studies, and will only briefly mention the research on psychosocial influences on course and outcome. Instead, the present article focuses on those epidemiological approaches to schizophrenia that have attempted to highlight the essential attributes of a disease: its incidence and prevalence, ecology, and associated features.

Historical Trends: Schizophrenia Before Kraepelin

Although the medical literature before the 18th century provides unequivocal descriptions of depressive, manic, hysterical, and organic-dementing clinical pictures, it contains no convincing references to schizophrenic syndromes. Before Torrey (1980) raised the speculative possibility that schizophrenia might be of recent origin, Jaspers (1963) commented that according to the existing sources, schizophrenia was not widespread in the Middle Ages, in contrast to the striking increase in its frequency in the last couple of centuries. A plausible early description of a schizophrenic disorder can be found in Pinel (1803), who referred, under the rubric of *démence ou succession d'idées incoherentes*, to "frequent or continual appearance of unconnected ideas and shallow, dissociated emotions; disorganized movements and extravagant actions . . . a noticeable weakening of the ability to perceive objects . . . a kind of automatic existence. . ." (p. 75). If Pinel's description still leaves room for doubt, the schizophrenic nature of the illnesses in several of the cases described in London by Haslam (1809) can hardly be questioned. Nevertheless, the occasional descriptions of schizophrenic disorders during the early part of the 19th century are completely overshadowed by the dominant notions of "melancholia" and "mania." How is it possible that a condition which in the 20th century was found to have a prevalence in the general population of about 5 per 1,000, and which accounted for over 50 percent of all inmates of mental hospitals, could escape recognition by 18th and early 19th century clinicians?

There are at least two possible explanations. One, recently re-stated by Hare (1983), is that until the second half of the 19th century schizophrenia indeed was a rare disease. Statistical data from the past can be invoked to support this viewpoint. One such source is Parchappe (1839), who published a statistical analysis of the "causes of mental aberrations," based on 575 cases treated at the hospital Saint-Yon in France. If we assume that at the time the two diagnostic categories most likely to contain a large proportion of schizophrenics were the "acute monomaniac insanity" and "chronic insanity," their sum would account for only 18.9 percent of all patients. Half a century later, Tuke (1892) quoted the diagnostic distribution of 14,336 patients admitted to all mental institutions in England in the course of 1 year. By guessing that "ordinary dementia" in his statistics may have camouflaged schizophrenia, we find that the proportion of those cases was 11.1 percent. Tuke also analyzed the first admissions to the asylum in the county of Somerset, where diagnosis was apparently more refined: the percentage of cases of "monomania (delusional insanity)" was only 5.3, while that of "mania" was 42.9 and of "melancholia" 18.4.

If we dismiss the possibility that most of the 19th century schizophrenics were classified into categories other than the ones mentioned above, then the most significant increase of the share of schizophrenia in treated psychiatric morbidity probably occurred early in the 20th century, and not in the last decades of the 19th century. Our own (unpublished) data obtained by screening the records of all the 753 patients admitted in 1908 to the University Psychiatric Clinic in Munich—some of them diagnosed personally by Kraepelin—indicate that as late as 10 years after the establishment of the diagnostic category of dementia praecox, the frequency of this diagnosis among first admissions was as low as 9.1 percent in men and 7.3 percent in women.

The quoted data refer to hospitalized cases; almost nothing is known about the frequency of untreated cases in the community during that period. However, it would appear justified to consider the relative rarity of schizophrenia among the institutionalized populations observed by the "alienists" as an indicator of its lower prevalence in the 19th century compared to more recent periods.

Indeed, if the epidemiological study of historical trends in the evolution of schizophrenia could prove that a dramatic increase in its incidence occurred in the span of several decades, the implications would be far-reaching; careful reconstruction of the circumstances in which this occurred might bring us closer to understanding the disorder's etiology and pathogenesis. The evidence, however, is not strong enough to support such a conclusion, and alternative explanations of the late arrival of the disease concept of schizophrenia must continue to be sought.

A second possible explanation is that schizophrenia did exist and was frequent, but it was assimilated with other psychiatric syndromes, "mania" and "melancholia" in particular. It should be recalled that for most of the 19th century diagnosis in psychiatry was symptom-oriented (the "dementias," "monomanias," and "deliria" were the psychiatric equivalents of the "fevers" in somatic medicine), while classification aimed to be etiological. For decades German psychiatry was dominated by the argument between *die Somatiker* (giving preponderance to physical causation) and *die Psychiker* (emphasizing the "moral causes"). Meynert (1884) proposed a classification in which the following distinctions were made:

- Clinical forms due to anatomical damage to the skull and brain during pregnancy, birth, or infancy (idiocy, surdomutism).
- Focal or massive brain damage as in tumors, hemorrhages, sclerosis, or syphilis (delirium, paralysis, and organic dementia).
- Diffuse changes such as atrophy, hypertrophy, and meningitis (senile dementia, epilepsy, and progressive paralysis).
- Disturbances of nutrition causing cortical excitation (mania, melancholia, or exalted ideas).
- Localized subcortical excitation and exhaustion (delusions, hallucinations, psychic stupor, hypochondriasis, hysteria, partial insanity, or persecution mania).
- Disturbances of the subcortical vascular centers (epilepsy, hysteroepilepsy, circular insanity, ascendent paralysis, or cretinism).

It is hardly possible to discern in this classification the nosological entities and syndromes with which we operate today, and which we seek to validate etiologically and pathogenetically. Paradoxically, real progress toward the delineation of the "modern" psychiatric taxonomic entities occurred in the 19th century only with the abandonment of the etiological principle of classification, and the focusing of attention primarily on the clinical structure of syndromes and their temporal sequence. This change of paradigm in psychiatric thinking, and the emergence of the nosological approach to diagnosis and classification, occurred largely as a result of the work of Wilhelm Griesinger (1861), whose theory of a clinical continuum of forms of disorders was the prerequisite for the systematic mapping of individual disease entities by Kahlbaum and Kraepelin.

Thus, if the evidence for the apparent rarity of schizophrenic disorders until the beginning of this century is placed in the context of the dramatic change in psychiatric theory and nosology that occurred between the middle and the end of the 19th century, it becomes highly plausible that schizophrenia was not identified earlier because clinicians were seeing reality from a different angle. There was no phenomenological description of schizophrenia as such because the concept did not exist, although all the individual components that later were to be combined into that concept had been there all the time. The second line of argument, of course, does not preclude the possibility that the frequency and the quality of some of the manifestations of schizophrenia may also have changed over time—indeed, it would be surprising if they had not. The consideration of the impact of psychopathological theory and clinical practice on empirical observation in psychiatry would, however, caution us against the sweeping conclusion that schizophrenia is a "new" disease.

Social and other nosocomial factors may also have played a role. Diminished tolerance of communities and the new image of the hospital as a place where scientific diagnosis and treatment is practiced may have combined their effects to facilitate the process of delineating new clinical entities (Cooper and Sartorius 1977). Table 1 demonstrates clearly that the rate of increase in the proportion of institutionalized psychiatric patients far outstripped the rate of increase of

Table 1. Increase of the mentally ill in Austria, 1873-1905

Year	"Insane persons" (*n*)	Rate per 1,000	Psychiatric Institutions (*n*)	Institutionalized patients (%)
1873	18,344	0.94	23	32.5
1880	26,197	1.18	27	27.9
1890	27,786	1.22	30	47.4
1894	29,706	1.21	37	51.1
1896	30,246	1.28	39	52.9
1900	32,106	1.24	39	57.4
1905	36,612	1.35	41	61.9

Source: *Osterreichisches statistisches Handbuch* (1898-1907).

administrative prevalence. The unprecedented concentration of psychotic individuals in medical establishments undoubtedly made it easier for clinicians to identify syndromes, subtypes, and other varieties of mental disorders.

Variations in the Concept of Schizophrenia in Europe

Unlike the epidemiological study of simple characteristics like blood pressure or weight, or of disorders like myocardial infarction for which established diagnostic indicators exist, research on schizophrenia has to rely on a complex set of inferences from a multitude of reported and observed facts, none of which singly is either necessary or sufficient to establish the diagnosis.

Kahlbaum's (1874) notion of the nosological entity was a prerequisite for the formulation of the concept of schizophrenia. This notion postulated a close correspondence between etiology, cerebral pathology, symptom pattern, and outcome as a basis for assuming that various clinical states can be brought together under the single rubric of a disease. This theory enabled Kraepelin to combine several clinical syndromes into the nosological entities of dementia praecox and manic-depressive insanity. Kraepelin's achievement was marked from the beginning by two features that were destined to become the kernels of much subsequent controversy. First, among the several criteria defining the nosological entity, the criterion of outcome was given a special dominant role in the definition of dementia praecox and manic-depressive psychosis. Second, outcome was defined mainly in terms of "curability" versus "incurability." For Kraepelin, the use of the criterion of outcome as a cornerstone of the nosology of psychoses was largely a matter of expediency: the reference to etiology or brain pathology was of no help in these disorders, while outcome was observable and could be described with considerable refinement.

For a good part of his life, Kraepelin hoped that experimental research would ultimately confirm the validity of course and outcome as nosological criteria and demonstrate discrete biological factors underlying the different patterns of evolution of syndromes. This view, however, was challenged within German psychiatry and, under the influence of his critics, Kraepelin adopted in his later years a radically different position, according to which schizophrenia was a biopsychological reaction type that could be activated by a variety of etiological factors (Kraepelin 1920). Developments in German psychiatry have tended to polarize around two divergent points of view: one postulating specific biological causation for every distinct clinical entity (Kleist 1928; Leonhard 1957), and another emphasizing the continuities among the forms of manifestation of the functional psychoses, and attempting to identify common structures or dimensions (Bonhoeffer 1912). Neither of these different theoretical viewpoints, however, had much impact on the diagnosis of schizophrenia in clinical practice, and it can be assumed that most of the German epidemiological work in schizophrenia has been based on a middle-of-the-road diagnostic concept, close to the original view of Kraepelin and that of Bleuler.

British psychiatry has adopted a position close to the classical German views on the diagnosis and nosology of schizophrenia (Mayer-Gross, Slater, and Roth 1969; Kendell 1981; Berner and Küfferle 1982). The need for explicit criteria and agreement on a standard classification has, perhaps, been emphasized in Britain more than elsewhere in Europe, and it is no coincidence that prominent British psychiatrists like A. Lewis took an active role in the development of the first psychiatric glossary to accompany the 8th revision of the *International Classification of Diseases* (World Health Organization 1974).

Scandinavian psychiatry, which has made eminent contributions to epidemiology, has traditionally adhered to a "narrow" concept of schizophrenia (more restrictive than the German one), excluding from its scope not only cases with an admixture of affective features and acute onset (most of which could be classified as schizophreniform psychoses; see Langfeldt 1939) but also cases of unequivocal paranoid manifestation in which a link could be inferred between the onset and content of symptoms and a psychotraumatic experience. Such cases are classified as psychogenic or reactive psychoses (Wimmer 1916; Faergeman 1963; Strömgren 1968), and they continue to represent a substantial proportion of all diagnoses made, although there appears to have been a decline in their frequency in recent years.

French psychiatry has resisted the influence of Kraepelin's nosological view of dementia praecox from the very beginning (Pichot 1984) and developed a taxonomy of its own in which the term "schizophrenia" is reserved for only one of the groups of syndromes comprised in the German notion: that characterized by profound and chronic personality disorganization. Acute schizophrenia, therefore, is a contradiction in terms for most French psychiatrists. On the other hand, chronic delusional psychoses of systematized content in which the personality is relatively preserved have an independent classificatory status.

Russian, and subsequently Soviet, psychiatry has always regarded schizophrenia as a disease entity in the Kraepelinian sense. Much of the Soviet epidemiological research in schizophrenia has evolved in the conceptual framework of the school of Snezhnevsky (1972, 1983), which developed a classification of the forms of the disorder giving particular weight to the pattern of course: continuous, recurrent, and shift-like. Subtypes based on grades of severity and progression of negative disorders are distinguished within each form, and a number of studies have been devoted to establishing correlations between such longitudinal features and the presenting clinical syndromes and symptoms. The scope of the diagnostic category of schizophrenia is somewhat wider in comparison to other European diagnostic systems (Snezhnevsky and Nadzharov 1985), mainly because of inclusion of conditions that would be classified as atypical affective psychoses and paranoid states elsewhere. Since, however, Soviet epidemiological studies as a rule report the form and subtype of conditions along with data on their frequency, adjustments for purposes of comparison are possible.

This brief overview of the most influential "schools" within European psychiatry indicates considerable variation in the approaches to the taxonomy of schizophrenia and its delimitation from other disorders. However, the extent to which such variation vitiates comparisons of epidemiological data should not be exaggerated. The differences between "schools" primarily concern the margins of the diagnostic concept, not its core. Such differences are likely to affect the reliability of estimates of the upper limits of the range of morbidity indices, but would have less effect on the lower limits. The basic comparability of different European notions of schizophrenia is amply illustrated by several diagnostic studies of British, French, German, Swiss, and Soviet concepts (Baumann et al. 1976; Seabra-Dines 1977; Busch et al. 1980) which indicate good agreement on "typical" or "core" cases of schizophrenia, in contrast to cases exhibiting borderline features, which give rise to marked diagnostic discrepancies (Shepherd et al. 1968).

Within most of Europe, however, the differences in the diagnostic interpretation of the concept of schizophrenia are nowhere near those that existed between Britain and the United States before the advent of *DSM-III* (Cooper et al. 1972). Most European psychiatrists would probably agree fairly easily on a "core" concept of schizophrenia that is based on Bleuler's fundamental and accessory symptoms (Sartorius et al. 1978), and discrepancies over the classification of patients are likely to arise with regard to atypical syndromes exhibiting various mixtures of affective and paranoid symptoms, neurotic features, or late age of onset.

Much can be learned, therefore, from European epidemiological research in schizophrenia, provided that the existence of certain conceptual differences between countries and "schools" is kept in mind. It is another matter that the degree of explicitness of the diagnostic criteria employed in past studies leaves much to be desired in the light of recent developments concerning the operationalization of such criteria. A great advantage of European research lies in the fact that field investigations are usually carried out by highly qualified professionals and, in quite a few instances (as, for example, mos

Scandinavian studies), with a remarkable meticulousness and completeness of coverage.

Since the design, instruments, and other methodological aspects of European epidemiological surveys of mental disorders have been reviewed elsewhere (Jablensky and Hugler 1982; Jablensky, in press), the following sections will focus primarily on substantive findings that tell us something about the nature and spread of schizophrenic disorders in Europe.

Prevalence, Incidence, and Morbid Risk

A first and essential step in the epidemiological study of a disease is to map out the frequency of its occurrence in the two sexes and different age groups, the geographical distribution and density of cases, and the associations of the condition with other disorders and various environmental factors. The detailed and valid description of the general epidemiological "behavior" of a disease may on occasions directly suggest clues to putative causes and determinants of pathogenesis. In the ideal case, this may lead to experimentally testable hypotheses and the ultimate demonstration of etiology—vide the celebrated example of the discovery of the causes of pellagra by Goldberger (Terris 1964) and the more recent one of kuru (Gajdusek and Zigas 1959).

Schizophrenia, however, has proved a refractory object for epidemiological study, and its epidemiological map worldwide still contains much uncharted space. Among the many reasons for this unsatisfactory state of affairs, the following are of particular importance because they are linked to the nature of the condition: (1) Schizophrenic disorders show a marked polymorphism of clinical manifestations and lack pathognomonic or sufficiently specific symptoms and signs. It is possible that the majority of the carriers of a "schizophrenic" genotype in the population are either asymptomatic or manifest only subclinical and atypical ("spectrum") traits and states. (2) The course of schizophrenia is varied and difficult to predict. In many instances, its onset is indistinguishable from a progressive aggravation or accentuation of traits of the premorbid personality. (3) Notwithstanding the intensive search for disease markers in recent years, no reliable indicators are yet available to facilitate rapid diagnostic screening of population samples, as is the case in some other diseases.

In addition to these obstacles, the dictum that most schizophrenics will sooner or later present themselves in the hospital (Ødegard 1952) is of less certain applicability in recent years than previously. Today, more and more patients are maintained in the community by general practitioners, psychiatric outpatient services, or alternative sources of social support, including "counter-culture" cults, live-in groups, and other niches for the eccentric and unconventional. All these factors tend to make epidemiological research in schizophrenia costly and complex. To them, of course, should be added the lack of agreed-upon uniform diagnostic criteria, insufficient standardization of research instruments, and various constraints on the intensity of case-finding procedures.

Prevalence (point, period, or lifetime) is defined as the number of all cases (per 1,000 persons at risk) present in the population at a given time or over a defined period. If individual lifetime is taken as base, the meaning of the prevalence index approximates that of morbid risk. Point prevalence, which is the most often determined measure of the frequency of schizophrenia, is useful for estimations of service needs, the extent of disability in the community and other social costs of the illness. It is, however, less suited to etiological research and comparisons between groups or populations because of its dependence on demographic factors such as mortality and age structure.

Incidence, i.e., the number of new cases or inceptions of a disease in a given population over a specified period (usually 1 year) per 1,000 persons at risk, is a particularly valuable index in the study of schizophrenia, in view of the possibility that the primary pathogenetic and triggering factors may be active in the period preceding the manifest onset of the disorder but cease to operate at later stages of its evolution. Therefore, the epidemiological search for significant associations between onset and proximal environmental and personal antecedents is a promising research strategy.

Morbid risk (or disease expectancy) is an index giving the actuarial probability of developing a disease, provided that a given individual survives until a specified age. Morbid risk is less dependent on differential mortality in the population and can be used in comparisons across groups and countries. The index of risk can be determined directly, by summing up the age-specific incidence figures (if such data are available), or indirectly, using an estimation method first proposed in Europe by Weinberg (1925) which assumes that the point prevalence of the condition and the age distribution of the population are known.

Four principal methods have been used in Europe to determine the prevalence and incidence of schizophrenia.

The first is the so-called genealogical random sample test (*genealogischer Stichprobentest*), introduced in Germany by Rüdin (1916) and used mainly in the 1920s and 1930s (Graemiger 1931; Panse 1936). In essence, this method requires the collection of psychiatric morbidity data on randomly selected probands and their biological relatives. The German genetic epidemiologists between the two world wars chose their random samples mainly among patients hospitalized because of somatic disease, assuming that the frequency of psychoses in their pedigrees would correspond to that in the population at large. The results of a number of such investigations turned out to be very similar and became accepted for some time as standard figures of the prevalence and morbid risk of schizophrenia. In fact, however, the absolute number of cases of schizophrenia detected in such samples was very small (of the order of two-three cases) and the generalization of the risk estimates was recognized later on to be misleading (Strömgren 1950). The technique was also criticized for the questionable assumption that series of hospitalized somatic patients could be representative of the general population.

The second method is the *birth cohort* study, first implemented by Klemperer (1933). He took a random sample ($x = 1,000$) of all individuals born during the decade 1881–90 in the city of Munich and attempted to trace them as adults in their fourth decade of life. He was able, however, to trace only 44 percent and to carry out interviews with 271 probands or key informants. The limited success of this survey exposed some of the intrinsic difficulties of the birth cohort method, such as the high attrition rate. Nevertheless, the same method produced remarkable results when applied to captive island populations in Denmark (Fremming 1947) and Iceland (Helgason 1964). Fremming succeeded in tracing 92 percent (and in examining most of them personally) out of a birth cohort of 5,500 people born between 1883 and 1887. Helgason traced as many as 99.4 percent of all those born in Iceland in 1895–97 who had been alive in 1910. The morbid risk figures determined from these two latter surveys are therefore more reliable than Klemperer's.

The third method is the *population census survey* which can be exhaustive and cover the totality of a geographically delimited population by door-to-door interviewing, or representative, based on a multi-stage or stratified sample taken from a large population. The door-to-door method was used by Brugger (1931, 1933) in community surveys in Thuringia and Bavaria, and later by Strömgren (1938), Sjögren (1948), Mayer-Gross (1948), Bremer (1951), Böök (1953), Larsson and Sjögren (1954), Essen-Möller et al. (1956), Andersen (1975), and Rotstein (1977). A particular contribution of European epidemiology is the *repeat* survey, in which the same population is reexamined after a certain number of years, using identical or comparable methods. Thus, Hagnell (1966) and Hagnell and Ojesjö (1975) carried out two consecutive reexaminations of the rural community originally surveyed by Essen-Möller, and Bjarnar, Reppesgaard, and Astrup (1975) investigated a population initially studied by Bremer. Böök, Wetterberg, and Modrzewska (1978) also revisited, after an interval of 20 years, the genetic isolate in northern Sweden that became known for its unusually high rate of psychosis, and carried out some biochemical investigations on a sample of pedigrees, as an addition to the repeat census of mental disorders. A good example of a sample survey is the two-stage investigation of a large cluster sample ($n = 9,201$) of households in an area in Croatia, Yugoslavia, characterized by a high prevalence rate for schizophrenia and other psychoses (Crocetti et al. 1971).

The fourth method consists of the monitoring of *first admissions* or any other first contacts that patients with psychotic disorders make with various services. This method is more economical if the universe of study is a large population, because it relies on already "declared" cases and uses the existing services as a case-finding network. Its limitations, however, stem from the uncontrollable consistency of the diagnostic procedures applied by many different diagnosticians, and from the unknown number of cases that either do not seek treatment or attend facilities outside the study area. However, there is no a priori reason why incidence or prevalence data obtained with this method—provided that the population under surveillance is large—should be less valid than those collected in door-to-door surveys of small communities. A research tool which significantly facilitates studies of declared morbidity is the *cumulative case register*. In countries like Norway and Denmark, such registers have existed since the 1920s and contain a wealth of data that have made possible some unique studies on psychiatric morbidity and its various associations. Similarly, in the U.S.S.R. and some East European countries, the cumulative patient list of the *area psychiatric dispensary* offers convenient facilities for epidemiological research. Most of the epidemiological studies of schizophrenia in Moscow are based on large cohorts, totaling several thousand cases, accumulated over time in the registers of the district dispensaries. Incidence is estimated by means of a retrospective search for the recorded year of onset, and the annual number of onsets is divided by the figure for the population at risk, taken from the population census that is nearest to that year.

A modification of the first contact method of monitoring the incidence of psychotic conditions was developed in the context of a recent collaborative study initiated and coordinated by the World Health Organization (to be published). The study involves 12 research centers in 10 countries worldwide, of which 5 (Denmark, Czechoslovakia, Ireland, United Kingdom, and U.S.S.R.) are European. Case finding was prospective over 2 or more years and was accomplished by close monitoring of a number of preselected "intercept points" in the community that included not only the psychiatric facilities but also other medical services and social agencies with which schizophrenic patients were thought likely to make a first-in-lifetime contact. Each potential case identified through this network was given a full clinical and social examination, using standardized instruments.

None of the existing methods of determining the incidence and prevalence have the capacity of entirely overcoming two of the methodological difficulties that accompany the study of a condition as protean in its manifestations as schizophrenia: the uncertainty in pinpointing onset and the fact that even in settings with well-developed services some of the cases never come to the attention of the helping agencies. As regards onset, a realistic strategy ensuring some consistency—until reliable disease markers are established—is the use of a well-defined event, such as the first contact with a service or the date on which a diagnosis is first made, as a reference point. With regard to the cases who never consult, estimates of their frequency can be derived from published door-to-door surveys, which have identified a certain number of schizophrenics who are well tolerated by the community and have never been hospitalized, in spite of persistent and occasionally florid symptoms. A survey of a random sample of the population aged over 60 in Moscow (Gavrilova 1979) showed that a lifetime diagnosis of a schizophrenic disorder could be made in 1.5 percent of the subjects who had never consulted a psychiatrist. Manifest psychosis, however, was diagnosed in only 0.5 percent; the remainder were classified as abortive, subclinical, or schizophrenia spectrum disorders. The majority of the cases who never consulted were women. It should be noted that the diagnoses in this survey were based on lifetime histories and that overt psychotic symptoms were present at the time of the interview in only 2 (0.1 percent) out of the 23 cases. If such data are indicative of the extent of hidden schizophrenic morbidity in the community, it would be justified to conclude that in settings where well-developed psychiatric services exist, as in the majority of European countries, the rate of undiagnosed psychotic forms of schizophrenia is a fraction of the rate of diagnosed cases, and certainly does not represent the larger, invisible part of the epidemiological "iceberg."

The principal results of a number of European studies on the frequency of schizophrenia are presented in table 2. As already pointed out, the uncertain comparability of the diagnostic criteria for schizophrenia across countries and across individual studies should call for caution when numerical results of different surveys are compared.

With this caveat, the data summarized in table 2 nevertheless reveal considerable similarities. Regarding the point prevalence of schizophrenia, most European studies indicate rates in the range of 2.5 to 5.3 per 1,000. Somewhat lower rates were found in the early surveys of Brugger (1931, 1933) and, more recently, in Dilling and Weyerer's (1980) studies in Bavaria, presumably because patients in institutions outside the study areas were missed. Higher than average prevalence rates were found by Essen-Möller (1956) in South Sweden, but particularly by Böök (1953, 1978) in a genetic isolate with a population around 6,000 in northern Sweden. The morbid risk in that population was estimated to be 2.68 percent for males and 2.27 percent for females, while the point prevalence (9.5 per 1,000) was three times as high as the average for Sweden. The population of the area shows an increased rate of consanguinity. All cases of psychosis have been traced back to three related "founder" families which migrated into the area from Finland in the 17th century. According to Böök, most of the illnesses in that population meet Bleuler's description of "fundamental" symptoms, but have an acute onset and display a predominance of catatonic disturbances. These features raise some doubts as to their classification as "nuclear" or "core" schizophrenic disorders, a reservation important both with regard to generalizations about the mode of inheritance (two-gene, dominant/recessive) and the interpretation of the more recent

Table 2. Occurrence of schizophrenic disorders in geographically defined populations (data from 26 surveys in Europe)

Author	Country	Point prevalence (per 1,000)	Incidence (per 1,000)	Morbid risk (per 100)	Remarks
Birth cohort studies					
Klemperer (1933)	Germany	10.0		1.40	Estimated with correction for cohort attrition
Fremming (1947)	Denmark			0.90	
Helgason (1964)	Iceland			0.57–0.69 (M) 0.90–1.02 (F)	Up to age 61
Census studies					
Brugger (1931)	Germany	2.4 (1.9)		0.38	Per 1,000, aged 10 + Per 1,000, all ages
Brugger (1933)	Germany	2.2		0.41	Per 1,000, all ages
Brugger (1938)	Germany	2.3 (1.8)		0.36	Per 1,000, aged 10 +
Strömgren (1938)	Denmark			0.58	Per 1,000, all ages
Sjögren (1948) and Larssen & Sjögren (1954)	Sweden	4.6		1.60	Equal for M and F
Böök (1953)	Sweden	9.5		2.66	Age 15–50
Böök, Wetterberg, & Modrzewska (1978)	Same population	17.0		2.68 (M) 2.27 (F)	Genetic isolate
Essen-Möller et al. (1956)	Sweden	6.7			Psychotic on census date
Hagnell (1966)	Same population	4.5			
Crocetti et al. (1971)	Yugoslavia	5.9			Area known for high rate of psychosis
Lehtinen et al. (1978)	Finland	15.0			
Rotstein (1977)	Sample U.S.S.R.	3.8			
Service contact studies					
Ødegard (1946)	Norway		0.24	1.87	Age 10 +
Norris (1959)	United Kingdom		0.17		

Table 2. Occurrence of schizophrenic disorders in geographically defined populations (data from 26 surveys in Europe)—*Continued*

Author	Country	Point prevalence (per 1,000)	Incidence (per 1,000)	Morbid risk (per 100)	Remarks
Adelstein et al. (1968)	United Kingdom		0.35 (M) 0.26 (F)		Age 15+
Walsh (1969)	Ireland		0.57 (M) 0.46 (F)		Age 10+
Hafner & Reimann (1970)	Fed. Rep. of Germany		0.54		
Lieberman (1974)	U.S.S.R.		0.20 (M) 0.19 (F)		Patient with onset in 1950–64 personally investigated by author
Temkov, Jablensky, & Boyadjieva (1975)	Bulgaria	2.8			
Nielsen (1976)	Denmark	2.7	0.20		Census on 1 Jan. 1964
Ouspenskaya (1978)	U.S.S.R.	5.3			Lifetime prevalence, per 1,000 age 14+
Helgason, L. (1977)	Iceland		0.27	0.43 (M) 0.54 (F)	

findings (Book, Wetterberg, and Modrzewska 1978) implicating monoamine oxidase (MAO) and dopamine-β-hydroxylase (DBH) as possible markers.

Another part of Europe where unusually high rates of schizophrenia have been found is a coastal area in Croatia, Yugoslavia. The area has about 400,000 inhabitants, and high rates of psychoses have been consistently reported for that population. The epidemiological survey carried out in the 1960s (Crocetti et al. 1971) confirmed these observations but established that the excess morbidity was not confined to schizophrenia. Higher than average rates were found for affective psychoses (especially with middle-age onset), as well as for some physical conditions, notably arterial hypertension and psoriasis. No convincing explanation of these raised levels of morbidity has yet been put forward, but effects of selective out-migration from the area over several generations have been considered. Genetic isolate factors can be excluded with certainty.

Ireland, and in particular its western counties, has been known for consistently exhibiting some of the highest prevalence rates of schizophrenia. This has been confirmed, as far as hospitalized morbidity is concerned, by several studies (Walsh 1969; Kelleher, Copeland, and Smith 1974). It has been shown that the rates are not inflated because of broader diagnostic criteria (Kelleher and Copeland 1974). Furthermore, first admission rates of schizophrenia appear to be higher in Dublin than in comparable urban centers in the United Kingdom, including Northern Ireland. These findings await explanation, but it has been noted that the counties with the highest rates of schizophrenia are by and large those most severely stricken by the agricultural catastrophe and the economic crisis of the mid-19th century. The same areas have had the highest rates of emigration in the 19th and the early part of the 20th century (Torrey 1980).

Apart from the above-mentioned World Health Organization (WHO) collaborative epidemiological study, whose results are to be published, few cross-cultural investigations in Europe have applied comparable case-finding and diagnostic methods. One such study, carried out in the U.S.S.R., actually compared the prevalence of schizophrenia in Moscow with that in three other regions in European Russia, in the far north of the country, and in the Uzbek Soviet Republic in Middle Asia. Morbidity was found to be lower in the Uzbek population, and a relatively high proportion of the cases there were characterized by acute onset and schizoaffective features. There were no significant differences between Moscow and the other two areas (Ouspenskaya 1978; Zharikov, Kalachev, and Sokolova 1979).

Most of the European data on the incidence of schizophrenic disorders are based on first admission statistics or on retrospective cohort analysis. The former approach tends to produce somewhat higher rates (presumably because some patients subsequently turn out to be false positives for the diagnosis of schizophrenia), while the latter underestimate the true incidence because of cohort attrition. The data shown in table 2 indicate that the rates vary between 0.17 and 0.57 new cases per 1,000 per year, with the lowest and the highest rates being reported in London and Dublin, respectively. Table 2 also lists a number of methodological differences between the studies quoted which have to be taken into account before conclusions are drawn. Schizophrenia is a disorder of a relatively low incidence, and minor methodological discrepancies between investigations, like the inclusion or noninclusion of a few diagnostically doubtful cases, or differences in the age-group composition, can easily account for seemingly diverging rates. Generally, there is good agreement between the incidence rates for Norway, Denmark, Iceland, and the U.S.S.R., which suggests that the figure 0.2 per 1,000 may be a reasonable estimate of the incidence of schizophrenia in European populations. Except for anecdotal reports, there is no real evidence of a gradient of incidence within Europe, be it from north to south, or from east to west.

In contrast to incidence rates calculated on the basis of service contacts, the estimates of the morbid risk of schizophrenia vary more widely, with a range between 0.36 percent and 1.87 percent for a randomly selected individual from the general population. Even if allowance is made for registration bias, effects of study design, or differences in the methods of computing risk, the variation obtained in European studies deserves attention. The birth-cohort method tends to produce higher risk figures than the census method. However, it is difficult to explain the fourfold difference in the risk estimates for the not too dissimilar populations of Norway and Iceland, obtained in two similarly thorough investigations of comparable design (Ødegård 1946; Helgason 1977). Since the two studies were separated by a period of more than 30 years, the hypothesis that the morbid risk for the disorder may have been on the decrease cannot be dismissed out of hand.

It should be noted that in recent years some authors have discussed the possibility that incidence and prevalence rates of schizophrenia in Europe may be on the decrease. For example, Weeke and Strömgren (1978) have pointed to the fact that the treated schizophrenic morbidity, according to the Danish national psychiatric case register, has shown a decline over the period 1957–72, from 1.32 per 1,000 to 0.97 per 1,000 for males, and from 1.40 per 1,000 to 0.86 per 1,000 for females. Whether this is a real decrease, or an effect of the greater probability for a schizophrenic to be treated by a general practitioner without being reported to the register, remains unknown. In a similar vein, Rotstein (1982) observed a marked drop in the incidence of new cases of paranoid schizophrenia in Moscow in the 1970s, compared to the 1950s and 1960s, but attributed this to the particular method of determining incidence (by retrospective cohort analysis) which underestimated the onsets falling within the most recent of several consecutive periods of observation.

Demography of Schizophrenia: Mortality and Fertility

Mortality and fertility are important variables for the understanding of the population distribution and frequency of a disease. If one assumes that the incidence rate is constant and the average duration of the condition is known, the prevalence will be determined primarily by the absolute and relative mortality of the affected individuals and by the rate of reproduction of those who carry the predisposition for the disease and pass it on to subsequent generations.

In the instance of schizophrenia, the mortality/fertility issue has yet another implication. Twenty-two years ago, Huxley et al. (1964) suggested that the existence of a balanced polymorphism might be the explanation for the apparently stable incidence and continued existence of schizophrenia over time. This means that the diminished reproductive fitness conferred by the "pathogenic" form of the gene must be compensated for by an increase in reproductive fitness associated with the nonpathological form of the gene. Heterozygous carriers therefore would be expected to have lower mortality, higher fertility, or both.

The balanced polymorphism hypothesis has never been properly tested because prohibitively large population samples would be required to detect even a small effect. Nevertheless, information about mortality and fertility rates of schizophrenic individuals is useful for establishing a data base on the issue.

Some of the most extensive European data on the mortality of psychiatric patients, including schizophrenics, have been collected in Norway, where Saugstad and Ødegård (1979) analyzed the psychiatric case register data for a period of 25 years (1950–74) and compared them with earlier data covering the period 1926–41. While for the totality of hospitalized psychiatric patients there was a clear and significant decrease in mortality (mainly accounted for by the drop in deaths of patients with organic and symptomatic psychoses), the relative mortality of schizophrenics was unchanged and remained higher than that of the general population (table 3). The age-standardized mortality coefficient for the period 1969–74 was 21 per 1,000 for males and 15 per 1,000 for females (higher than in affective psychoses, where it was 17 per 1,000 for males and 11 per 1,000 for females). Similar relative mortality rates of schizophrenic patients, i.e., about twice the rate for the general population, have been reported by Herrman, Baldwin, and Christie (1983) on the basis of a record linkage study in the Oxford area in England.

The relationship between a diagnosis of schizophrenia and fertility has been investigated predominantly in series of women; relatively little is known about the fertility of schizophrenic men. The detailed data collected by Essen-Möller (1935) in Sweden pointed to a significantly lowered fertility of schizophrenic women. More recent data have been published by Shmaonova et al. (1976) and by Ødegård (1980). The study of Shmaonova et al. included a sample of 901 women with a diagnosis of schizophrenia and 230 women with a diagnosis of manic-depressive illness in Moscow. Ødegård's data are derived from the Norwegian national case register and comprise all the 30,428 women with mental disorders who were hospitalized in the period 1936–75. In both studies the summary birth rates were determined, i.e., average number of live births over the entire reproductive period. Table 4 shows that the two studies produced very similar results, indicating moderately reduced fertility rates in schizophrenic women, compared to the general population. Patients with affective psychosis consistently showed higher fertility rates than the schizophrenics, but the differences were not of a magnitude that would suggest the operation of underlying biological causes. Both Ødegård and Shmaonova et al. emphasize the social factors limiting the reproductive changes of schizophrenic patients and point out that their fertility rate is showing a slight upward trend in the last decades, owing to the increasing deinstitutionalization of the treatment of the disorder.

At least one study, which was part of the joint American-Danish project on adopted offspring of schizophrenics (Rimmer and Jacobsen 1976), examined the fertility of siblings of schizophrenic probands. No difference was found in comparison to the control population, and it was concluded that there is no evidence of a reproductive advantage for the nonpsychotic first degree relatives of schizophrenic patients. The issue of a balanced polymorphism in schizophrenia has not been resolved, however, because even small but consistent reproductive advantages in more distant relatives of the probands (e.g., aunts, uncles, or cousins) could equilibrate the transmission of schizophrenia in the population.

Table 3. Relative mortality rate of patients with a diagnosis of schizophrenia in Norway, 1926–74 [1]

	Period			
	1926–41	1950–62	1963–68	1969–74
Males	3.2	1.8	1.8	2.0
Females	4.8	2.3	2.1	2.3

[1] From Sangstad & Ødegård (1979).

Table 4. Summary fertility rates of schizophrenic women and women in the general population [1]

Fertility rates	U.S.S.R.	Norway
Fertility of schizophrenic women	1.54	1.80
Fertility of women in the general population	2.01	2.21

[1] From Shmaonova et al. (1979); Ødegård (1980).

Social Ecology of Schizophrenia

Although European epidemiological research into schizophrenia never went through a phase of sociological theory-building comparable to the period dominated by the social ecology and social stratification research in the United States, the ideas about social causation and social drift in schizophrenia did exert some influence. In Britain, Harris et al. (1956) established the existence of intrageneration downward social drift for patients whose fathers belonged to either social class I or social class II. Cooper (1961) found a significant relation between social class and social prognosis (especially the outcome of rehabilitation) in a series of 224 schizophrenic patients. A number of studies in Britain, Finland, Federal Republic of Germany, and other countries found a social class gradient in relation to the general population, both in hospitalized schizophrenic patients and in the spatial distribution of cases in urban agglomerations. The interest in social causation hypotheses, however, waned following the publication of findings (Goldberg and Morrison 1963) indicating clearly that the social class distribution of the occupations of fathers of schizophrenic patients did not differ from that of the general population. As a result, the downward drift hypothesis gained favor, and it was indeed shown that the decline in occupational status and social prestige in schizophrenics mainly preceded the onset of manifest psychotic symptoms, i.e., it was related to premorbid personality and behavioral patterns.

Another study which put in question the alleged role of macrosocial stresses in the causation of schizophrenic disorders was carried out by Noreik and Ødegård (1967), who compared the age at onset for a total of 38,414 first admissions for functional psychoses in Norway in two periods: 1931–35, a time of serious economic crisis with high unemployment rates, and 1951–55, a period characterized by stable socioeconomic conditions and a well-developed system of social welfare. The hypothesis was that if social stress had a causal or triggering role on schizophrenic conditions, it would advance the outbreak of psychosis toward an earlier age. However, no significant change was found in the age at onset between the two periods, and the investigators concluded that stresses resulting from macrosocietal changes were unlikely to be a major contributor to the causation of schizophrenia. Instead, they suggested that "if we want to relate schizophrenia to social conditions, we shall have to concentrate upon the more personal sides of the social situation and upon the nearest social milieu: the family" (p. 247). This indeed has happened, and over the last two decades much of the European research on social determinants in schizophrenia has been focused on the impact of life stresses of a personal nature, e.g., stressful life events (Birley and Brown 1970), or on aspects of social interaction and communication within the family unit (Brown, Birley, and Wing 1972) in relation to the risk of relapse and the outcome of treatment. This work, which has important practical implications for the design of treatment and rehabilitation programs for patients with schizophrenic disorders, has been extensively reviewed (e.g., Leff 1976).

Unusual Epidemiological Findings: Seasonality of Births

One of the most consistent and yet quite puzzling epidemiological findings in schizophrenia is the seasonality in the distribution of births of persons who subsequently develop schizophrenia. The seasonality phenomenon has attracted considerable attention in recent years, but the first observations of its existence date back to the 1920s. Tramer (1929) was the first author to publish data on the season of birth of 3,100 schizophrenic patients in Switzerland. He was followed soon by Lang (1931), who reported on the months of birth of 3,976 schizophrenics in Germany. In the last two decades, data on seasonality of birth in schizophrenic patients in Europe have been reported by Hare and Price (1968), Videbech, Weeke, and Dupont (1974), O'Hare, Walsh, and Torrey (1980), and Shur and Hare (1983). The main findings are summarized in table 5. All reports indicate that the annual curve of the distribution of months of birth of schizophrenics has a high peak in winter and early spring. This is different from the typical birth distribution curve for most European populations, which shows a less marked peak in spring, another small

Table 5. Seasonality of births of schizophrenic patients in Europe

Author	Country	Samples studied	Seasonal peak of births	Difference from controls
Hare & Price (1968)	United Kingdom	(1) 3,596 patients with schizophrenia (2) 11,076 patients with neuroses	January-April	$p < .02$
Videbech, Weeke, & Dupont (1974)	Denmark	(1) 7,427 patients with schizophrenia (2) 8,212 patients with affective psychoses (3) General population	January-April	$p < .05$
O'Hare, Walsh, & Torrey (1980)	Ireland	(1) 4,855 patients with schizophrenia (2) General population	April-June	11% excess
Shur & Hare (1983)	United Kingdom	(1) 11,728 patients with schizophrenia (2) General population	January-April	$p < .05$

peak in autumn, and a minimum in late autumn and winter. The difference between the winter-early spring peak in schizophrenic births and the spring peak in the general population is statistically significant and amounts to 5-11 percent. Comparable seasonality has not been found in series of patients with other psychiatric disorders, and the phenomenon may be characteristic of schizophrenia.

The European data should be examined in conjunction with analogous data from other geographical regions. Reports from North America (Torrey, Torrey, and Peterson 1977; Pulver et al. 1983) indicate a pattern similar to that in Europe. However, data from the southern hemisphere, e.g., Australia, show a peak between June and November (Jones and Frei 1979). Since the latter period is the coolest season in Australia, the common denominator of all these findings from different latitudes appears to be the season with the lowest average temperatures.

It should be noted that seasonality of births has been described in some other disorders—for example, diabetes mellitus (Christy et al. 1982). An excess of winter births has been reported for certain congenital anomalies and diseases, such as anencephaly, malformations of the cardiovascular system, congenital hip dislocation, and Down's syndrome (Jongbloet, Mulder, and Hamers 1982). Four different hypotheses have been proposed to explain the seasonality of birth phenomenon in schizophrenia.

1. It has been suggested that the reproductive behavior of parents of schizophrenics may be atypical and the conception maximum may deviate from that for the general population. Ødegård (1977) has contested this proposition because the typical autumn peak of the birth curve can also be found in series of schizophrenics; it is the excessive height of the winter-spring peak that produces the statistical difference.

2. The excess of births of schizophrenics in the cold months of the year may be spurious. It is well known that the rate of stillbirths and of perinatal mortality in the general population is higher in winter than in other seasons. If it could be assumed that the schizophrenic genotype confers some selective advantage to the fetus and the newborn to increase its chances of survival, then it is the reduced fetal wastage and perinatal mortality among the carriers of the genotype, instead of an absolute antecedent increase in the conception rate, that could explain the difference.

3. Little-understood hormonal cycles in the maternal organism may interact with genetic vulnerability in ways that increase the morbid risk for conceptions falling within a particular season.

4. Nonspecific pathogens—and, in particular, viral infection—may affect the fetus selectively during certain seasons. For example, ductus arteriosus apertus tends to occur more frequently in individuals born in the winter months, but it is a sequel of rubella infection of the mother contracted in the early months of pregnancy, i.e., in late spring when the rubella virus has its maximum spread in the population.

None of these hypotheses have been satisfactorily tested or disproved. An increasing number of investigators are considering the possible role of viruses in the pathogenesis of at least some of the schizophrenic disorders (Morozov 1983), and one of the arguments brought forward to support such a view is the seasonality of birth phenomenon. As indicated above, however, this phenomenon can have several different plausible explanations, and it furnishes no more than circumstantial evidence in favor of a viral involvement in schizophrenia.

Associated Factors: Physical and Neurological Conditions

Numerous instances of positive or negative associations between schizophrenia and a variety of physical and neurological conditions have been reported in the European literature. This important issue in the epidemiological study of schizophrenia has two distinct aspects. On the one hand, physical diseases may occur in schizophrenic patients with a significantly higher, or lower, frequency than expected. Such relationships may point to particular biological or behavioral factors that explain the deviation from the coincidental occurrence of the two conditions in the same individual. On the other hand, schizophrenia-like syndromes may result causally from a variety of congenital or acquired conditions affecting the central nervous system. Since the nature of the pathological lesion is known in some of these conditions, they offer models for the study of cerebral mechanisms capable of producing schizophrenic symptoms.

As regards the first problem, much of the evidence unfortunately derives from samples that are limited in size or not representative of either the schizophrenic or the other disease cases. Nevertheless, there are several systematic studies that merit more attention than they have received so far.

Tuberculosis was one of the first diseases observed to occur with excessive frequency among schizophrenics. The fact that these were mainly institutionalized populations which were more heavily exposed to infection, lived in overcrowded conditions, and received inadequate diet, as well as the general decline of tuberculosis morbidity with the introduction of modern treatment and the improvements in living conditions, has tended to obscure the well-documented observation that schizophrenic patients were far more vulnerable to the infection than nonschizophrenic individuals living under comparable conditions. Alstrom (1938) analyzed the records of all the 2,273 patients who died in the period 1924–36 in mental hospitals in Stockholm. After standardizing the data for length of illness and hospitalization, he concluded that schizophrenic patients had a significantly higher risk of dying of tuberculosis than patients with manic-depressive psychosis, presenile or senile dementia, and other diagnoses. In a census study that he carried out in one of the hospitals, he found active tuberculosis in 10 percent of the schizophrenic patients, compared to 3–4 percent in the patients with other diagnoses. More recent studies, e.g., the Oxford Record Linkage Study (ORLS) (Baldwin 1979), which used morbidity register linkage data have shown a highly significant excess of pulmonary tuberculosis in individuals subsequently hospitalized with a diagnosis of schizophrenia, and a generally increased rate of pneumonia and other infectious disease among schizophrenics.

The ORLS also demonstrated increased relative risks in schizophrenics for arteriosclerotic heart disease (but not for other cardiovascular disease), for the malabsorption syndrome associated with celiac disease, and for mixoedema. The link with celiac disease, for which some pathogenetic basis has been proposed (Dohan 1966), is less certain, however, because at least one controlled study in which adequate diagnostic tests for celiac disease were applied to a chronic schizophrenic population (Stevens et al. 1977) failed to show an increased frequency of the condition.

Among the diseases reported to occur with significantly reduced incidence among schizophrenic patients, the best documented evidence pertains to rheumatoid arthritis. Osterberg (1978) reviewed the records of all discharges from psychiatric hospitals in Sweden for 1962–73 and found a strikingly reduced rate of seropositive inflammatory joint disease in schizophrenics (0.047 percent) compared to patients with other psychiatric diagnoses, although the rate in the latter was also lower than expected (the population rate in Sweden is 2.2 percent). In contrast, ankylosing spondylitis and uroarthritis, both associated with the HLA B-27 antigen, were more frequent among psychiatric patients than rheumatoid arthritis, but the clinical picture in these cases was one of atypical psychosis and not of nuclear schizophrenia. These data suggest an interesting area for future investigations of possible disease markers, which should be based on record linkage data on large samples, in order to investigate the relations among schizophrenia, joint disease, and the major histocompatibility complex.

Several reports have suggested either a significantly increased, or a significantly decreased, rate of occurrence of cancer in schizophrenic patients. The ORLS program failed to produce conclusive evidence. A WHO-coordinated study on cancer and schizophrenia, based on morbidity registers in Denmark, Japan, and the United States (Hawaii and Rochester, N.Y.), has now been completed, and the results will be reported in the near future.

The second aspect of the problem, the occurrence of symptomatic schizophrenias in association with cerebral and physical disease, raises important questions about the nosology of schizophrenia. If the clinical characteristics of the psychotic illnesses secondary to demonstrable cerebral disease, or to physical disease affecting the central nervous system, are phenomenologically indistinguishable from "true" schizophrenia, then, in the long run, the nosological status of the schizophrenic disorders may turn out not to be very different from that of epilepsy or mental retardation, where a number of discrete entities of genetic or exogenous causation have been identified, resulting in a progressive shrinkage of the share of "idiopathic" forms. How soon and to what an extent such developments may take place in schizophrenia remains uncertain, but the accumulating evidence of occurrence of schizophrenic or schizophreniform disorders in association with cerebral and extracerebral pathologies makes this proposition plausible. The evidence up to 1967 has been reviewed by Davison and Bagley (1969), who established that no less than 12 major groups of neurological, endocrine and infectious diseases, intoxications, and space-occupying intracranial lesions can give rise to disorders with schizophrenic features. Epilepsy is perhaps the best known example (Slater 1969; Flor-Henry 1983), but more recent additions to the list include idiopathic basal ganglia calcification (Francis and Freeman 1984) and aqueduct stenosis (Roberts, Trimble, and Robertson 1983). Propping (1983) has called attention to the fact that many genetic disorders, ranging from XXX karyotype to acute intermittent porphyria, are associated with a significantly increased risk of schizophreniform psychosis. Some genetic conditions, like metachromatic leukodystrophy, are sometimes discovered, either by chance or through systematic screening, in patients diagnosed as schizophrenic.

Unfortunately, the search for cerebral or somatic pathologies that can cause schizophrenic symptoms has not been systematic, and few of the observations collected so far lend themselves to a quantitative epidemiological analysis. It is to be hoped that this line of research will receive new impetus from the introduction of effective screening and diagnostic techniques, suitable for use in large samples. The eventual convergence between this type of epidemiological research and the investigation of the nature and extent of morphological cerebral abnormalities, immunological disorders, and biochemical findings in clinical series of schizophrenic patients may be a promising strategy of inquiry into the biological pathways leading to the schizophrenic phenotype.

Conclusions and Summary

It is difficult to generalize about European epidemiologcial research in schizophrenia, because of the coexistence of a variety of "schools," traditions, and approaches. In addition, studies are conducted, and their findings reported, in many different languages—a communication barrier of no small significance if the European situation is compared to the almost unobstructed flow of information within North American psychiatry. In spite of the increased use of English as a *lingua franca* of oral and written scientific communication in psychiatry in Europe, important data and theoretical viewpoints continue to be published in languages accessible to small segments of researchers. Therefore, a comprehensive picture of European research in psychiatric epidemiology is extremely hard to obtain.

With this provision, however, it is nevertheless possible to discern several clear trends in European epidemiological investigations of schizophrenia that can, to some extent, be contrasted to North American developments, especially if the latter are examined over a longer period of time and not limited to the current movement of nosological revival.

A first distinguishing feature of European research is its clinical slant: epidemiological field studies have mainly been conceived as extensions of clinical observations and concepts. This is also linked to the fact that most European epidemiological studies of schizophrenia and other severe disorders have been carried out by psychiatrists and the involvement of psychologists, sociologists, or public health professionals has been far less extensive than in the United States. The involvement and primacy of psychiatrists in field work also explains, to some extent, the less frequent use of rating scales and other psychometric devices, and the greater reliance in Europe on clinical judgment. It is characteristic, for example, that one of the first and most widely used standardized instruments for clinical and epidemiological research in the major psychoses, the Present State Examination (Wing, Cooper, and Sartorius 1974), has the format of a clinical interview that requires qualified judgment for deciding on the presence or absence of a given symptom. The predilection for the clinical approach further explains why in so many European studies the assessment of the individual case always results in a diagnosis, but much less frequently in some sort of a quantitative score. For all the advantages of clinical diagnosis, the insufficient attention to quantitative measurement of psychopathology in most past European studies on the epidemiology of schizophrenia has perhaps impeded some of the more analytical applications of epidemiological methods, e.g., the articulation of the schizophrenic phenotype into separate components or dimensions.

A second feature of European research, logically linked with the above-mentioned clinical outlook, is that schizophrenia has always been conceptualized as a nosological entity. Also, notwithstanding Bleuler's "group of schizophrenias," it has usually been investigated epidemiologically as if it were a single disease. This approach has certainly contributed to the determination of prevalence, incidence, and morbid risk rates for the core syndromes of schizophrenia, but it has left unanswered many questions about the frequency and distribution of the various borderline, latent, and other atypical forms of manifestation which, in the light of more recent genetic studies (especially those on adopted offspring of schizophrenics), belong to the wider spectrum of schizophrenic disease. Some of the best field studies in Europe have been guided by Kraepelin's 1899 concept of dementia praecox, but paradoxically, his later, 1920 ideas about the nature of the disorder have had little influence on epidemiological research. Exceptions, in a certain sense, are some of the Soviet epidemiological studies that have proceeded from a concept of schizophrenia as a broad continuum of forms, distinguished primarily by their different prognoses and patterns of progression. However, this approach too has been confined to the "endogenous" types of schizophrenic illnesses, and little attention has been given to exploring "exogenous" or "symptomatic" schizophreniform disorders, secondary to cerebral disease. Although extensive information has been accumulated about the occurrence of schizophrenia-like illnesses in association with a variety of other diseases, it has resulted mainly from clinical observation of small series of cases. The ultimate epidemiological test—is the observed incidence of cases of joint occurrence of two conditions significantly different from the product of the population incidence rates for each of the conditions?—has not been consistently applied because of insufficient empirical data. The present interest in the neuropathology and the organic lesions underlying schizophrenic symptoms may lead to a revival of Bonhoeffer's views on the nosology of the "reaction types" of the central nervous system and to a new perspective on the epidemiology of schizophrenia.

A third feature of European research is that it has pioneered some methodological innovations, like the comprehensive survey, and the repeat survey of defined populations, which have proved extremely fruitful. A unique contribution has been provided by the cumulative psychiatric case registers, and especially those in countries like Denmark and Norway which cover a span of several decades and thus allow types of research that would be impossible to carry out by other means. Other morbidity registers, of more recent origin, have also proved to be valuable research instruments, especially through their linkages to other disease registers and mortality data.

Against this background of research styles, concepts, and tools, the epidemiological picture of schizophrenia that has been outlined by European studies to date is fairly uniform, though far from complete. The syndrome of schizophrenia has been found to occur in all European populations, with an annual incidence of 0.2–0.6 per 1,000, a point prevalence in the range of 2.5–5.3 per 1,000, and an individual morbid risk between 0.36 percent and 1.87 percent. Several pockets, or larger areas, of high prevalence have been described, but with the exception of effects of a genetic isolate, the factors accounting for such high rates are not fully understood. The rates of the disorder are similar in males and females, and the incidence rate is highest in early adult age. Schizophrenic patients have a significantly higher mortality rate than the general population, and a somewhat lower fertility rate. No clear evidence for any kind of selective reproductive advantage for schizophrenics or their biological relatives has been produced, but the hypothesis of a balanced polymorphism has not been conclusively tested. Studies of the social ecology of schizophrenia, including social class distribution, urban/rural comparisons, and effects of macrosocial stress and migration, have not resulted in any significant etiological clues, but it has been shown that stress of a more personal nature (e.g., life events and emotional interactions within the family) may have some impact on the course of the disorder.

There is an excess of winter and spring births among schizophrenics in Europe, and this phenomenon may be related to the pathogenesis of the condition, although the evidence is open to several interpretations. It should be seen, however, in the context of other findings which suggest the possibility of multiple etiologies for the schizophrenic syndrome—findings such as its occurrence in association with a variety of central nervous system lesions and disorders, and somatic diseases. If a continuum of genetic and nongenetic etiologies for the schizophrenic syndromes can be demonstrated, and a continuum of reliably diagnosable psychotic and nonpsychotic forms of manifestation is established, then challenging possibilities for new types of epidemiological studies would be opened. Such studies might take us at least one step further in the understanding of a condition that for a good part of a century has been the scientific obsession of European psychiatry. It is to be hoped that European epidemiological research has not yet pronounced its last word on the subject.

References

Adelstein, A.M.; Downham, D.Y.; Stein, Z.; and Susser, M.W. The epidemiology of schizophrenia in an English city. *Social Psychiatry,* 3:47-59, 1968.

Alström, C.H. Schizophrenie und Tuberkulose. *Zeitschrift fur die gesamte Neurologie und Psychiatie,* 162:25-50, 1938.

Andersen, T. Physical and mental illness in a Lapp and a Norwegian population. In: Andersen, T.; Astrup, C.; and Forsdahl, A., eds. "Social, Somatic and Psychiatric Studies of Geographically Defined Populations." *Acta Psychiatrica Scandinavica,* Suppl. 263, 1975.

Baldwin, J.A. Schizophrenia and physical disease (editorial). *Psychological Medicine,* 9:611-618, 1979.

Baumann, U.; Schneidewind, G.; Angst, J.; Helmchen, H.; and Hippius, H. Untersuchungen zum AMP-System. Symptomvergleich zwischen Berlin und Zurich. *Arzneimittel-Forschung (Drug Research),* 26:1111-1145, 1976.

Berner, P., and Küfferle, B. British phenomenological and psychopathological concepts: A comparative review. *British Journal of Psychiatry,* 140:558-565, 1982.

Birley, J.L.T., and Brown, G.W. Crises and life changes preceding the onset or relapse of acute schizophrenia: Clinical aspects. *British Journal of Psychiatry,* 116:327-333, 1970.

Bjarnar, E.; Reppesgaard, H.; and Astrup, C. Psychiatric morbidity in Berlevaag. In: Andersen, T.; Astrup, C.; and Forsdahl, A., eds. "Social, Somatic and Psychiatric Studies of Geographically Defined Populations." *Acta Psychiatrica Scandinavica,* Suppl. 263, 1975.

Bleuler, E. *Dementia Praecox oder die Gruppe der Schizophrenien.* Leipzig-Wien: Deuticke, 1911.

Bonhoeffer, K. Die Psychosen im Gefolge von akuten Infektionen, Allgemein-Erkrankungen und inneren Erkrankungen. In: Aschaffenburg, W., ed. (Hrsg) *Handbuch der Psychiatrie.* Teil B. Leipzig-Wien: Deuticke, 1912. pp. 1-110.

Böök, J.A. A genetic and neuropsychiatric investigation of a North Swedish population (with special regard to schizophrenia and mental deficiency). *Acta Genetica,* 4:1-100, 1953.

Böök, J.A.; Wetterberg, L.; and Modrzewska, K. Schizophrenia in a North Swedish geographical isolate, 1900-1977: Epidemiology, genetics and biochemistry. *Clinical Genetics,* 14:373-394, 1978.

Bremer, J. A social-psychiatric investigation of a small community in Northern Norway. *Acta Psychiatrica et Neurologica Scandinavica,* Suppl. 62, 1951.

Brown, G.W.; Birley, J.L.T.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry,* 121:241-258, 1972.

Brugger, C. Versuch einer Geisteskrankenzählung in Thüringen. *Zeitschrift fur die gesamte Neurologie und Psychiatrie,* 133:252-390, 1931.

Brugger, C. Psychiatrische Ergebnisse einer medizinischen, anthropologischen und soziologischen Bevolkerungsuntersuchung. *Zeitschrift fur die gestamte Neurologie und Psychiatrie,* 146:489-524, 1933.

Brugger, C. Psychiatrische Bestandesaufnahme im Gebiet eines medizinisch-anthropologischen Zensus in der Nähe von Rosenheim. *Zeitschrift fur die gesamte Neurologie und Psychiatrie,* 160:189-207, 1938.

Busch, H.; von Cranach, M.; Gulbinat, W.; Renford, E.; and Tegeler, J. Reliability of the AMDP-System. *Acta Psychiatrica Scandinavica,* 62:382-392, 1980.

Christy, M.; Christau, B.; Molbak, A.G.; and Nerup, J. Diabetes and month of birth. Letter to editor. *Lancet,* II:216, 1982.

Cooper, B. Social class and prognosis in schizophrenia: Parts I and II. *British Journal of Preventive Social Medicine,* 15:17-41, 1961.

Cooper, J., and Sartorius, N. Cultural and temporal variations in schizophrenia: A speculation on the importance of industrialization.

British Journal of Psychiatry, 130:50–55, 1977.

Cooper, J.E.; Kendell, R.E.; Gurland, B.J.; Sharpe, L.; Copeland, J.R.M.; and Simon, R. *Psychiatric Diagnosis in New York and London.* London: Oxford University Press, 1972.

Crocetti, G.J.; Lemkau, P.V.; Kulcar, A.; and Kessic, B. Selected aspects of the epidemiology of psychoses in Croatia, Yugoslavia: II. The cluster sample and the results of the pilot survey. *American Journal of Epidemiology,* 94:126–134, 1971.

Davison, K., and Bagley, C.R. Schizophrenia-like psychoses associated with organic disorders of the central nervous system: A review of the literature. In: Herrington, R.N., ed. *Current Problems in Neuropsychiatry.* (*British Journal of Psychiatry* Special Publication No. 4) Ashford, Kent: Headley Brothers, 1969. pp. 113–184.

Dilling, H., and Weyerer, S. Incidence and prevalence of treated mental disorders: Health care planning in a small-town rural region of Upper Bavaria. *Acta Psychiatrica Scandinavica,* 61:209–222, 1980.

Dohan, F.C. Cereals and schizophrenia: Data and hypothesis. *Acta Psychiatrica Scandinavica,* 42:125–152, 1966.

Essen-Möller, E. Untersuchungen über die Fruchtbarkeit gewisser Gruppen von Geisteskranken. *Acta Psychiatrica Neurologica,* Suppl. 8, 1935.

Essen-Möller, E.; Larsson, H.; Uddenberg, C.E.; and White, G. Individual traits and morbidity in a Swedish rural population. *Acta Psychiatrica et Neurologica Scandinavica,* Suppl. 100, 1956.

Faergeman, P.M. *Psychogenic Psychoses.* London: Butterworth, 1963.

Flor-Henry, P. *Cerebral Basis of Psychopathology.* Boston: John Wright, 1983.

Francis, A., and Freeman, H. Psychiatric abnormality and brain calcification over four generations. *Journal of Nervous and Mental Disease,* 172:166–170, 1984.

Fremming, K.H. *Sygdomsrisikoen for Sindslidelser og Andre Sjaelelige Abnormtilstande i den Danske Gennemsnitsbefolkning.* Copenhagen: Munksgaard, 1947.

Gajdusek, D.C., and Zigas, V. Clinical, pathological and epidemiological study of an acute progressive degenerative disease of the central nervous system among natives of the Eastern Highlands of New Guinea. *American Journal of Medicine,* 26:442–469, 1959.

Gavrilova, S.I. Ne utochnennye dispanserom shizofrenicheskije rasstrojstva vyjavlennye pri kliniko-epidemiologicheskom obsledovanii pozdnyh vozrastnyh grupp obshchego naselenija [Schizophrenic disorders not identified by the dispensary and revealed in a clinical and epidemiological study of advanced age groups in the general population]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 79:1366–1372, 1979.

Goldberg, E.M., and Morrison, S.L. Schizophrenia and social class. *British Journal of Psychiatry,* 109:785–802, 1963.

Graemiger, O. Beitrag zur Frage der Haufigkeit der Psychosen und der erblichen Belastung. *Schweizerische Medizinische Wochenschrift,* 24:561–569, 1931.

Griesinger, W. *Die Pathologie und Therapie der psychischen Krankheiten.* Stuttgart: Krabbe, 1861.

Häfner, H., and Reimann, H. Spatial distribution of mental disorders in Mannheim, 1965. In: Hare, E.H., and Wing, J.K., eds. *Psychiatric Epidemiology.* London: Oxford University Press, 1970. pp. 341–354.

Hagnell, O. *A Prospective Study of the Incidence of Mental Disorder.* Lund: Svenska Bokforlaget, 1966.

Hagnell, O., and Ojesjo, L. A prospective study concerning mental disorders of a total population investigated in 1947, 1957 and 1972. In: Andersen, T.; Astrup, C.; and Forsdahl, A., eds. "Social, Somatic and Psychiatric Studies of Geographically Defined Populations." *Acta Psychiatrica Scandinavica,* Suppl. 263, 1975.

Hare, E.H. Was insanity on the increase? *British Journal of Psychiatry,* 142:439–455, 1983.

Hare, E.H., and Price, J.S. Mental disorders and season of birth: Comparison of psychoses with neurosis. *British Journal of Psychiatry,* 115:533–540, 1968.

Harris, A.; Linker, I.; Norris, V.; and Shepherd, M. *British Journal of Preventive and Social Medicine,* 10:107–114, 1956.

Haslam, J. *Observations on Madness and Melancholy.* 2nd ed. London: Callow, 1809.

Helgason, L. Psychiatric services and mental illness in Iceland. *Acta Psychiatrica Scandinavica,* Suppl. 268:1–140, 1977.

Helgason, T. Epidemiology of mental disorders in Iceland. *Acta Psychiatrica Scandinavica,* Suppl. 173, 1964.

Herrman, H.E.; Baldwin, J.A.; and Christie, D. A record-linkage study of mortality and general hospital discharges in patients diagnosed as schizophrenic. *Psychological Medicine,* 13:581–593, 1983.

Huxley, J.; Mayr, E.; Osmond, H.; and Hoffer, A. Schizophrenia as a genetic morphism. *Nature,* 204:220-221, 1964.

Jablensky, A., and Hugler, H. Moglichkeiten und Grenzen psychiatrischer epidemiologischer Surveys fur geographisch definierte Population in Europa. *Fortschritte der Neurologie. Psychiatrie und Ihrer Grenzgebiete,* 50:215-239, 1982.

Jablensky, A. Epidemiological surveys of mental health of geographically defined populations in Europe. In: Weissman, M.; Myers, J.; Ross, C.; and Slaby, A., eds. *Community Surveys.* New Brunswick, NJ: Rutgers University Press, in press.

Jaspers, K. *General Psychopathology.* Manchester: Manchester University Press, 1963.

Jones, J.H., and Frei, D. Seasonal births in schizophrenia: A southern hemisphere study using matched pairs. *Acta Psychiatrica Scandinavica,* 59:164-172, 1979.

Jongbloet, P.H.; Mulder, A.; and Hamers, A.J. Seasonality of pre-ovulatory non-disjunction and the etiology of Down syndrome: A European collaborative study. *Human Genetics,* 62(2):134-138, 1982.

Kahlbaum, K. *Die Katatonie oder das Spannungsirresein.* Berlin: Hirschwald, 1874.

Kelleher, M.J., and Copeland, J.R. Psychiatric diagnosis in Ireland: A videotape study. *Journal of the Irish Medical Association,* 67:87-92, 1974.

Kelleher, M.J.; Copeland, J.R.; and Smith, A.J. High first admission rates for schizophrenia in the west of Ireland. *Psychological Medicine,* 4:460-462, 1974.

Kendell, R.E. The International Classification and the diagnoses of English psychiatrists, 1968-1980. *British Journal of Psychiatry,* 139:177-180, 1981.

Kleist, K. Über zykloide, paranoide und epileptoide Psychosen und über die Frage der Degenerationspsychosen. *Schweizer Archiv fur Neurologie und Psychiatrie,* 23:1-35, 1928.

Klemperer, J. Zur Belastungsstatistik der Durchschnittsbevölkerung. Psychosehaufigkeit unter 1000 stichprobemassig ausgelesenen Probanden. *Zeitschrift fur die gesamte Neurologie und Psychiatrie,* 146:277-316, 1933.

Koller, J. Beitrag zur Erblichkeitsstatistik der Geisteskranken im Kanton Zurich. *Archiv fur Psychiatrie,* 27:268-294, 1895.

Kraepelin, E. Die Erscheinungsformen des Irresseins. *Zeitschrift fur die gesamte Neurologie und Psychiatrie,* 62:1-29, 1920.

Kraepelin, E. *Psychiatrie: ein Lehrbuch fur Studierende und Aerzte.* VI. Aufl. Leipzig: Abel, 1899.

Lang, T. Zur Frage: Geisteskrankheit und Geburtsmonat. *Archiv fur Rassen-und Gesellschafts-Biologie,* 25:42-57, 1931.

Langfeldt, G. *The Schizophreniform States: A Catamnestic Study Based on Individual Re-examinations.* Copenhagen: Munksgaard, 1939.

Larsson, T., and Sjögren, T. A methodological, psychiatric and statistical study of a large Swedish rural population. *Acta Psychiatrica et Neurologica Scandinavica,* Suppl. 89, 1954.

Leff, J.P. Schizophrenia and sensitivity to the family environment. *Schizophrenia Bulletin,* 2:566-574, 1976.

Lehtinen, V.; Väisänen, E.; Alanen, Y.O.; and Tienari, P. Preventive implications of a social-psychiatric survey in the Finnish population. *Psychiatria Fennica,* 78:143-151, 1978.

Leonhard, K. *Die Aufteilung der endogenen Psychosen.* Berlin: Akademie Verlag, 1957.

Lieberman, Y.I. K probleme zabolevaemosti shizofreniej: materialy kliniko-epidemiologicheskogo obsledovanija [The problem of incidence of schizophrenia: Material from a clinical and epidemiological study]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 74:1224-1232, 1974.

Mayer-Gross, W. Mental health survey in a rural area (a preliminary report). *The Eugenics Review,* 40:140-148, 1948.

Mayer-Gross, W.; Slater, E.; and Roth, M. *Clinical Psychiatry.* 3rd ed. London: Bailliere, Tindall & Cassell, 1969.

Meynert, T. *Lehrbuch der Psychiatrie.* Wien: Deuticke, 1884.

Morozov, P.V., ed. *Research on the Viral Hypothesis of Mental Disorders.* Basel: Karger, 1983.

Nielsen, J. The Samsö project from 1967 to 1974. *Acta Psychiatrica Scandinavica,* 54:198-222, 1976.

Noreik, K., and Ødegård, Ø. Age at onset of schizophrenia in relation to socio-economic factors. *British Journal of Social Psychiatry,* 1:243-249, 1967.

Norris, V. *Mental Illness in London.* Maudsley Monograph. London: Chapman & Hall, 1959.

Ødegård, Ø. A statistical investigation of the incidence of mental disorder in Norway. *Psychiatric Quarterly,* 20:381-401, 1946.

Ødegård, Ø. The incidence of mental diseases as measured by census investigations versus admission statistics. *Psychiatric Quarterly,* 26:212-218, 1952.

Ødegård, Ø. Season of birth in the population of Norway, with particular reference to the September birth maximum. *British Journal of Psychiatry,* 131:339–344, 1977.

Ødegård, Ø. Fertility of psychiatric first admissions in Norway 1936–1975. *Acta Psychiatrica Scandinavica,* 62:212–220, 1980.

O'Hare, A.; Walsh, D.; and Torrey, E.F. Seasonality of schizophrenic births in Ireland. *British Journal of Psychiatry,* 137:74–77, 1980.

Osterberg, E. Schizophrenia and rheumatic disease. *Acta Psychiatrica Scandinavica,* 58:339–359, 1978.

Osterreichisches statistisches Handbuch. Wien: Deuticke, 1898–1907. pp. 17:50–51; 21:38–39; 26:53–59.

Ouspenskaya, L.Y. Nekotorye voprosy metodiki sravnitelnogo epidemiologicheskogo issledovanija i kharakteristiki rasprostranennosti shizofreni v otedlnyh rajonah strany [Some problems of the methodology of comparative epidemiological study and the characteristics of prevalence of schizophrenia in various areas of the country]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 78:724–748, 1978.

Panse, F. Beitrag zur Belastrungsstatistik einer Durchschnittsbevolkerung. *Zeitschrift fur die gesamte Neurologie und Psychiatrie,* 154:194–222, 1936.

Parchappe, M. *Recherches Statistiques sur les Causes de l'Alienation Mentale.* Rouen: Briere, 1839.

Pichot, P. The French approach to psychiatric classification. *British Journal of Psychiatry,* 144:113–118, 1984.

Pinel, P. *Nosographie Philosophique, ou la Méthode de l'Analyse Appliquée a la Médecine.* 2nd ed. Vol. III. Paris: Brosson, 1803.

Propping, P. Genetic disorders presenting as "schizophrenia." Karl Bonhoeffer's early view of the psychoses in the light of medical genetics. *Human Genetics,* 65:1–10, 1983.

Pulver, A.E.; Stewart, W.; Carpenter, W.T., Jr.; and Childs, B. Risk factors in schizophrenia: Season of birth in Maryland, U.S.A. *British Journal of Psychiatry,* 143:389–396, 1983.

Rimmer, J., and Jacobsen, B. Differential fertility of adopted schizophrenics and their half-siblings. *Acta Psychiatrica Scandinavica,* 54:161–166, 1976.

Roberts, J.K.A.; Trimble, M.R.; and Robertson, M. Schizophrenic psychosis associated with aqueduct stenosis in adults. *Journal of Neurology, Neurosurgery and Psychiatry,* 46:892–893, 1983.

Rotstein, V.G. Materialy osmotra psihiatrom vyborochnyh grupp vzroslogo naselenija v rjade rajonov SSSR [Material from a psychiatric survey of sample groups from the adult population in several areas of the USSR]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 77:569–574, 1977.

Rotstein, V.G. O zabolevaemosti paranoidnoj schizofreniej [On the incidence of paranoid schizophrenia]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 83:571–579, 1982.

Rüdin, E. *Zur Vererbung und Neuentstehung der Dementia praecox.* Berlin: Springer, 1916.

Sartorius, N.; Jablensky, A.; Strömgren, E.; and Shapiro, R. Validity of diagnostic concepts across cultures: A preliminary report from the International Pilot Study of Schizophrenia. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment.* New York. John Wiley & Sons, 1978. pp. 657–669.

Saugstad, L.F., and Ødegård, Ø. Mortality in psychiatric hospitals in Norway. *Acta Psychiatrica Scandinavica,* 59:431–447, 1979.

Schulsinger, F., ed. Schizophrenia in Europe. *Schizophrenia Bulletin,* 6:555–638, 1980.

Seabra-Dines, T. Sopostavlenije diagnosticheskoj ocenki shizofrenii s pozicij sovetskoj i franzuskoj psihiatrii [A comparison of the diagnostic assessment of schizophrenia from the positions of Soviet and French psychiatry]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 77:1413–1423, 1977.

Shepherd, M.; Brook, E.M.; Cooper, J.E.; and Lin, T.Y. An experimental approach to psychiatric diagnosis. *Acta Psychiatrica Scandinavica,* Suppl. 201, 1968.

Shmaonova, L. M.; Lieberman, Y.I.; Panicheva, E.V.; and Rotstein, V.G. Formirovanie sem'i u bolnyh shizofreniej i maniakalno-depressivnym psihozom po dannym epidemiologicheskogo obsledovanija—rozhdaemost [Family formation in schizophrenic and manic-depressive patients' according to data from an epidemiological study—Fertility]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova,* 76:754–759, 1976.

Shur, E., and Hare, E. Age-prevalence and the season of birth effect in schizophrenia: A response to Lewis and Griffin. *Psychological Bulletin,* 93:373–377, 1983.

Sjögren, T. Genetic-statistical and psychiatric investigations of a West Swedish population. *Acta Psychiatrica et Neurologica,* Suppl. 52, 1948.

Slater, E. The schizophrenia-like illnesses of epilepsy. In: Herrington R.N., ed. *Current Problems in*

Neuropsychiatry. (*British Journal of Psychiatry*, Special Publication No. 4) Ashford, Kent: Headley Brothers, 1969. pp. 77-81.

Snezhnevsky, A.V., ed. *Shizofrenija: multidisciplinarnoje issledovanie* [*Schizophrenia: A Multidisciplinary Study*]. Moscow: Medicina, 1972.

Snezhnevsky, A.V., ed. *Rukovodstvo po psihiatrii* [*Manual of Psychiatry*]. Moscow: Medicina, 1983.

Snezhnevsky, A.V., and Nadzharov, R.A. Features of the Soviet approach to psychiatric nosology. In: *WHO/ADAMHA Mental Disorders, Alcohol and Drug Related Problems: International Perspectives for Their Diagnosis and Classification*. Amsterdam: Excerpta Medica International Congress Series No. 669, 1985. pp. 45-49.

Stevens, F.M.; Lloyd, R.S.; Geraghty, S.M.J.; Reynolds, M.T.G.; Sarsfield, M.J.; McNicholl, B.; Fottrell, P.F.; Wright, R.; and McCarthy, C.F. Schizophrenia and coeliac disease—The nature of the relationship. *Psychological Medicine*, 7:259-263, 1977.

Strömgren, E. Beiträge zur psychiatrischen Erblehre, auf Grund von Untersuchungen an einer Inselbevölkerung. *Acta Psychiatrica Neurologica*, Suppl. 19, 1938.

Strömgren, E. Statistical and genetical population studies within psychiatry: Methods and principal results. *Proceedings of the First International Congress of Psychiatry, Paris, 1950*. Vol. VI. Paris: Hermann, 1950. pp. 155-192.

Strömgren, E. Contributions to psychiatric epidemiology and genetics. *Acta Jutlandica*, 40:1-86, 1968.

Temkov, I.; Jablensky, A.; and Boyadjieva, M. Use of reported prevalence data in cross-national comparisons of psychiatric morbidity. *Social Psychiatry*, 3:111-117, 1980.

Terris, M., ed. *Goldberger on Pellagra*. New Orleans: Louisiana State University Press, 1964.

Torrey, E.F. *Schizophrenia and Civilization*. New York: Jason Aronson, 1980.

Torrey, E.F.; Torrey, B.B.; and Peterson, M.R. Seasonality of schizophrenic births in the United States. *Archives of General Psychiatry*, 37:1065-1070, 1977.

Tramer, M. Über die biologische Bedeutung des Geburtsmonats, insbesondere für die Psychosenerkrankung. *Neurologie, Neurochirurgie, und Psychiatrie*, 24:17-24, 1929.

Tuke, D.H. *A Dictionary of Psychological Medicine*. Philadelphia: Blakiston, 1892.

Videbech, T.; Weeke, A.; and Dupont, A. Endogenous psychoses and season of birth. *Acta Psychiatrica Scandinavica*, 50:202-218, 1974.

Walsh, D. Mental illness in Dublin—First admissions. *British Journal of Psychiatry*, 115:449-456, 1969.

Weeke, A., and Strömgren, E. Fifteen years later: A comparison of patients in Danish psychiatric institutions in 1957, 1962, 1967 and 1972. *Acta Psychiatrica Scandinavica*, 57:129-144, 1978.

Weinberg, W. Methoden und Technik der Statistik mit besonderer Berücksichtigung der Sozialbiologie. In: Goldstein, A.; Schlossman, A.; and Teleky, L., eds. *Handbuch der sozialen Hygiene und Gesundheitsfürsorge*. Bd. I. Berlin: Springer, 1925. pp 71-148.

Wimmer, A. *Psykogene Sindssygdomsformer*. Copenhagen: Gads Vorlag, 1916.

Wing, J.K.; Cooper, J.E.; and Sartorius, N. *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press, 1974.

World Health Organization. *Glossary of Mental Disorders and Guide to Their Classification*. Geneva: WHO, 1974.

Zharikov, N.M.; Kalachev, V.F.; and Sokolova, E.D. Sravnitelnoje izuchenie bolnyh shizofreniej prozhivajushchih v centralnom rajone i na krajnem severo-vostoke SSSR [A comparative study of schizophrenic patients inhabiting the central area and the extreme North-East of the USSR]. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 79:453-460, 1979.

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Research on Schizophrenia in Southern European Countries

by Dargut Kemali

Abstract

The research on schizophrenia during the last 50 years in the Latin language countries of Southern Europe is briefly surveyed. Particular emphasis is given to the contributions of French authors to the diagnostic concept of schizophrenia, as well as to the phenomenological studies concerning the "essence" of schizoidia/schizophrenia, and some initial schizophrenic "Erlebnisse." The development of biological, psychological, and sociological theories concerning the etiology of schizophrenia is outlined, and contributions such as the "aminotoxic theory" of V.M. Buscaino and the theoretical principles of "alternative psychiatry" are examined in detail. Lastly, the role of French, Italian, Spanish, and Portuguese psychiatrists in the introduction and improvement of somatic treatments is described.

Research on schizophrenia in the Latin language countries of Southern Europe has developed along the following principal lines: (1) nosology—definition and delimitation of the concept of schizophrenia; exploration of conditions on the border between schizophrenia and neuroses; (2) psychopathology—phenomenological analysis of the "essence" of schizoidia and schizophrenia, as well as of some experiences peculiar to the initial stages of schizophrenia; studies concerning schizophrenic thought and language; (3) etiology—exploration of factors involved in the pathogenesis of schizophrenic psychoses, carried out on the basis of the three "paradigms": biological, psychological, and sociological; (4) clinical observation and intervention—study of natural course, prognosis, and treatment modalities.

The present article provides a brief survey of the development and present state of knowledge in each of these research areas. It also includes reference to earlier articles, probably unknown to the Anglo-Saxon reader, which have anticipated currently topical ideas and research trends.

Nosology

Definition and Delimitation of the Concept of Schizophrenia. The most significant contributions in this field have come from the French. They represent a reaction to the growing extension (and consequent indetermination) that the concept of schizophrenia has acquired over time in the Anglo-Saxon literature ("la volatilisation du concept de schizophrénie"), and an attempt to redefine the clinical nucleus of this syndrome.

Thus, according to Ey (1955), the essence of schizophrenia is to be found in the "profound and progressive transformation of the person, who ceases to construct his world in communication with others, to lose himself in an autistic thought" (p. 1). In this view, one cannot be schizophrenic for just a few hours (as under the effect of a psychotomimetic drug) or for a few weeks or months (as in the so-called "acute schizophrenias" of the Anglo-Saxon tradition). The diagnosis of schizophrenia also is inapplicable to a stably delusional ideation that "does not touch the bonds relative to the contact of the Ego with the environment, affecting only a section of reality, or superimposing itself on it" (p. 1). From this arises the necessity of isolating from schizo-

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This article reflects research carried out through 1982; therefore, some more recent references may not be included.

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phrenia the so-called "bouffées delirantes" (which can precede the onset of schizophrenic psychosis, but from which complete recovery is possible) on the one hand, and the "chronic delusional psychoses without development toward defect" on the other.

The concept of "bouffée délirante," outlined by Magnan (1893) and his group, has been further developed by Ey (1954). The essential features of the syndrome are its sudden onset ("d'emblée"), the polymorphism of the themes of delusion ("délire protéiforme"), the intensity of the delusional experience, the preservation of an orientation and adaptation to the environment, and, finally, the alteration of mood (with elation and depression frequently coexisting). The episode comes to a sudden end, usually after a few weeks, and the risk of relapse is quite high (high enough to make Magnan suppose the existence of a sort of "faiblesse congénitale").

In the presence of an episode of this kind, it is very difficult to say whether we are dealing with an acute psychosis "sans conséquence, sinon sans lendemain" or with the first manifestation of a psychosis that will later reveal itself as schizophrenic. Ey notes that if we call these delusional psychoses acute schizophrenias, we "escape from a difficult decision," but at the same time we renounce the possibility of giving "diagnostic and prognostic significance to the notion of schizophrenia" (p. 1).

The French classification of the "chronic delusional psychoses without development toward defect" includes the "chronic systematized delusional psychoses," the "fantastic psychoses," and the "chronic hallucinatory psychoses" (Ey 1955).

The concept of "chronic hallucinatory psychosis" (De Clérambault 1942) is a relatively original product of the French school, and refers to a psychosis "characterized by the prominence of psychosensorial phenomena, in which delusion represents a secondary superstructure, and the contact with the environment is not impaired" (p. 455). The "chronic systematized delusional psychoses" (including, in their turn, the "passional delusions" and the "delusions of revindication") and the "fantastic psychoses" recall the Kraepelinian concepts of "paranoia" and "paraphrenia," respectively.

The three conditions described above, together with schizophrenia, constitute "the different species of a single genus, that of chronic delusional psychoses" (Ey 1955, p. 1).

The delimitation of the concept of schizophrenia is not so well articulated in the Italian (Bina and Bazzi 1949) and Spanish (Cabaleiro Goas 1953) schools. Their contribution is to separate from the four categories proposed by Bleuler (simple, hebephrenic, catatonic, and paranoid schizophrenia) the concept of "paranoia," which is used in the classic Kraepelinian sense. As in France, there is a traditional tendency in Italy and Spain to reject the notion of "acute schizophrenia" and to regard the evolution toward defect as essential for the diagnosis of schizophrenia.

Studies on the "Pseudoneurotic" Forms of Schizophrenia. Contributions about the "pseudoneurotic" forms of schizophrenia are found in both the Italian and French literature.

In Italy, one of the most significant studies is that of Rubino and Piro (1954), in which three types of syndromes "on the border between schizophrenia and neuroses" are distinguished: those bordering neurasthenia and schizophrenia (with hypochondriacal features), those bordering psychasthenia and schizophrenia (with a paranoid aspect), and those bordering hysteria and schizophrenia (resembling catatonia). All these forms show a chronic course, with alternating phases of deterioration and remission, but do not evolve into a full psychosis. Insight is partially preserved:

> [the subject] regards as morbid his somatic and cenesthetic . . . disturbances, but accepts as corresponding to reality his interpretative, subdelusional beliefs, which are not, however, as absurd as schizophrenic thought. [Rubino and Piro 1954, p. 2]

In France, Ey (1955) identifies in the articles of Claude and his group (Claude, Borel, and Robin 1924; Robin and Borel 1925) the first germ of the concept of "schizoneurosis." This concept describes a morbid condition characterized by a neurotic personality structure, on which acute episodes (the "schizomanic crises" of Claude) characterized by elation and a tendency to "rêverie" or, on the contrary, by mutism, refusal to eat, and violent impulses ("pseudo-catatonic crises") are superimposed. These crises are usually provoked by environmental factors (especially social or familial tensions) and followed by the full restoration of habitual neurotic attitudes.

Psychopathology

Phenomenological Analysis[1] of Schizoidia and Schizophrenia. The phenomenological analysis of the "essence" of schizoidia and schizophrenia finds its most significant expression, within the Southern European countries, in the writings of E. Minkowski, an author of Polish origin and Russian birth, who lived and worked in Paris.

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[1] Phenomenology, in the Husserlian meaning, signifies the study of experience ("to go back to things"), grasping its general essential content ("categorical intuition"). Phenomenological studies are limited to conscious experiences, of which they do not postulate causes, in the sense of natural sciences, and which they investigate from the point of view of the subject.

Minkowski developed (1953, 1966) the constitutional typology and concept of autism elaborated by Bleuler, integrating them with the philosophical conceptions of Husserl and Bergson.

He distinguished three main types of constitution: the syntone, the epileptoid, and the schizoid. The syntone is a person capable of the optimal kind of contact with the environment (continuative, adaptive, constructive). The epileptoid is identified with the "sensorial"; he lives in the concrete or, rather, in the "hyperconcrete," without being able to move away from it; and he "feels" much more than he "thinks." Lastly, the schizoid is identified with rationality; he feels at ease in the abstract, the firm, and the solid; he "thinks" more than he "feels"; he delimits, separates, and discerns; and he is "cold" and "distant."

The transition from schizoidia to schizophrenia can be characterized by the accentuation of this rationalistic tendency, to the point of "morbid rationalism"; or by the progressive loosening of, to the point of breaking off, the vital contact with reality.

In Italy, the most significant phenomenological contributions have been concerned not with the mode of being of the schizophrenic patient, but with experiences peculiar to the initial phases of schizophrenia, such as those of "fearful mutation" (Coppola 1961) and of "end of the world" (Callieri and Semerari 1954).

In the experience of "fearful mutation," the subject feels himself "overwhelmed" and "transformed" in a world which, in its turn, becomes more and more foreign and incomprehensible. Surrounding objects lose their usual significance, becoming fearfully obscure and threateningly allusive symbols. This transformation is experienced with a profound anguish and, sometimes, with a feeling of complete impotence. In the experience of "end of the world," the prominent feeling is that of collapse, "horrible and dreadful annihilation," and "foundering" of the whole universe.

These two "Erlebnisse" are substantially analogous, and very similar to those described by other European authors (Bleuler 1911; Mauz 1930; Müller-Suur 1950). Piro (1967) has noted that schizophrenics, although they are among the psychiatric patients with the highest degree of semantic freedom, tend to communicate their initial experiences by always using the same terms (mutation, transformation), and this is true in all languages.

Studies on Schizophrenic Thought and Language. The contributions of Italian and French authors in the field of the psychopathology of schizophrenic thought and language are particularly significant. It is in the Italian psychiatric tradition, for instance, that we can find the roots of the theory that schizophrenic language and thought represents a regression to primitive, archaic, "paleologic" models—a theory that has found its most complete expression, in the United States, in the work of Arieti (1955). As a matter of fact, it was Tanzi (1905) who first emphasized the cult that psychotic patients (just like the primitives) have for words ("logolatry") and the magic power (ability to modify the external world and their own person) that they ascribe to some verbal expressions.

This concept has been further developed by the French author Dumas (1934), who described the paranoid, like the primitive, as believing in the magic power of some words (especially the neologisms), as well as of some numbers and gestures.

Another notable Italian contribution is that of Barison (1949), who identified as an essential feature of schizophrenic thought the "tendency to formal abstraction," i.e., the schizophrenic's tendency to express himself at an abstract level even when the content of the thought is constituted by concrete facts. This tendency was interpreted by Barison (1949) as an attempt at an "instinctive reparation of the dissociations intervening between the ideas" (p. 430).

Important French studies include those of Teulié (1927), who distinguished in schizophrenic language automatic components that expressed a negative, defectual aspect, and constructive components (mainly represented by neologisms); and those of Delmond (1935), who emphasized the "phonetic primacy" of schizophrenic language, i.e., the prevalence of the phonetic connections over the semantic ones.

Finally, a complete study of the various alterations of language in schizophrenics was carried out by the Italian Piro (1967). He singled out as an essential characteristic of such language the "semantic dissociation," i.e., the weakening of the connection between the significant (the verbal sign) and its significance. Moreover, Piro introduced the notion of "fluctuaction of the semantic halo." The schizophrenic, in Piro's view, from time to time, tends to use concrete terms at a higher abstractive level, so that his language becomes metaphoric and sententious (increase of semantic halo), or else to use abstract terms in place of corresponding concrete ones, so that his language becomes ambiguous and indeterminate (restriction of semantic halo).

Etiology

The Biological[2] Approach. Research aimed at detecting the biological factors involved in the etiology and pathogenesis of schizophrenia flourished in the Southern European countries, particularly during the years between 1930 and 1960.

In Italy, great interest was shown in the theories of V.M. Buscaino (1924, 1953, 1958). Buscaino believed that schizophrenia should be considered as an "aminic toxicosis," with an anatomo-pathological substrate constituted, at the cerebral level, by small degenerative foci scattered through the white matter (from which would come the dissociative symptomatology) and in the basal ganglia (from which would come the extrapyramidal and catatonic symptomatology). At the origin of such toxicosis there would be a hepatic dysfunction, for whose existence the author supplied, within the limits allowed by the laboratory techniques available at that time, a series of proofs (mainly obtained in chronically hospitalized subjects whose conditions, due to inactivity, poor feeding, and somatic diseases, were far from optimal).

Among the "dissociative" symptoms arising from this biohumoral and anatomopathological substrate, V.M. Buscaino included phenomena such as verbal hallucinations, pseudohallucinations, and thought echoing. He maintained, in fact, that mental work is constantly accompanied, in the normal subject, by the articulation of words, and that the return to the center of the information relative to this last motor activity is essential for the thought to be "recognized as his own" by the subject. Buscaino theorized that in the schizophrenic, as a consequence of "dissociation," such a return component is missing. That is to say, the motor activity which accompanies his thought is sent to the periphery, but the relative information does not return to the center. This activity, therefore, is perceived as extraneous (Buscaino 1946, 1963).

The "aminotoxic" theory of schizophrenia was formulated before the demonstration of the psychotomimetic action of amine substances such as bulbocapnine, mescaline, and LSD, but received from it, obviously, further impetus.

A model to which both V.M. Buscaino (1933) and other authors (De Jong and Baruk 1930; De Giacomo 1951) attached special significance was the "catatonic" state induced in the experimental animal by the administration of bulbocapnine. It was held, in fact, that this "experimental catatonia" reproduced the spontaneously occurring human one. The report by Baruk (1933) of a catatonic condition appearing in several laboratory animals following the subcutaneous injection of colibacillary exotoxin derived from a human source was considered decisive proof of the "entero-hepatotoxic" etiology of schizophrenia.

Along with the studies of V.M. Buscaino and Baruk, a certain interest was awakened, in the same period, by those of Fiamberti (1948), who postulated the existence, within schizophrenia, of an "acetylcholine psychosis," resulting from a "deficit of acetylcholine at the level of the neurons with a psychic function" (p. 212). This theory stemmed from the therapeutic efficacy of so-called acetylcholine shock therapy (the intravenous injection of large doses of acetylcholine), as well as some experimental evidence, such as from the observation of a reduced level of cholinesterase in the serum of schizophrenic patients.

During the 1960s and 1970s, interesting contributions were made by Southern European researchers in support of the so-called "transmethylation hypothesis" of schizophrenia. We can cite, in particular, an article by G.A. Buscaino, Spadetta, and Carella (1969), in which it was claimed that the transformation of nicotinamide into its methylated derivatives increased in the presence of blood samples obtained from schizophrenic patients. In another influential article, Fazio et al. (1974) reported that a worsening of psychotic symptomatology occurred in schizophrenics treated with the methyl donor *S*-adenosylmethionine.

A revision of the "dopaminergic hypothesis" of schizophrenia was proposed by Lecrubier et al. (1980). They postulated the existence, in addition to the classic hyperdopaminergic form of schizophrenia, of a hypodopaminergic form (characterized by "negative" symptoms and sensitive to "disinhibiting" neuroleptics such as sulpiride whose mechanism of action is proposed to involve a blockade of dopamine presynaptic receptors).

Another line of research worth noting is that attempting to identify "genetic markers" of vulnerability to schizophrenia, to be used eventually for the early recognition of high-risk subjects. Among the proposed markers are the antigens of HLA system A9 and B7 (Cazzullo et al. 1974; Mercier et al. 1977) and dermatoglyphic characteristics (Kemali et al. 1976).

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[2] The term "biological" refers to the earlier studies, which ascribed to lesions of anatomic structures or to biochemical alterations of the organism (and, especially, of the central nervous system) the "cause" of mental diseases. It also refers to more recent studies, which explore the "biological correlates" of mental disturbances, eschewing the problem of etiology, or postulating for such disorders a multifactorial etiology.

A recent Spanish contribution of some interest is that of Lopez-Ibor et al. (1981), who reported evidence of cerebral atrophy in computed tomographic studies of a subgroup of schizophrenics.

The Psychological Approach. The contributions provided by psychoanalysis to the study of schizophrenia have been, at least in the Southern European countries, rather modest.

Some points raised in the writings of the French author J. Lacan (1966), however, seem worth noting. Lacan ascribes an essential role in the genesis of psychosis to a mother who does not attribute the function of law to the word of the father, thus preventing the child from attaining the "paternal metaphor," i.e., the father seen as an authority who separates the mother from the child. This attitude leaves the young subject involved in the dual relationship of identification with the mother and bars him from any possibility of access to the order of symbolism and language. In other words, if the mother treats the child as the complement of herself, as the phallus; if the child is everything for her, and confuses himself with her in a diffuse union, then the child will not be able to form his own individuality, nor will he be able to understand the meaning of the paternal figure. On the contrary, if the mother acknowledges the father's function to impose the social law (through the respect for his word), the child, acceding to the "paternal metaphor," will have access at the same time to the order of symbolism and language.

Among the studies focused on the features of communication in the families of schizophrenics, one should cite the contributions of Delay, Deniker, and Green (1962) and Selvini Palazzoli et al. (1975).

According to Delay, Deniker, and Green (1962), the most significant aspect emerging from the analysis of the configuration of the families of male schizophrenics is the symbolic destruction of the father, depending on external events (death or being away from home), on the outcome of conflicts (divorce or separation), or on a functional exclusion (as occurs when the father, although present, is a weak and inefficient figure, or establishes an excessive distance between himself and his son). In all these cases the subject is deprived of a living, tangible role model.

More complex, and directly inspired by the "double-bind" theory of the Palo Alto school (Bateson et al. 1956), is the approach of Selvini Palazzoli et al. (1975). They emphasized the "paradoxical" character of communication in the families of schizophrenics, in which every decision is blocked by the impossibility of divining an unequivocal meaning and, therefore, of choice. They also describe in such families a perverse mechanism of "homeostasis," which opposes any attempt to change.

The Sociological Approach. The sociological approach to schizophrenia became popular in the Southern European countries during the 1960s. It was a natural outgrowth, in a cultural context sensitized by the ferments of young people and by the enormous influence of Marxian ideology, of the theories elaborated by the Palo Alto group in America. These reseachers hypothesized that schizophrenic behavior is the "logically illogical" response to a familial environment in which communication depends upon paradoxical messages and contradictory signals.

Since it is likely that contradictions inherent to intrafamilial communication are the product of the influence of social environment, from which the family draws rules and behavioral codes, it is easy to arrive at the hypothesis that deviance is produced by a social structure marked by "generalized conflict." Such a social structure demands that the individual either adapt to its rules or be estranged as a deviant. Thus, the alienated individual is deviant only with respect to the rules of the society to which he belongs (Gabel 1962; Basaglia 1968; Jervis 1975). With this assertion, the biological and psychological paradigms are completely reversed, and the "specificity" of the mental illness is called into question.

It is not surprising that theoretical premises of this kind led to more or less radical operational proposals, which reflected the degree of "politicization" of their authors. Some theorists asserted that the sphere of action of a psychiatric intervention should be enlarged from the individual to his "relational network" (Hochmann 1971), since the individual pathology is a reflection of and is perpetuated by the network. Others declared that only the overthrow of the model of Western industrial and capitalistic society would lead to the elimination of its pathogenic consequences.

In this review, we will consider only the first approach, which has inspired the recent "alternative" trends in the field of psychiatry both in France and in Italy. These trends find their most coherent expression in the ideology of the "sector" (intended as a spatial unit and a cultural dimension), which Mignot (1967) summarized as follows:

> Mental health care is organized in sectors when, in a defined and sufficiently limited territory, the whole responsibility of prevention therapy and rehabilitation is entrusted to a single team, which can rely on a range of different structures. In this way, removal from the natural environment is avoided, since the reduced area of the sector involves only small displacements. Moreover, hospitalization becomes only one of the steps of the treatment, which has been started and will be continued by the organisms outside the hospital. [p. 1]

During the 1960s, this ideology inspired a number of enterprises both in France (such as that of the 12ᵉ Arrondissement of Paris) and in Italy (such as those of Varese, Gorizia, Trieste, and Bologna). In Italy, the ideology achieved official recognition in the well-known "Law 180" (subsequently absorbed in Sanitary Reform Law No. 833), which has sanctioned the closure of mental hospitals and the establishment of psychiatric services in the community.

The promulgation of this law has coincided, paradoxically, with a profound disorientation of alternative psychiatry in Italy. Jervis (1980) has become the spokesman of this uneasiness, underlining "the diffuse weariness for dogmatic attitudes" and condemning the "factious and acritical demonization of the opponents," the "contempt for culture and science," and the "oversimplified identifications of targets."

On the other hand, the application of the law, where it has been possible, has brought to light a number of problems, such as the risk of a "psychiatricization" of social discomfort, the creation of strongly repressive and authoritarian decentralized structures ("diffuse institution"), the abandonment of chronic patients (both those still present in the hospitals and those scattered in the community), and finally, the shifting of the burden of the costs of an eminently social problem onto families (Sergio 1980) due to the scarcity of facilities for transitional care.

These difficulties are most dramatic in environments that are still socially and economically backward, such as those in certain areas of Southern Italy (cf. Kemali, Gambini, and Gritti 1982). The deinstitutionalization movement has called attention to the need for better programming of psychiatric care (Torre and Marinoni 1982).

Observation and Intervention

Studies on the Course and Prognosis of Schizophrenia. The natural course of schizophrenia has been the subject of systematic studies by Fromenty (1937) and Ey (1955). Fromenty (1937) observed a continuous progressive course toward a pronounced defect in 70 percent of his patients, and an alternation of remissions and relapses in the remaining 30 percent. Within these latter patients, one half evolved, in the long term, toward defect, and the other half maintained a lasting remission of symptomatology. Ey (1955) reported a lasting remission after a single crisis in 25 percent of his cases; a lasting remission following several crises in another 22 percent; a continuous development toward chronicity after a single crisis in about 20 percent; and a deterioration after periods of acute crisis and remission in the remaining 33 percent.

According to Ey (1955), favorable prognostic signs are the pyknic biotype, the presence of mental diseases with a cyclic course in the family history, and the existence of important precipitating factors. Unfavorable signs are the leptosomic biotype and the slow, progressive evolution of disorder, discernible before the age of 20 years.

Studies on the Treatment of Schizophrenia. The contributions of Southern European psychiatry to the treatment of schizophrenia are very significant.

Electroconvulsive therapy, still employed with success in several countries, was introduced by two Italian psychiatrists, Cerletti and Bini (1938). Cerletti (1947) formulated a theory of the mechanism of action of this treatment, which postulated the production by the organism of "substances of supreme struggle" ("acroagonine").

The first attempts at psychosurgery in schizophrenia were performed by a Portuguese, the Nobel Prize winner E. Moniz (1936).

Two French psychiatrists, Delay and Deniker (1952), are credited with having introduced into the treatment of schizophrenic psychoses the first neuroleptic, chlorpromazine. To the French school we owe also several attempts to classify antipsychotic drugs on a clinical basis. Lambert and Revol (1960) divided these agents into sedative and incisive types, whereas Deniker (1977) classified them as sedative, medium, polyvalent, and disinhibitory. This classificatory approach, which is in contrast to the views prevailing in Anglo-Saxon countries, has also found favor among Italian clinicians.

French and Italian authors have made significant contributions to the debate concerning the mechanism of action of neuroleptics (Scatton 1981), the usefulness of monitoring neuroleptic plasma levels (Morselli 1981), and the validity of some biological indices, such as HLA antigens, as predictors of drug response (Smeraldi et al. 1976).

In the area of psychotherapy, the most important innovations from Southern European countries involve the so-called "institutional therapies." The French author Racamier (1970) has outlined a complex therapeutic strategy, involving the patient's activity, his relationships with his family, his social environment, and the dynamics within the institution. Therapy always passes, according to Racamier, through concrete objects (persons or things) called "mediators" and, depending on the degree of psychic disturbance, assumes a function of stimulus or guarantee of the subject's Ego.

Conclusion

The present review is not intended to be comprehensive. Some contributions, even very important ones, have undoubtedly been omitted, and for this we apologize to the relevant researchers and to the reader. As a partial excuse for the inevitable omissions, it should be noted that the distances between the Southern European countries (or, at least, between some of them), with regard to the circulation of ideas and scientific contributions, are still far greater than the geographical ones. The Italian reader, for instance, is certainly better informed on the progress of American or English psychiatry than on that of Spain or Portugal.

It is to be hoped that the present review, for all its omissions, will provide a stimulus for the deepening of scientific relationships between countries that have many traditions in common.

References

Arieti, S. *Interpretation of Schizophrenia.* New York: Brunner/Mazel, 1955.

Barison, F. Dissociazione e incomprensibilità schizofreniche. *Rivista di Neurologia,* 19:430-432, 1949.

Baruk, H. La catatonie colibacillaire expérimentale et clinique. *Annales Médico-Psychologiques,* 91 (II):449-469, 1933.

Basaglia, F. *L'Istituzione Negata.* Torino: Einaudi, 1968.

Bateson, G.; Jackson, D.; Haley, J.; and Weakland, J. Toward a theory of schizophrenia. *Behavioral Science,* 1:241-264, 1956.

Bini, L., and Bazzi, T. *La Schizofrenia.* Roma: Abruzzini, 1949.

Bleuler, E. Dementia praecox oder Gruppe der Schizofrenien. In: Aschaffenburg, K., ed. *Handbuch der Psychiatrie.* Wien: Deuticke, 1911.

Buscaino, G.A.; Spadetta, V.; and Carella, A. Il test di metilazione nella schizofrenia. *Acta Neurologica,* 24:113-118, 1969.

Buscaino, V.M. Dati riassuntivi sulla presenza di sostanze basiche abnormi e loro significato, nelle sindromi extrapiramidali, d'amenza e di demenza precoce. *Rivista di Patologia Nervosa e Mentale,* 29:501-509, 1924.

Buscaino, V.M. Catatonia sperimentale nell'uomo. *Rivista Sperimentale di Freniatria,* 57:374-384, 1933.

Buscaino, V.M. *Neurobiologia delle Percezioni.* Napoli: Edizioni Scientifiche Italiane, 1946.

Buscaino, V.M. Patologia extraneurale della schizofrenia. Fegato, tubo digerente, sistema reticoloendoteliale. *Acta Neurologica,* 8:1-60, 1953.

Buscaino, V.M. Pathogénèse et étiologie biologiques de la schizophrénie. *Acta Neurologica,* 13:1-26, 1958.

Buscaino, V.M. Udito e linguaggio dal punto di vista neurobiologico. X. pensiero e linguaggio. *Acta Neurologica,* 18:418-432, 1963.

Cabaleiro Goas, M. *Problèmes actuels des psychoses schizophréniques.* Madrid: Paz Montabro, 1953.

Callieri, B., and Semerari, A. Alcuni aspetti metodologici e critici della esperienza delirante di fine del mondo. *Rassegna di Studi Psichiatrici,* 63:55-77, 1954.

Cazzullo, C.L.; Smeraldi, E.; and Penati, G. The leucocyte antigenic system HLA as a possible genetic marker of schizophrenia. *British Journal of Psychiatry,* 125:25-27, 1974.

Cerletti, U. Sostanze di estrema difesa prodotte dall'elettroshock (a-croagonine). *Il Lavoro Neuropsichiatrico,* 1:367-399, 1947.

Cerletti, U., and Bini, L. L'elettroshock. *Archivio Generale di Neurologia, Psichiatria e Psicoanalisi,* 19:266-271, 1938.

Claude, H.; Borel, A.; and Robin, G. Demence precoce, schizomanie et schizophrénie. *Encéphale,* 19:145-149, 1924.

Coppola, C.F. Nota a proposito del mutamento pauroso. *L'Ospedale Psichiatrico,* 29:291-302, 1961.

De Clérambault, G.G. Automatisme mentale et psychoses hallucinatoires chroniques. In: *Oeuvre Psychiatrique.* Paris: Presses Universitaires, 1942. pp. 455-656.

De Giacomo, U. La catatonie toxique expérimentale. *Acta Neurologica,* 7:5-10, 1951.

De Jong, H., and Baruk, H. *La Catatonie Expérimentale par la Bulbocapnine.* Paris: Masson, 1930.

Delay, J., and Deniker, P. 38 cas de psychoses traités par la cure prolongée et continue de 4560 RP. In: *Comptes Rendus du lème Congrès des Aliénistes et Neurologues de Langue Française* Paris: Masson, 1952. pp. 497-502.

Delay, J.; Deniker, P.; and Green, A. Le milieu familial des schizophrénes, III. Resultats et hypotheses. *Encéphale,* 51:5-73, 1962.

Delmond, J. *Essai sur la Schizophasie.* Paris: Le Francois, 1935.

Deniker, P. *Entretiens de Bichat.* Paris: Expansion Scientifique Française Edit., 1977. p. 260.

Dumas, G. Mentalité paranoide et mentalité primitive. *Revue Neurologique,* 2:171, 1934.

Ey, H. Les bouffées délirantes et les psychoses hallucinatoires aigües. *Etudes Psychiatriques. Etude n. 23.* Paris: Desclée de Brouwer, 1954.

Ey, H. Groupe des schizophrénies. *Encyclopédie Médico Chirurgicale, Psychiatrie,* 37282, A10, 1955.

Fazio, C.; Andreoli, V.; Agnoli, A.; Casacchia, M.; Cerbo, R.; and Pinzello, A. Therapy of schizophrenia and depressive disorders with S-adenosyl-L-methionine. *Clinical Pharmacology and Therapeutics,* 2:1015, 1974.

Fiamberti, A.M. Terapie di shock e terapie a fondamento etiopatogenetico nella schizofrenia. *Rivista Sperimentale di Freniatria,* 72:212-218, 1948.

Fromenty, L. Les rémissions dans la schizophrénie. Statistique sur leur fréquence et leur durée avant l'insulinothérapie. *Encéphale,* 32:275-286, 1937.

Gabel, J. *La Fausse Conscience.* Paris: Ed. de Minuit, 1962.

Hochmann, J. *Pour une Psychiatrie Communautaire.* Paris: Ed. du Seuil, 1971.

Jervis, G. *Manuale Critico di Psichiatria.* Milano: Feltrunelli, 1975.

Jervis, G. Intervento. In: Onnis, L., and Lo Russo, G., eds. *Dove Va la Psichiatria?* Milano: Feltrinelli, 1980. pp. 68-97.

Kemali, D.; Gambini, F.; and Gritti, P. "Evaluation of Psychiatric Care Delivery After the Italian Reform Act." Presented at the International Symposium on "Alternatives to Institutional Psychiatric Care. Issues of Evaluation," Umea, Sweden, March 15-17, 1982.

Kemali, D.; Polani, N.; Polani, P.E.; and Amati, A. A dermatoglyphic study of 219 Italian schizophrenic males. *Clinical Genetics,* 9:51-60, 1976.

Lacan, J. *Ecrits.* Paris: Ed. du Seuil, 1966.

Lambert, P.A., and Revol, P. Classification psychopharmacologique et clinique des différents neuroleptiques. Indications thérapeutiques générales dans les psychoses. *Presse Médicale,* 68:1509-1511, 1960.

Lecrubier, Y.; Puech, A.J.; Simon, P.; and Widlöcher, D. Schizophrénie: hyper ou hypofonctionnement du système dopaminergique? Une hypothèse bipolaire. *Psychologie Médicale,* 12:2431-2441, 1980.

Lopez-Ibor, J.J., Jr.; Gardeur, D.; Messimy, R.; Metzger, J.; Trujillo, M.; Viano, J.; and Lopez-Ibor, L. "Computerized Tomography in Schizophrenia: Clinical and Morphological Correlations." (Abstract No. 5229) Presented at the III World Congress of Biological Psychiatry, Stockholm, June 28-July 3, 1981.

Magnan, V. *Leçons Cliniques.* Paris: Bataille, 1893.

Mauz, F. *Die Prognostik der Endogenen Psychoses.* Berlin: Thieme, 1930.

Mercier, P.; Sutter, J.M.; Julien, R.A.; and Kieffer, N. Schizophrénie: Association de la forme paranoide aux antigènes HLA A9 et B5. *Encéphale,* 3:49-53, 1977.

Mignot, H. Le secteur. *Encyclopédie Médico Chirurgicale. Psychiatrie,* 37915, A10, 1967.

Minkowski, E. *La Schizophrénie.* Paris: Desclée de Brouwere, 1953.

Minkowski, E. *Traité de Psychopathologie.* Paris: P.U.F., 1966.

Moniz. E. *Tentatives Opératoires dans le Traitement de Certaines Psychoses.* Paris: Masson, 1936.

Morselli, P.L. Clinical significance of neuroleptic plasma level monitoring. In: Usdin, E.; Dahl, S.G.; Gram, L.F.; and Lingjaerde, O., eds. *Clinical Pharmacology in Psychiatry.* London: MacMillan, 1981. pp. 199-210.

Müller-Suur, H. *Der Psychische Abnorme.* Berlin: Springer, 1950.

Piro, S. Il linguaggio schizofrenico. Milano: Feltrinelli, 1967.

Racamier, P.C. Le soin institutionnel des psychotiques: nature et fonction. *Information Psychiatrique,* 46:745-761, 1970.

Robin, G., and Borel, A. *Les Rêveurs Éveillés.* Paris: Gallimard, 1925.

Rubino, A., and Piro, S. Psicopatologia delle forme marginali tra nevrosi e schizofrenia. *Acta Neurologica,* Quaderno 7, 1954.

Scatton, B. Modes d'action biochimique des neuroleptiques. *Encéphale,* 7:201-214, 1981.

Selvini Palazzoli, M.; Boscolo, L.; Cecchin, G.; and Prata, G. *Paradosso e Controparadosso.* Milano: Feltrinelli, 1975.

Sergio, C. *Psichiatria senza Rete.* Cosenza: Lerici, 1980.

Smeraldi, E.; Bellodi, L.; Sacchetti, E.; and Cazzullo, C.L. The HLA system and clinical response to treatment with chlorpromazine. *British Journal of Psychiatry,* 129:486–489, 1976.

Tanzi, E. *Trattato delle Malattie Mentali.* Milano: Società Ed. Libraria, 1905.

Teulié, G. *Les Rapports des Langages Néologiques avec les Idées Délirantes en Médicine Mentale.* Paris: Picard, 1927.

Torre, E., and Marinoni, A. "Evaluation of a Psychiatric Service Before and After an Act Abolishing Large Mental Hospitals." Presented at the International Symposium on "Alternatives to Institutional Psychiatric Care. Issues of Evaluation," Umea, Sweden, March 15–17, 1982.

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Announcement

The first annual meeting of the **Pennsylvania Conference on Schizophrenia** will be held March 6-7, 1986, in the Bellevue Stratford Hotel, Philadelphia, Pennsylvania. The theme of this year's meeting is **"Schizophrenia and the Life Cycle."** The presentation of the first annual Arthur P. Noyes Award in Schizophrenia will be made at the meeting. The list of participants includes Bertram S. Brown, M.D., Hahnemann University; Carol M. Anderson, Ph.D., University of Pittsburgh; C. Christian Beels, M.D., New York State Psychiatric Institute; Sheila L. Cantor, M.D., University of Manitoba, Canada; Gerard Hogarty, M.S.W., University of Pittsburgh; Scott H. Nelson, M.D., Pennsylvania Office of Mental Health; Loren Roth, M.D., University of Pittsburgh; and George M. Simpson, M.D., Ch.B., The Medical College of Pennsylvania.

For further information about the meeting, please contact:

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Biological Studies of Schizophrenia in Europe

by Leonid Leonidovich Prilipko

Abstract

The main achievements in the field of genetics, biochemistry, and immunology of schizophrenia in the laboratories of European research centers are surveyed. Despite the rapid development of scientific research techniques and methodology, the abundant hypotheses on the pathogenesis of schizophrenia are riddled with so many contradictory facts that it is impossible to formulate any concrete model of the disease. One factor impeding the progress of biological research in schizophrenia is the inadequate development of standardized clinical descriptions. This is an obstacle to the study of clinical-biological correlates, which are among the principal criteria used to verify the importance of biological parameters chosen for study in the pathogenesis of the disease. A current strategy in the biology of schizophrenia, which may obviate some of those problems, is the discovery and study of biological markers.

Throughout the history of biological studies of schizophrenia, the appearance of new lines of research in this field has been determined by progress in the basic sciences. Every stage in the development of science that has been accompanied by the appearance of more precise techniques, the discovery of new phenomena, or the birth of ideas has led to a reexamination of the accumulated facts in biological psychiatry and to the formulation of new hypotheses.

At present, the study of schizophrenia is marked by a steady broadening of biological research into the nature of the disease and attempts to understand the material processes that determine its origin and development. A distinguishing feature of the biology of schizophrenia was, and still is, the problem created by the diversity of the clinical picture, course, and outcome of the disease. This creates exceptional difficulties in the definition of its nosological entity and the recognition of clinically homogeneous groups of patients—steps that are essential if adequate biological research is to be undertaken.

Nowadays, the biology of schizophrenia is a widely ramified field of psychiatry that has developed on the basis of progress in modern molecular biology, biochemistry, immunology, neuroanatomy, genetics, endocrinology, neurophysiology, and other basic sciences that determine the principal lines of research in this field of science.

The present review is limited chiefly to an examination of the principal achievements in the field of genetics, biochemistry, and immunology of schizophrenia from European research centers.

Genetic Aspects of Research in Schizophrenia

Proof of the role of hereditary factors in the genesis of schizophrenia is one of the principal arguments for the biological nature of this disease. Studies of the genetics of schizophrenia must therefore be classed without reservation as being among the most important trends in biological psychiatry.

The first 60 years of the history of the genetic study of schizophrenia in Europe (1911-73) have been a long

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period of accumulation of family histories and data on twins, and also of constant disputes over contradictory views on the general principles governing the inheritance of this disease. In the modern view, the advances made during this period, despite the extensive accumulation of empirical facts, may be regarded as relatively modest: from the point of view of techniques, the investigations carried out were imperfect and were bound to lead to disputed conclusions. The methodology of classical genetic analysis proved completely inadequate for the study of such complex phenotypical manifestations as the endogenous functional psychoses. This state of affairs was aggravated by some aspects of psychiatry itself, and in particular, the inadequate development of the tradition of studying strictly defined phenomena and using standardized clinical descriptions. Even today, these demands cannot be met sufficiently reliably, but they were, and still are, the basis for successful genetic analysis.

The imperfection and ambiguity of generalizations on the genetics of schizophrenia led a number of investigators in the 1960s to reevaluate the role of genetic and environmental factors in the development of mental disturbances. The basis for this reevaluation was provided by two original approaches to the comparative analysis of prevalence of the disease in families: (1) among blood relatives and foster relatives of adopted probands (Rosenthal et al. 1968) and (2) among half-siblings who are children of monozygotic twins, discordant for schizophrenia (Fischer 1973). In both cases conclusive proof was obtained of the important role of heredity in the development of disturbances associated with schizophrenia.

The modern period of genetic study of schizophrenia, which began in Europe in the 1970s, was thus preceded by real advances both in psychiatric genetics itself and in the field of the general theory of genetic-mathematical analysis of complex phenotypical manifestations. Modern requirements for the planning of genetic research in schizophrenia can be stated briefly as follows: (1) standardization of clinical descriptions based on criteria reproducible in different psychiatric settings and allowing elements of quantification of clinical manifestations; (2) epidemiological orientation toward the accumulation of data on families and populations, ensuring the genetic representativeness and statistical stability of the characteristics that are being looked for in genetic models; (3) a multidisciplinary type of study of the pathogenesis of the disease, with a view toward discovering pathogenetically important markers of the hereditary predisposition; and (4) the widespread use of modern methods of human analytical genetics, aided by computers (Gindilis and Vartanian 1979).

Two major investigations into the genetics of endogenous functional psychoses, based on different clinical material and using different analytical genetic methods, have been undertaken. A group of Soviet investigators (Gindilis and Shahmatova-Pavlova 1979; Moskalenko and Gindilis 1981) studied the extensive familial and epidemiological data gathered in the clinics and laboratories of the Research Institute of Clinical Psychiatry, All-Union Mental Health Research Center, Academy of Medical Sciences of the U.S.S.R., using genetic dispersion and multiparametric segregation analysis. A second group of investigators (Rao et al. 1981) used Wright's method of path analysis in a study of pooled empirical data gathered in countries of Western Europe and the United States. The major results of these two investigations were in essential agreement. It was found, for instance, that the genetic component of liability to exhibit mental disturbances within the schizophrenia spectrum extends to 60-70 percent of all the etiopathogenetic factors of this pathology. Meanwhile, systematic environmental factors common to all members of the family (the so-called "cultural inheritance"), accounting for 13-20 percent of the total, also were found to be significant. It was also demonstrated that there is a specific twin component of liability to the disease (about 10 percent of all factors), and this is a clear indication of the limitations of the twin method in its traditional interpretation. Finally, an important characteristic of the overall system of liability to schizophrenia was the significant influence of positive phenotypical assortativeness of mating.

In the past, the phenomenon of "anticipation," i.e., the earlier and more severe manifestation of a disease in a series of generations, was viewed as a "statistical artifact" or the simple result of differences in the way that families of an affected offspring are recorded. Recently, this interpretation has been revised. Relative to schizophrenia, the reality of the phenomenon has been tested on the basis of extensive factual material and stricter methods of analysis. It has been shown that the traditional view is valid only in relation to features with a simple dominant inheritance. In the case of multifactorial diseases with the trait present in both families, anticipation is an essential result of phenotypical assortativeness of mating (Trubnikov, Gindilis, and Shahmatova-Pavlova 1978).

Nonrandom choice of partners leads to an increase in the mean level of liability in the progeny because of an increase in homozygosity for genes involved in the general system of inherited predisposition in endogenous psychoses. A similar process, known as "oriented homozygotization," extends over three or four generations and then becomes exhausted for two reasons: (1) Persons with severe progressive forms of endogenous psychoses are usually unable to produce offspring and therefore do not fall into the next sample of parents. (2) Healthy siblings of such patients marry with a tendency toward negative phenotypical assortativeness, as a result of which the level of heterozygosity for the corresponding genes is higher in the offspring, and the mean index of liability to manifestation of the disease is consequently lowered. This concept provides an explanation for the genetic-population mechanisms of spread of endogenous psychoses as a whole and of schizophrenia in particular.

Another important branch of modern genetics of schizophrenia is the use of genetic-correlation analysis to establish the basis for pathogenetic systematics of clinical forms of this disease. The three widely adopted classifications of endogenous psychoses (those of Kraepelin, Leonhard, and Snezhnevsky) were subjected to comparative analysis on the basis of genetic criteria independent of any a priori clinical concept. It was shown (Ungvari 1982, 1983) that as a first approximation the best agreement between real groupings of patients by genotypical similarity is obtained with Snezhnevsky's clinical classification, based on assessment of the character of the course and progressiveness of the psychopathic process. The syndromal principle of differentiation between clinically homogeneous groups of patients may prove constructive, under these circumstances, only within the limits of forms already differentiated and, moreover, allowing for the stereotype of development of the psychosis.

Jakimov-Venulet (1981) reports a similar investigation, the aim of which was to study genetic differences between bipolar and unipolar affective disorders. The investigation showed that liability to the development of both forms includes an important hereditary component, described most adequately by a multifactorial threshold model. The results do not allow bipolar and unipolar affective psychoses to be regarded as independent genetic entities. On the basis of the family records he studied, Jakimov-Venulet established a weak but significant genetic correlation between liability to develop bipolar and liability to develop unipolar affective disorders. Similar results regarding the heterogeneity of affective psychoses were obtained by Angst, Frey, and Zerbin-Rudin (1980).

The results of a collaborative study conducted under the aegis of the World Health Organization, in which four international centers took part (three of them in European countries), should be discussed in connection with the problem of genetic heterogeneity of affective psychoses (Gershon et al. 1980). In this investigation the hypothesis of possible genetic linking of the principal gene of the bipolar form of manic-depressive psychosis with the X chromosome was tested. However, the results did not confirm this hypothesis unequivocally, for it was impossible to demonstrate a close linking between the parameters studied. At the same time, these workers did not rule out the possibility of genetic heterogeneity of the bipolar form itself, and they suggested that some of its clinical variants may be genetically linked with the X chromosome, in particular, in the region of the color blindness locus.

The results mentioned above illustrate a well-known fact—namely, that many difficulties in the use of formal genetic analysis in mental diseases stem not only from the considerable difficulties in clinical diagnosis, but also from the small number of informative families in real genealogical samples. Further evidence of this is given by the work of Debray, Caillard, and Stewart (1978, 1979), who analyzed a large number of simple and complex genetic models of inheritance of schizophrenia. However, these workers were unable to make an unambiguous choice among the models they studied, mainly because of the insufficient volume of available genealogical material.

One other main trend in the study of the role of inheritance in the development of endogenous psychoses is the search for their genetic markers. This line of research is closely linked with other sections of biological psychiatry (e.g., biochemistry and immunology) and is currently in a state of active development. Discovery of genetic markers in endogenous psychoses must be the scientific basis for medical genetic counseling and the identification of increased risk groups—an extremely important development for the successful solution of such difficult problems (for this particular group of diseases) as those involved in prevention (Vartanian 1983).

In conclusion, it should be emphasized that this short survey of European investigations of the role of hereditary factors in the development of schizophrenia cannot claim to be a full account of the problem, so that the impression may be obtained that genetic research into endogenous psychoses in this region is fragmentary in character. This impression, however, may be the result, in particular, of the fact that in some cases strong traditions in science on the genetics of human behavior—for example, Slater's school (cf. Shields and Gottesman 1971)—originated in Europe, but later developed more intensively in other regions of the world.

On the other hand, a trend appeared, for example, in Scandinavian countries (Strömgren 1938; Böök 1953; Larsson and Sjögren 1954) that continued with research by later workers (Lindelius 1970) and is still developing vigorously today (Wetterberg 1982).

Genetic research in Europe is currently devoted to a wide range of aspects of the role of hereditary factors in mental diseases. However, there is insufficient consolidation of these investigations with modern problems in clinical psychiatry. At the same time, many difficult problems in psychiatry—especially problems in the diagnosis and clinical standardization of nosological forms—which clinical psychiatry continues to tackle with its own resources, could probably be solved more quickly and more effectively on the basis of clinical-genetic investigations. The situation that has arisen in these fields of science appears to justify an attempt to combine the efforts of research workers within the framework of a major project, whose aim could be a goal-directed approach to the fundamental problem of genetics of endogenous psychoses on the basis of a unified methodology.

Biochemical Hypotheses of Schizophrenia

Modern biochemical hypotheses of schizophrenia were preceded by many years of research during the period from the 1940s to the 1960s. The aim of this research was to discover and identify abnormal products of metabolism in patients with schizophrenia. Investigators confined their attention to the study of the toxic action of biological fluids and tissue extracts on the behavior and physiological functions of animals, and also of various biological objects. The efforts directed toward isolation and identification of these "toxic factors" from schizophrenic patients led to very contradictory conclusions, and this state of affairs is reflected in a number of general reviews (Vartanian 1966; Lozovsky 1968). Nevertheless, the stage of searching for the biochemical basis of schizophrenia was in many respects highly instructive, and played an important role in the development of modern views on the nature of the pathogenesis of this disease.

In particular, it became evident that the study of the biochemical basis of psychoses without regard to clinical features of their development (the type of course, relations between positive and negative symptoms, the existence of hereditary traits, and so on) is unpromising. Interaction between psychotropic drugs and the chemical systems of the body (catecholamines, indoleamines, and certain enzyme and receptor proteins of nerve and other tissue membranes) considerably modifies the state of these systems, making it difficult to evaluate any biochemical disturbances discovered, or their link with the true mechanisms of the disease. Consequently, in the course of biochemical research, a complex problem of selection of groups of patients suitable for biochemical study has arisen.

Besides the factors mentioned above, which may possibly affect the end result of the investigation, such characteristics of samples of patients and control groups as, for example, sex, age, diet, time of year, and time of day must be taken into account. These methodological precautions make modern biochemical investigations much more difficult to undertake, and even slight deviations from these conditions may introduce an element of contradiction into the results. However, despite all the difficulties, intensive biochemical searches for a biological substrate in schizophrenia have continued and have led to the formulation of several hypotheses.

One of these is the dopamine hypothesis of schizophrenia, which has stimulated a wide range of investigations in Europe as in the rest of the world. This hypothesis is based on the view that the dopamine system in schizophrenic patients is hyperactive (Randrup and Munkvard 1972), a view based on findings indicating that chemical compounds (amphetamine, apomorphine, methylphenidate, and L-dopa) which elevate concentrations of dopamine in the brain, at the same time, induce mental states analogous to acute paranoid schizophrenia. At the same time, it has been observed that most antipsychotic drugs (e.g., phenothiazines and butyrophenones) appreciably reduce the activity of dopamine-dependent systems in the body (Van Pragg 1977; Fredrickson and Richelson 1979). One result of these observations has been a large series of studies attempting to estimate the concentration of dopamine and its metabolites in the cerebrospinal fluid (CSF) of patients and in brain tissues post-mortem.

However, most investigators have been unable to find significant, still less specific, changes in the concentrations of dopamine and its metabolites in schizophrenia (Crow et al. 1979*a*; Rodnight 1983). The results of an investigation in which an elevated dopamine level was found in post-mortem brain tissues of schizophrenic patients (Bird, Spokes, and Iversen 1979; Mackay et al. 1982), or in which hyperactivity of the sympathoadrenal system in the phase of exacerbation of the disease was discovered in the course of clinical-biochemical tests (Vasiliev 1980), must be regarded with caution. The extensive material available, providing evidence of a wide variety of changes in neurotransmitter metabolism arising under the influence of neuroleptics, must also be taken into account under these circumstances (Le Fur et al. 1979; Mitchell and Doggett 1980; Tissari 1982).

The subsequent development of research exploring the dopamine hypothesis is reflected in a study of activity of dopamine-β-hydroxylase, an enzyme converting dopamine into noradrenalin. Comparative analysis of the activity of this enzyme in six different parts of brains from schizophrenic patients revealed no abnormality (Crow 1979). At the same time, however, there is evidence of reduced activity of dopamine-β-hydroxylase in the peripheral blood of schizophrenic patients (Arató et al. 1982).

Recent years have seen a sharp increase of interest in the study of dopamine receptors in the limbic region and corpus striatum. For instance, an increase in the density of dopamine receptors in the striatum and nucleus accumbens, correlating mainly with positive symptoms of schizophrenia (Mackay et al. 1982), was demonstrated in post-mortem material from schizophrenic patients (Owen et al. 1978, 1981*b*). A more detailed analysis of this finding led to the discovery of an increased density of D_2 receptors in tissues of the striatum isolated after death from the brains of untreated patients, and absence of any such changes in the properties of D_1 receptors. From this, Cross, Crow, and Owen (1981) concluded that the increase in number of D_2 receptors is linked with the pathogenesis of schizophrenia. However, this conclusion requires rigorous testing, for there is much evidence of significant changes in the properties of different neuronal receptors (including receptors for dopamine) under the influence of psychotropic drugs (Clow et al. 1980; Hall and Ögren 1981; Murugaiah et al. 1982).

Another approach that may be used to evaluate dopamine-dependent systems in schizophrenic patients is the study of prolactin levels in patients' plasma and CSF (Cotes, Crow, and Johnstone 1978; Pusmurova et al. 1979; Naber et al. 1980). Secretion of prolactin from the pituitary is controlled by the brain dopamine system, so that any hyperactivity of that system ought to lead to an increase in the blood level of the hormone. However, no appreciable changes in prolactin levels were found in untreated patients. In the course of these investigations, correlation between changes in the psychotic state of the patients and the prolactin level in the body could be detected only during treatment with neuroleptics.

It should also be pointed out that interpretations of the effect of dopamine agonists on the mental state of schizophrenic patients are currently being revised. For example, it has been shown that in some cases apomorphine does not induce a psychosis, but causes an improvement in the patient's psychotic state (Corsini et al. 1981). This effect, according to these investigators, is evidence of predominant stimulation of presynaptic receptors, leading to a fall in the level of dopamine secreted and, correspondingly, reduced stimulation of dopamine receptors on the postsynaptic membrane.

Hence, with all the experimental data now available, it can be confidently asserted that deviations in the function of the dopamine system exist in schizophrenia. It must be recognized, however, that the dopamine hypothesis, in its present form, is still an example of a highly simplified, although productive biological model of the pathogenesis of schizophrenia. Substantial progress apparently will only be achieved after an awareness is gained of the principles of interaction between different biochemical systems of the human brain.

Attempts have already been made to use an integral approach to explain disturbances of the biochemical homeostasis of the brain discovered in schizophrenia (Heimann 1983). For instance, the author of a critical survey (Hornykiewicz 1978) suggests that at least three basic systems of the brain are involved in the development of the schizophrenic process: the reticular formation of the forebrain and the midbrain, the limbic system of the forebrain, and the corpus striatum. In Hornykiewicz' opinion, this is manifested as a complex combination of disturbances of the noradrenergic and dopaminergic systems of the brain, and it accordingly suggests that other neurotransmitters in addition to dopamine are also involved in the pathophysiological process: serotonin, acetylcholine, γ-aminobutyric acid, and noradrenalin.

In recent years interest has been renewed in the study of the noradrenergic system, whose hyperactive state in schizophrenia is supported by several facts. For example, an increase in the noradrenalin concentration in the limbic region and putamen has been demonstrated in post-mortem material from patients with schizophrenia (Farley et al. 1978; Crow et al. 1979*a*). The study of the CSF revealed a significant elevation in the level of this neurotransmitter in patients in the acute phase of illness (Kemali, Del Vecchio, and Maj 1982). Similar results were obtained in an investigation of untreated patients with chronic schizophrenia (Gomes et al. 1980). Elevation of the noradrenalin level also was demonstrated in peripheral blood plasma (Kemali, Del Vecchio, and Maj 1982). In the last case, however, the authors express doubts about the etiological significance of the results. Some particularly interesting results were obtained in a study (Kemali et al. 1984) in which a significant correlation was demonstrated between the increase in the noradrenalin concentration in the CSF and computerized electroencephalographic findings, indicating an excited state of the patients. The correlation thus revealed confirms a connection between the hyperactive state of the noradrenergic system of patients with schizophrenia and factors inducing an anxiety state. However, any conclusions about the role of these factors in the pathophysiological process in schizophrenia must await further study.

In the mid-1970s the development of modern biochemical methods, in particular, acted as a stimulus to the intensive study of the role of serotonin in the pathogenesis of schizophrenia. The development of this line of research was a corollary to the now traditional methodology of biochemical research in the field of biological psychiatry. The principal approaches were: (1) determination of concentrations of serotonin and its metabolites in the body fluids of patients with schizophrenia (urine, blood, and CSF); (2) use of post-mortem material for direct evaluation of the state of the serotonin system of the brain (determination of metabolite levels and studies of receptors); (3) the study of platelets as an extracerebral model in order to analyze the rate of metabolism of serotonin and the mechanisms of its uptake and release, and also to determine the changes in the properties of serotonin receptors with time during the development of the disease.

With the accumulation of often contradictory experimental results, the problems of interpretation intensified. First and foremost, this applies to the figures reflecting the level of 5-hydroxyindoleacetic acid (5-HIAA), the principal metabolite of serotonin, in the peripheral blood and urine of schizophrenic patients. The problems of interpretation are not particularly surprising, for the concentrations of serotonin and its metabolites that reflect their metabolism in the brain do not account for more than 1-2 percent of the main pool of these compounds at the periphery.

Contradictory results also were obtained in CSF studies of patients with schizophrenia. Wode-Helgodt et al. (1977) and Gomes et al. (1980) found a very slightly increased concentration of 5-HIAA in CSF, whereas Ashcroft et al. (1966) and Gattaz, Waldmeir, and Beckmann (1982) demonstrated a significant decrease. In the Gattaz study, it was shown that the fall in the 5-HIAA level is not connected with treatment. In this connection, attention is drawn to data obtained in several laboratories in the U.S.A., which showed a fall in the 5-HIAA concentration in the CSF and an increase in the serotonin concentration in the blood and platelets of patients with chronic schizophrenia, with abnormalities detectable by computed tomography (DeLisi et al. 1981; Potkin et al. 1983; Stahl et al. 1983).

No less contradictory results were obtained by the study of post-mortem material. Winblad et al. (1979), investigating serotonin and 5-HIAA concentrations in different parts of the brain, found a decreased level of these compounds in schizophrenics. Meanwhile Crow et al. (1979*a*) found an elevated serotonin concentration in the putamen. Still another conclusion was reached by Joseph et al. (1979), who found no abnormalities in the levels of tryptophan, 5-HIAA, and kinurenin in the brain tissues of schizophrenics. Negative results were obtained in an attempt to find differences in the properties of serotonin receptors in tissues of the frontal cortex of schizophrenic patients (Whitaker, Crao, and Ferrier 1981). Fillon and Fillon (1981) demonstrated the ability of antidepressants to induce marked changes in the properties of serotonin receptors, a fact which makes special care necessary in the interpretation of results of receptor studies, both in brain tissues and in platelets, in patients with various mental diseases.

The studies reviewed above reflect the typical situation confronting investigators who have tried to evaluate the role of serotonin in the pathogenesis of schizophrenia. At present, therefore, we can only hope that the development of fresh approaches will lead to more productive research in this direction. As an example of the most promising of these approaches, the preliminary selection of patients on the basis of computed tomography and the study of mechanisms of action of psychotropic drugs, modifying the state of the serotonin system in the human body selectively, may be mentioned.

Another promising trend in biological psychiatry is, as before, the study of enzymes participating in the metabolism of biogenic amines.

The properties of monoamine oxidase (MAO) in schizophrenia and other mental diseases have received the most detailed study in recent years. In the modern view (Gorkin 1981), two types of this enzyme can be distinguished: MAO of types A and B. Type A monoamine oxidases are called amine oxidases, and their activity is blocked by low concentrations of clorgyline; noradrenalin and serotonin are specific substrates for MAO A. The activity of type B MAO is inhibited only by much higher concentrations of clorgyline; deprenyl is a selective inhibitor of this type of MAO, and its specific substrates are benzylamine and the biogenic monoamines 2-phenylethylamine and *N*-methylhistamine.

Interest in the study of MAO in mental diseases is based on two suppositions. The first is that reduction or alteration of the substrate specificity of MAO must lead to a disturbance of metabolism of vital neurotransmitters and may be accompanied by high body levels of amines such as, for example, *N,N*-dimethyltryptamine, a powerful psychotomimetic. The second supposition is based on the idea that parameters reflecting MAO activity in the body can be used as biological markers.

However, to date, all attempts to obtain proof of the validity of the first hypothesis, based on the study of post-mortem material, have ended in failure. For instance, Crow et al. (1979*a*) and Reveley et al. (1981) used biochemical methods to study MAO activity in various parts of the brain of schizophrenics and a control group. They used a broad spectrum of substrates for this purpose, suitable for estimation of activity of MAO of both type A and type B. However, these workers were unable to find any differences between the groups studied. These results are in good agreement with those of electron cytochemistry of the brain in schizophrenia (Anders and Orlovskaya 1982).

It must be pointed out, however, that these negative results were obtained during investigations of unpurified MAO preparations without separation of the numerous forms of this enzyme. The first positive results in this direction, obtained by the present writer jointly with the staff of Gorkin's laboratory (Moskvitina et al., in press), are therefore interesting. The concentration of SH-groups and substrate specificity of a purified MAO fraction from the cerebral cortex of 14 schizophrenic patients and 6 mentally healthy individuals were studied. Both in normal subjects and in schizophrenics, the enzyme was shown to be present in a partially oxidized form. The purified enzyme contains two SH-groups per 10^5 daltons of protein and, besides MAO substrates (serotonin and β-phenylethylamine), it also deaminates the substrate of diamine oxidase (histamine). Reduction of partially oxidized SH-groups of MAO to 15 SH-groups per 10^5 daltons of protein in the fraction obtained from schizophrenics, unlike that from normal subjects, does not lead to disappearance of histamine-deaminase activity but, on the contrary, potentiates it considerably. These authors suggest the existence of structural changes in MAO in schizophrenia and discuss their findings in connection with activation of lipid peroxidation, discovered in such patients (Prilipko and Liedeman 1982*b*; Prilipko 1984). On the basis of these preliminary findings, a more detailed study of the properties of the many different forms of monoamine oxidase in various brain structures in endogenous psychoses appears indicated.

By contrast with the few studies of MAO conducted on post-mortem material, there is a very extensive literature mainly describing reduced activity of this enzyme in platelets of patients with chronic schizophrenia (see, for reference, Wyatt, Potkin, and Murphy 1979; Sandler et al. 1981). There is no doubt at the present time that MAO activity in the platelets is reduced in schizophrenia, but the view that this phenomenon plays a role in the pathogenesis of the disease is being drastically revised. For example, it has been shown that neuroleptics can significantly reduce MAO activity both *in vivo* and *in vitro* (Gattaz et al. 1981; Owen et al. 1981*a*; Del Vecchio et al. 1983). The presence of low-molecular-weight proteins inhibiting platelet MAO activity has been demonstrated in the plasma of schizophrenic patients and healthy blood donors (Becker and Giambalo 1982). A correlation has been found between parameters characterizing MAO activity in the platelets and the sex of the individuals studied; this indicates the necessity for strict verification of results obtained for this parameter (Gattaz et al. 1981).

Evidence in support of the role of depressed platelet MAO activity in the pathogenesis of schizophrenia could be provided by the changes mentioned above in biogenic amine metabolism. However, no direct proof of the presence of correlation between parameters reflecting MAO activity and the neurotransmitter level *in vivo* has yet been obtained.

Some particularly interesting research in this direction was undertaken by Reveley et al. (1983), whose results prove convincingly that depression of MAO activity in schizophrenia can be explained by genetic factors and not by the influence of the disease or its treatment. As a result of twin investigations, these workers showed that platelet MAO activity is largely under genetic control, but at the same time environmental factors do exert some influence. No significant differences were found within pairs of monozygotic twins discordant for schizophrenia. On the whole, however, MAO activity in these twins with a schizophrenic genotype was 25-28 percent lower than that in a group of healthy monozygotic twins and of individuals born singly. The degree of genetic control over the activity of this enzyme is determined quantitatively at 70-80 percent. These observations are in agreement with the results obtained by Winter et al. (1978), who studied platelet MAO activity in healthy twins and also showed that the activity of this enzyme is under genetic control. The results of these investigations suggest that a parameter characterizing MAO activity in platelets may be used as a biological marker for schizophrenia.

Nevertheless, in the light of the data examined above, it must be recognized that the monoamine oxidase hypothesis of schizophrenia demands further analysis, taking many factors capable of influencing the end results of the investigations into consideration, including the role of the distinctive clinical features of the disease.

In the mid-1970s, a new and promising trend in biological psychiatry appeared, aimed chiefly at the study of neuropeptides, which are low-molecular-weight compounds with unusually high and diverse biological activity. In a short period, many investigations were reported, and in most of them special attention was paid to the study of endogenous morphines during the development of mental disorders, including those of schizophrenia. Typical representatives of the group of compounds are α-, β-, and γ- endorphins, methionine-endorphin, and leucine-endorphin, which from the structural point of view are fragments of the β-lipotropin molecule.

Two competing hypotheses linking endogenous opiates to the development of schizophrenia currently coexist. One of them postulates a decrease in the concentrations of endogenous opiates in the body; the other postulates the directly opposite state of affairs.

Recent research to test the validity of these hypotheses has developed along traditional lines (direct determination of these compounds in the patients' tissues and body fluids), and also in the form of psychopharmacological investigations based on the study of the effect of agonists and antagonists of opiate receptors on the patients' mental state.

It must be admitted that the results of determination of endorphin levels in the body fluids of schizophrenic patients are still highly contradictory. For example, it follows from the work of Hole et al. (1979) and of Drysdale et al. (1982) that a fraction with increased ability to activate opiate receptors can be isolated from the urine and blood of patients with schizophrenia. In the case of blood, it has been shown that a fraction isolated from the blood of schizophrenics induces a state of hyperactivity in animals, and *in vitro* it inhibits binding of ^{3}H-naloxone. On the basis of data such as these, an attempt was made to explain the therapeutic effect of hemodialysis in schizophrenia, which has been observed in a series of cases. In recent publications, however, the therapeutic effectiveness of this procedure was not confirmed (Vanherweghem, Linkowski, and Mendlewicz 1983). Meanwhile Emrich et al. (1979) found no difference between the plasma β-endorphin concentration in schizophrenic patients and in patients with other mental disorders.

A careful study of the CSF by a group of Swedish workers (Terenius et al. 1976; Rimón, Terenius, and Kampman 1980) revealed an increase in opioid activity in patients with schizophrenia. However, in another study carried out in the United States, directly opposite results were obtained (Naber et al. 1981). The authors cited found a decrease in opioid activity in the CSF of schizophrenic males.

It is still too early to attempt to explain these contradictions, for the research is still in a stage of development, and the existing data are clearly insufficient to allow valid analysis.

The results of studies of the pharmacological action of agonists and antagonists of the opiate receptors likewise do not yet permit any unambiguous conclusion to be drawn on the role of endogenous opiates in the pathogenesis of schizophrenia. Several detailed surveys, covering virtually all the principal results obtained in the last 6 years of research in this field, have recently been published (Davis, Buchsbaum, and Bunney 1979; Mueser and Dysken 1983). We can therefore be content here with a short comment on the present status of this research. Endogenous opiates undoubtedly play an important role in the formation of an individual's mental status, but the role of these compounds in the development of schizophrenia has not been defined.

This indeterminacy is due to the existence of directly opposite pharmacological effects of endorphins and other agonists of opiate receptors. The nonproportional dependence of the pharmacological effect of the preparation on its dose, differences in susceptibility of individuals due to previous treatment with psychotropic drugs, and, of course, clinical heterogeneity of the populations of patients studied are currently regarded as possible explanations for these opposite effects.

The same list of explanations may be equally applicable to the diversity of results obtained in studies using antagonists of opiate receptors as a probe to assess the role of the opiate system in the development of schizophrenia. An example of one of the most extensive investigations of the therapeutic effect of an opiate antagonist, namely naloxone, in schizophrenia is the combined study undertaken as part of an international program of the World Health Organization, in which seven research centers from different regions of the world participated (Pickar et al. 1982). The sample comprised 32 patients with schizophrenia and 26 patients with manic-depressive psychosis. The effect of naloxone proved most marked in a group of schizophrenics who continued to receive psychotropic drugs; in particular, their hallucinatory manifestations were appreciably diminished. In the group of untreated patients, the diminution of hallucinatory manifestations was substantially less marked and the patients' condition as a whole actually worsened. A more detailed analysis of the section of this investigation conducted in the U.S.S.R. (Liedeman et al. 1980) showed that clinically homogeneous groups of patients with manic-depressive psychosis and schizophrenia were extremely heterogeneous in their response to naloxone. Some patients did not respond to naloxone at all, whereas administration of the drug to others led to a transient (for a few hours) improvement of their clinical state. Patients with mania became less manic, and patients with schizophrenia became less hallucinatory. Thus, the endorphin system is evidently involved in processes determining the clinical manifestations of the disease in some patients with endogenous psychoses.

Further evidence in support of the above hypothesis is found in the results obtained by a group of workers from The Netherlands (van Praag et al. 1982). In a series of investigations, this group of workers found a positive therapeutic effect of des-tyrosine-γ-endorphin (DTγE) in the treatment of schizophrenia. This peptide belongs to the endogenous endorphin family but does not possess opiate-like activity. A few days after the beginning of treatment with DTγE, van Praag et al. (1982) observed an improvement in the clinical state in about 70 percent of schizophrenic patients. This effect was reproduced when the course of treatment was repeated. It must be pointed out, however, that other investigators were unable to confirm these results (Emrich et al. 1980). On the basis of analysis of the data so far available on the use of DTγE in schizophrenia, the authors of a critical survey (Manchanda and Hirsch 1982) concluded that the question of the therapeutic efficacy of this peptide still awaits an answer. Nevertheless, the results so far available indicate that further study of neuropeptides as part of biological research in schizophrenia may prove fruitful.

Only a few of the modern lines of research in the field of biochemical investigation of schizophrenia have been examined in this survey. Other, no less intriguing, biochemical hypotheses exist, and they are attracting the attention of a large group of investigators. They include such topics as, for example, the study of methylation processes in endogenous psychoses, the role of γ-aminobutyric acid in the regulation of the dopamine system in schizophrenia, and so on. This gap may perhaps be filled by the series of excellent reviews by Baldessarini, Stramentionoli, and Lipinski (1979), Berger (1981), and Rodnight (1983), among many others.

Immunological Components of the Pathogenesis of Schizophrenia

The immunological subdivision of biological psychiatry was developed in Europe at the beginning of the present century. The idea of a possible disturbance of the immune system in mental diseases was first put forward by the Russian investigator Horoshko (1912). The role of autoimmunization in the development of mental diseases also was postulated by Snesarev (1934).

The development of fundamentals of classical immunology also stimulated the formulation of various hypotheses linking immunopathology to schizophrenia. Among the first of these was the infectious theory of schizophrenia, which postulated an etiological and pathogenetic role in the development of schizophrenia for a change in the intestinal and coccal flora of the human body (Buscaino 1953). The infectious hypothesis was soon followed by that of the viral etiology of schizophrenia, based on the discovery of virus-like particles in the CSF and nasal secretions of a certain proportion of schizophrenic patients (M.A. Morozov 1954*a*;

V.M. Morozov 1954*b*). The virological study of the CSF in schizophrenia was further developed by Italian investigators from Buscaino's clinic (Scarlato and Mastogiovanni 1956) and also in the U.S.S.R. (Malis 1959).

It must be pointed out, however, that in the 1950s and 1960s the results of these investigations aroused strong skepticism on the part of psychiatrists, and the low standard of the research techniques available at that time did not permit supporters of the viral hypothesis of schizophrenia to develop their ideas successfully, so that interest in this field of biological psychiatry waned appreciably.

The viral hypothesis of schizophrenia is currently experiencing its rebirth, and this was largely stimulated by the discovery of latent viruses (Gajdusek and Gibbs 1977). Interest in the role of viruses in the pathogenesis of endogenous psychoses has definitely increased in the last few years in the European region. Groups from Great Britain, Finland, Czechoslovakia, Sweden, and Belgium are pursuing active research in this direction. One result of their investigations has been the discovery of virus-like particles with a marked cytopathic effect in the CSF of schizophrenics (Crow et al. 1979*b*; Tyrrell et al. 1979). The interferon level and the titer of antibodies against herpes viruses have been shown to be elevated in patients with schizophrenia (Libikova et al. 1979*a*, 1979*b*). Epidemiological studies have yielded facts which, in Hare's (1983) opinion, support the viral theory of schizophrenia. Suggestions regarding the possible role of viral infection in changes in neurotransmitter metabolism in schizophrenia are now being discussed in the literature (Koos 1984). Attempts are being made to examine results of histopathological investigations as well as data from computed tomography of the brain of schizophrenic patients in the light of the viral hypothesis (Crow 1983; Rimón 1983; van Kammen and DeLisi 1984).

Despite definite experimental advances, it must be recognized that no unequivocal proof of the leading role of viruses in the genesis of schizophrenia has yet been obtained. Nevertheless, the prospects of viral research in the study of the etiology and pathogenesis of schizophrenia are now regarded with much less skepticism than previously.

More substantial progress has been achieved in the field of the autoimmune hypothesis of schizophrenia. The first positive results were obtained in Europe in 1937–39 by Lehmann-Facius, who found antibodies against brain antigens, extracted from brain tissues with organic solvents, in the blood serum and CSF of patients with schizophrenia (Lehmann-Facius 1937). The results acted as a stimulus for the systematic study of antibrain antibodies in this disease. In the 1960s, it was established that schizophrenia is accompanied by clearly defined autoimmune reactions with the appearance of brain antigens and antibodies against brain tissue in the blood stream (Vartanian 1963). Clinical-biological correlations have shown that autoimmune processes, manifested in schizophrenia by the appearance of brain antigens and antibodies against them in the peripheral blood reflect the acuteness of the disease, and the rate and severity of its course (Kolyaskina and Kushner 1969; Semenov and Glebov 1969). The appearance of antigens in the blood stream, moreover, usually precedes the appearance of antibrain antibodies (Glebov 1970). With the appearance of more sophisticated preparative immunochemical methods, many investigations attempted to evaluate the level of the main classes of immunoglobulins (IgG, IgM, IgA) in the CSF and blood serum of schizophrenic patients (Durell and Archer 1976). In the course of analysis of the results of these investigations, the first feature to draw attention was the contradictory nature of the results. Some workers found a marked increase in the levels of all three types of immunoglobulins in patients with both acute and chronic forms of schizophrenia (Amkraut, Solomon, and Allansmith 1973; Domino et al. 1975; Zarrabi, Zucker, and Miller 1979). Others discovered a significant increase in the IgM level (Pulkkinen 1977) and, finally, a series of experiments by Bock, Weeke, and Rafaelsen (1971) revealed a significant decrease in the IgM concentration and no change in the IgG and IgA levels.

Clearer results were obtained by the use of brain-specific proteins as antigens in immunochemical reactions. For instance, Vartanian et al. (1978) found a significant rise in the titer of antibodies against "fraction 10," containing several types of brain-specific proteins, the characteristics of which were described by Klyushnik and Burbaeva (1983) and Zayko et al. (1984). The use of neurospecific protein S-100 in skin tests revealed a positive reaction of delayed type in patients with schizophrenia, and in the opinion of the workers concerned (Jankovic, Jakulic, and Horvat 1980), this indicated participation of reactions of humoral and cellular immunity in the pathogenesis of schizophrenia.

A link between the characteristics of reactions in cellular immunity and the pathophysiological process in schizophrenia is supported by the results of an extensive systematic study of peripheral blood lymphocytes (Kolyaskina 1983; Prilipko and Liedeman 1982*b*). The discovery of enhanced blast transformation of blood lymphocytes from patients with schizophrenia in response to the addition of brain antigen to the culture (Kolyaskina 1972) can be regarded as the beginning of research in this direction.

A subsequent study of T lymphocytes revealed a decrease in the relative percentage of these cells in the peripheral blood of patients with schizophrenia (Loseva 1977; Duorakova, Zuolosky, and Herzog 1980), including a decrease in the proportion of T-suppressor cells (Maznina et al. 1984). A study of the functional state of these cells showed a decrease in proliferative activity of the T lymphocytes in response to phytohemagglutinin, due to biologically active factors in the blood serum of schizophrenic patients (Babayan, Sekoyan, and Prilipko 1976; Kerepcic, Bamburac, and Jurin 1979; Pivovarova and Kolyaskina 1980). Later investigations showed that antibodies against O-antigen, present on membranes both of nerve cells and T lymphocytes, may be one of the factors capable of changing the number of lymphocytes in patients with schizophrenia and their function both *in vitro* and *in vivo* (Luria and Domashneva 1974; Kushner and Maznina 1980). This fact was confirmed in a detailed study of antithymic immune factor in schizophrenia, as a part of the World Health Organization Collaborative Program. Seven international research centers in biological psychiatry, including six centers in the European region, took part (Kolyaskina et al. 1980).

In addition to the topics reviewed above, the morphology and physiological state of the lymphoid cells of schizophrenics have been analyzed. This approach revealed an increase in the subpopulation of activated lymphocytes with atypical morphology, due to the effect of membranotropic factors in the patients' blood serum (Prilipko and Liedeman 1982*a*). Data indicating the existence of a subpopulation of activated B lymphocytes in the peripheral blood of schizophrenic patients have been reported in recent years (Mach, Schutts, and Borner 1983).

The results as a whole indicate the existence of changes in the immune status of patients with schizophrenia. However, may difficulties still remain to be solved before we can understand the true causes of the change in the immune responses in this disease. For instance, one of them may be the effect of psychotropic drugs on the patient's immunological reactivity, including the function of his immunocompetent cells through the direct action of xenobiotics on receptors of the plasma membranes of the lymphocytes (Loseva 1981; Zozulja, Parsakova, and Kost 1982; Shaskan et al. 1983). It is thus too early to draw unequivocal conclusions on the autoimmune nature of schizophrenia, but an autoimmune component does appear to be implicated in the pathogenesis of this disease.

We may conclude this short survey of immunological research in schizophrenia by stating that many of these investigations in Europe are linked with the study of human leukocytic antigens of the HLA system, controlled by several loci: A, B, C, and D, which are closely interlinked and are located in the sixth human chromosome.

The association between antigens of the HLA system and various clinical parameters in schizophrenia has now been investigated in some detail, but experimental data in this field are still extremely contradictory (Goudeman et al. 1981). For instance, a positive association of the disease with HLA-A9 has been found in data for the Swedish and Czech populations (Eberhard, Franzen, and Low 1975; Ivanyi, Zemek, and Jvanya 1978). Italian workers (Cazzullo, Smeraldi, and Penati 1974; Cazzullo and Smeraldi 1979) found an increase in the frequency of HLA-A11 and a decrease in the frequency of A3 and A10. A decrease in the frequency of A10 and B5 was found in France in a group of 75 patients with chronic schizophrenia (Singer et al. 1982). In the German population, a statistically significant increase in the frequency of HLA-A27 has been demonstrated both in the total group of patients with schizophrenia and in certain subgroups (in patients with paranoid schizophrenia, in patients with a chronic type of disease with a poor prognosis, and in patients in whom the disease began before the age of 20 years). An increase in the frequency of HLA-A9 was discovered in these same subgroups (Gattaz and Beckmann 1982). In the U.S.S.R., on the basis of an investigation of 87 patients with different forms of schizophrenia and 130 normal blood donors, an increase in the frequency of HLA-A10 was found in patients with a continuous type of course, together with an association of HLA-B12 with an episodic type of schizophrenia (Mitkevich, 1981; Mitkevich and Eliava 1983).

These results encourage the hope that a more detailed analysis of the association of antigens of the HLA system with the clinical parameters of the disease will explain contradictions in the data reported to date.

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Conclusion

This survey of recent progress in the field of biological psychiatry, although restricted to an examination of the main results obtained in the fields of genetics, biochemistry, and immunology of schizophrenia, nevertheless provides evidence of the rapid development of research methodology and techniques in this field. The scope of the biological problems has broadened dramatically in concert with the appearance of new and the development of traditional lines of research in biological psychiatry. Advances are due, in particular, to progress in basic research in the biochemistry of the central nervous system, in molecular genetics, and in modern immunology.

It must be recognized, however, that the many different hypotheses of the pathogenesis of schizophrenia are still riddled with gaps and contradictory experimental facts, so that a concrete model of the pathogenesis of this disease cannot yet be formulated. One explanation for the failure to overcome these difficulties, as already mentioned, is the polymorphism of schizophrenia. It is responsible for the inadequate development of standardized clinical descriptions—a serious difficulty impeding the study of clinical-biological correlates, since diagnosis is one of the principal criteria used to test the significance of biological parameters in the pathogenesis of the disease.

Another objective difficulty is the lack of adequate biological models of schizophrenia, and this is aggravated by the latent contradiction between the principles of experimental biology, which envisages in most cases active intervention on the test object, and the classical postulate of medicine, which prohibits any intervention that may harm the patient.

These circumstances determine the current strategy of research into the biology of schizophrenia, which is to discover and study biological markers, i.e., parameters whose changes in schizophrenia reflect the pathophysiological process or accompany its development.

References

Amkraut, A.; Solomon, G.F.; and Allansmith, H. Immunoglobulins and improvement in acute schizophrenic reactions. *Archives of General Psychiatry*, 28:673-677, 1973.

Anders, V., and Orlovskaya, D. Mediator enzymes in the brain of patients with schizophrenia. *Vestnik Akademii Meditsinskih Nauk SSSR*, 1:68-73, 1982.

Angst, J.; Frey, R.; and Zerbin-Rudin, E. Bipolar manic-depressive psychoses: Results of a genetic investigation. *Human Genetics* 55:237-254, 1980.

Arató, M.; Bagdy, G.; Blumel, F.; Perenyi, A.; and Rihmer, Z. Csökkent szérum dopamin-β-hidroxiláz aktivatas paranoid schizopréniaban. *Ideggyogyaszati Szemle*, 35:355-360, 1982.

Ashcroft, G.; Crawford, T.; Eccleston, D.; Sharman, D.; MacDoudall, E.; Stanton, J.; and Binns, J. 5-Hydroxyindole compounds in the cerebrospinal fluid of patients with psychiatric or neurological disease. *Lancet*, II:1049-1052, 1966.

Babayan, N.G.; Sekoyan, R.V.; and Prilipko, L.L. The effect of the serum of schizophrenic patients and of normal donors on the DNA synthesis in lymphocytes. *Bulletin of Experimental Biology and Medicine*, 81:430-432, 1976.

Baldessarini, R.; Stramentionoli, G.; and Lipinski, J. Methylation hypothesis. *Archives of General Psychiatry*, 36:303-307, 1979.

Becker, R., and Giambalo, C. Endogenous modulation of monoamine oxidase in schizophrenic and normal humans. *American Journal of Psychiatry*, 139:1567-1570, 1982.

Berger, P. Biochemistry and the schizophrenias: Old concepts and hypotheses. *Journal of Nervous and Mental Disease*, 169:90-99, 1981.

Bird, E.D.; Spokes, E.G.S.; and Iversen, L.L. Increased dopamine concentration in limbic areas of brain from patients dying with schizophrenia. *Brain*, 102:347-360, 1979.

Bock, E.; Weeke, B.; and Rafaelsen, O.J. Serum proteins in acutely psychotic patients. *Journal of Psychiatric Research*, 9:1-9, 1971.

Book, J.A. Schizophrenia as a gene mutation. *Acta Genetica et Statistica Medica*, 4:133-139, 1953.

Buscaino, V.M. Patalogia extraneurale della schizophrenia. *Acta Neurologica*, 8:1-7, 1953.

Cazzullo, C.L.; Smeraldi, E.; and Penati, G. The leucocyte antigenic system HL-A as a possible genetic marker of schizophrenia. *British Journal of Psychiatry*, 125:25-27, 1974.

Cazzullo, C.L., and Smeraldi, E. HLA system, psychiatry and psychopharmacology. *Progress in Neurology and Psychopharmacology*, 3:137-146, 1979.

Clow, A.; Theodorou, A.; Jenner, P.; and Marsden, C.D. Changes in rat striatal dopamine turnover and receptor activity during one year's neuroleptic administration. *European Journal of Pharmacology*, 63:135-144, 1980.

Corsini, G.U.; Pitzalis, G.F.; Bernardi, F.; Bocchetta, A.; and Del Zompo, M. The use of dopamine agonists in the treatment of schizophrenia. *Neuropharmacology*, 20:1309–1313, 1981.

Cotes, P.M.; Crow, T.J.; and Johnstone, E.C. Serum prolactin as an index of dopamine receptor blockade in acute schizophrenia. *British Journal of Clinical Pharmacology*, 4:651–655, 1978.

Cross, A.J.; Crow, T.J.; and Owen, F. ^{3}H-Flupenthixol binding in postmortem brains of schizophrenics: Evidence for a selective increase in dopamine D_2 receptors. *Psychopharmacology*, 74:122–124, 1981.

Crow, T.J. Catecholamine reward pathways and schizophrenia: The mechanism of the antipsychotic effect and the site of the primary disturbance. *Federation Proceedings*, 38:2462–2467, 1979.

Crow, T.J. Is schizophrenia an infectious disease? *Lancet*, I:173–175, 1983.

Crow, T.J.; Baker, H.; Cross, A.; Joseph, M.; Lofthouse, R.; Longden, A.; Owen, F.; Riley, G.; Glover, V.; and Killpack, W. Monoamine mechanisms in chronic schizophrenia: Post mortem neurochemical findings. *British Journal of Psychiatry*, 134:249–256, 1979*a*.

Crow, T.J.; Ferrier, I.N.; Johnstone, E.C.; MacMillan, J.F.; Owens, D.G.C.; Parry, R.P.; and Tyrrell, D.A.G. Characteristics of patients with schizophrenia or neurological disorder and virus-like agent in cerebrospinal fluid. *Lancet*, I:842–844, 1979*b*.

Davis, G.; Buchsbaum, M.S.; and Bunney, W.E., Jr. Research in endorphins and schizophrenia. *Schizophrenia Bulletin*, 5:244–250, 1979.

DeLisi, L.; Neckers, L.; Weinberger, D.; and Wyatt, R.J. Increased whole blood serotonin concentrations in chronic schizophrenic patients. *Archives of General Psychiatry*, 38:647–650, 1981.

Debray, Q.; Caillard, V.; and Stewart, G. Schizophrenia: A study of genetic models and some of their implications. *Neuropsychobiology*, 4:257–269, 1978.

Debray, Q.; Caillard, V.; and Stewart, G. Schizophrenia: A study of genetic models. *Human Heredity*, 29:27–36, 1979.

DeLisi, L. Is immune dysfunction associated with schizophrenia? A review of the data. *Psychopharmacology Bulletin*, 20:509–513, 1984.

Del Vecchio, M.; Maj, M.; D'Ambrosio, A.; and Kemali, D. Low platelet MAO activity in chronic schizophrenics: A long-term effect of neuroleptic treatment? *Psychopharmacology*, 79:177–179, 1983.

Domino, E.F.; Krause, R.R.; Thiessen, M.M.; and Batsakis, J.G. Blood protein fraction comparisons of normal and schizophrenic patients. *Archives of General Psychiatry*, 32:717–721, 1975.

Drysdale, A.; Deacon, R.; Lewis, P.; Olley, J.; Electricwala, A.; and Sherwood, R. A peptide-containing fraction of plasma from schizophrenic patients which binds to opiate receptors and induces hyperreactivity in rats. *Neuroscience*, 7:1567–1573, 1982.

Duorakova, M.; Zuolosky, P.; and Herzog, P. Endogenous psychoses and T and B lymphocytes. *Folia Haematologica*, 107:221–228, 1980.

Durell, J., and Archer, E. Plasma proteins in schizophrenia: A review. *Schizophrenia Bulletin*, 2:147–159, 1976.

Eberhard, G.; Franzen, G.; and Low, B. Schizophrenia susceptibility and HLA antigens. *Neuropsychobiology*, 1:211–217, 1975.

Emrich, H.; Holit, V.; Kissling, W.; Fischer, M.; Laspe, H.; Heinemann, H.; von Zerssen, D.; and Herz, A. β-Endorphin-like immunoreactivity in cerebrospinal fluid and plasma of patients with schizophrenia and other neuropsychiatric disorders. *Pharmakopsychiatria*, 12:269–276, 1979.

Emrich, H.; Zandig, M.; Kissling, W.; Dirlich, G.; von Zerssen, D.; and Herz,, A. Des-tyrosyl-γ-endorphin in schizophrenia: A double blind trial in 13 patients. *Pharmakopsychiatrie*, 13:290–298, 1980.

Farley, I.J.; Price, K.S.; McCullough, E.; Deck, J.N.; Hordynski, W.; and Hornykiewicz, O. Norepinephrine in chronic paranoid schizophrenia: Above normal level in limbic forebrain. *Science*, 200:465–468, 1978.

Fillon, G., and Fillon, M. Modulation of affinity of postsynaptic serotonin receptors by antidepressant drugs. *Nature*, 292:349–351, 1981.

Fischer, M. *Genetic and Environmental Factors in Schizophrenia*. Copenhagen: Munksgaard, 1973.

Fredrickson, P., and Richelson, E. Dopamine and schizophrenia—A review. *Journal of Clinical Psychiatry*, 40:399–405, 1979.

Gajdusek, D., and Gibbs, C. Kuru, Creutzfeldt-Jakob disease, and transmissible presenile dementias. In: ter Meulen, Katz. *Slow Virus Infections of the Central Nervous System*. New York: Springer, 1977. pp. 15–49.

Gattaz, W.F., and Beckmann, H. O sistema de histocompatibilidade nas esquizofrenias. *Arquivos de Neuropsyquiatria*, 40:223–229, 1982.

Gattaz, W.; Casper, S.; Propping, P.; Friedl, W.; and Beckmann, H. Low platelet MAO activity and schizophrenia: Sex differences. *Acta Psychiatrica Scandinavica*, 64:165-174, 1981.

Gattaz, W.; Waldmeir, P.; and Beckmann, H. CSF monoamine metabolites in schizophrenic patients. *Acta Psychiatrica Scandinavica*, 66:350-360, 1982.

Gershon, E.; Mendlewicz, J.; Gastpar, M.; Bech, P.; Goldin, L.; Kielholz, P.; Rafaelsen, O.; Vartanian, F.; and Bunney, W.E., Jr. A collaborative study of genetic linkage of bipolar manic-depressive illness and red/green colorblindness. *Acta Psychiatrica Scandinavica*, 61:319-338, 1980.

Gindilis, V., and Shahmatova-Pavlova, I. Analysis of genetic factors of clinical polymorphism in endogenous functional psychosis. *Vestnik Akademii Meditsinskih Nauk SSSR*, 7:14-23, 1979.

Gindilis, V., and Vartanian, M. Diseases with hereditary predisposition. In: *Materials of 14th International Congress of Genetics: Genetics and Medicine*. Moscow: Medizina, 1979. pp. 89-101.

Glebov, V.S. The succession of immunization by brain antigens of the organism in the period of exacerbation of the schizophrenic process. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 70:580-596, 1970.

Gomes, V.C.R.; Shanley, B.C.; Potgieter, L.; and Roux, J.T. Noradrenergic overactivity in chronic schizophrenia: Evidence based on cerebrospinal fluid noradrenaline and cyclic nucleotide concentrations. *British Journal of Psychiatry*, 137:346-351, 1980.

Gorkin, V.Z. *Amine Oxidases: Medical Aspects*. Moscow: Medizina, 1981.

Goudeman, M.; Goudeman, J.; Parguet, P.J.; and Fontan, M. Psychoses schizophreniques et antigenes HLA: Données personneles et revue de la literature. *Encephale*, 7:609-622, 1981.

Hall, H., and Ogren, S.-O. Effects of antidepressant drugs on different receptors in the brain. *European Journal of Pharmacology*, 70:393-407, 1981.

Hare, E.H. Epidemiological evidence for a viral factor in the aetiology of the functional psychoses. *Advances in Biological Psychiatry*, 12:52-75, 1983.

Heimann, H. Biologie der Schizophrenie. *Schweizer Archiv fur Neurologie und Psychiatrie*, 132:193-205, 1983.

Hole, K.; Bergslien, H.; Jorensen, H.; Berge, O.; Reichelt, K.; and Trygstad, O. A peptide-containing fraction in the urine of schizophrenic patients which stimulates opiate receptors and inhibits dopamine uptake. *Neuroscience*, 4:1883-1893, 1979.

Hornykiewicz, O. Psychopharmacological implications of dopamine antagonists: A critical evaluation of current evidence. *Neuroscience*, 3:773-783, 1978.

Horoshko, V.K. *Reactii Zivotnogo Organisma na Vveedenie Nervnoi Tkani (Nevrotoxini, Anafilaksia, Endotoksini)*. Moskva: Moskovskii Universitet, 1912.

Ivanyi, D.; Zemek, P.; and Jvanya, P. HLA antigens as a possible marker of heterogeneity in schizophrenia. *Journal of Immunogenetics*, 5:165-172, 1978.

Jakimoy-Venulet, B. Hereditary factors in the pathogenesis of affective illnesses. *British Journal of Psychiatry*, 139:450-456, 1981.

Jankovic, B.D.; Jakulic, S.; and Horvat, J. Schizophrenia and other psychiatric diseases: Evidence for neurotissue hypersensitivity. *Clinical and Experimental Immunology*, 40:515-522, 1980.

Joseph, M.; Baker, H.; Crow, T.J.; Riley, G.; and Risby, D. Tryptophan metabolism in schizophrenia: A post mortem study of the serotonin and kynurenine pathways in schizophrenic and control subjects. *Psychopharmacology*, 62:279-285, 1979.

Kemali, D.; Del Vecchio, M.; and Maj, M. Increased noradrenaline levels in CSF and plasma of schizophrenic patients. *Biological Psychiatry*, 17:711-717, 1982.

Kemali, D.; Maj, M.; Iorio, G.; Marciano, F.; Nolfe, G.; Lovero, N.; and Campajola, P. The overarousal hypothesis of schizophrenia: Relationship between CSF noradrenaline levels, C-EEG indicators of activation and psychosis ratings. In: Kemali, D.; Morozov, P.V.; and Toffano, G., eds. *New Research Strategies in Biological Psychiatry*. London: John Libbey, 1984. pp. 185-190.

Kerepcic, I.; Bamburac, J.; and Jurin, M. Immunological events in schizophrenic patients. *Periodicum Biologorum*, 81:221-224, 1979.

Klyushnik, T., and Burbaeva, G. Isolation and some physicochemical properties of a new neurospecific protein 10-40-4 from human brain. *Biokhimiia*, 48:1203-1208, 1983.

Kolyaskina, G.I. Some peculiarities of lymphocytes of schizophrenic patients in conditions of cultivating: 2. Study of peripheral lymphocytes reactions to implantation of tissue antigens. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 72:61-65, 1972.

Kolyaskina, G.I. Blood lymphocytes in schizophrenia: Immunological and virological aspects. *Advances in Biological Psychiatry*, 12:142-149, 1983.

Kolyaskina, G.I.; Kielholz, P.; Bunney, W.E., Jr.; Rafaelsen, O.; Coppen, A.; Hippius, H.; and Vartanian, F. Antithymic immune factor in schizophrenia: A WHO study. *Neuropsychobiology*, 6:349–355, 1980.

Kolyaskina, G.I., and Kushner, S.G. Concerning some regularities in the appearance of antibrain antibodies in the blood serum of schizophrenic patients. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 69:1679–1683, 1969.

Kushner, S.G., and Maznina, T.P. Concerning the nature of antigens, the antibodies to which are detected in the blood-serum of schizophrenic patients. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 80:1071–1074, 1980.

Larsson, T., and Sjogren, T. Methodological, psychiatric and statistical study of a large Swedish rural population. *Acta Psychiatrica et Neurologica Scandinavica*, Suppl. N89:40–63, 1954.

Le Fur, G.; Burgevin, M.-C.; Malgouris, C.; and Uzan, A. Differential effects of typical and atypical neuroleptics on α-noradrenergic and dopaminergic postsynaptic receptors. *Neuropharmacology*, 18:591–594, 1979.

Lehmann-Facius, H. Uber die Liquordiagnose der Schizophrenien. *Klinische Wochenschrift*, 16:1646–1648, 1937.

Libikova, H.; Breir, S.; Kosikova, M.; Pogady, J.; Stunzner, D.; and Ujhazyova, D. Assay of interferon and viral antibodies in the cerebrospinal fluid in clinical neurology and psychiatry. *Acta Biologica et Medica Germanica*, 38:879–893, 1979*a*.

Libikova, H.; Pogady, J.; Rajcani, J.; Skodacek, I.; Ciamper, F.; and Kosikova, M. Latent herpes virus hominis I in the central nervous system of psychotic patients. *Acta Virologica*, 23:231–239, 1979*b*.

Liedeman, R.; Panteleeva, G.; Tsutsulkovskaya, M.; Vartanian, F.; and Belyaev, B. Naloxone administration in patients with endogenous psychoses. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 80:231–237, 1980.

Lindelius, R., ed. A study of schizophrenia: A clinical, prognostic, and family investigation. *Acta Psychiatrica Scandinavica*, Suppl. 216. Copenhagen: Munksgaard, 1970. p. 125.

Loseva, T.M. Thymus-dependent lymphocytes in reaction of spontaneous rosette-formation in schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 77:692–695, 1977.

Loseva, T.M. Examination of lymphocytes B in the peripheral blood of schizophrenic patients by the complementary rosette method. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 81:697–700, 1981.

Lozovsky, D.V. The biochemical investigation of schizophrenia in the USSR. *International Review of Neurobiology*, 7:199–225, 1968.

Luria, E.A., and Domashneva, I.V. Antibodies to thymocytes in sera of patients with schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 71:235–236, 1974.

Mach, D.M.; Schutts, C.; and Borner, I. Schizophrenic und B-Lymphozyten-alteration—eine Hypothese. *Psychiatrie, Neurologie und medizinische Psychologie*, 35:390–397, 1983.

Mackay, A.V.P.; Iversen, L.L.; Rossoz, M.; Spokes, E.; Bird, E.; Arregui, A.; Creese, I.; and Snyder, S.H. Increased brain dopamine and dopamine receptors in schizophrenia. *Archives of General Psychiatry*, 39:991–997, 1982.

Malis, G. Yu. *On the Aetiology of Schizophrenia*. Moscow: Medizina, 1959.

Manchandra, R., and Hirsch, S. (Des-Tyr¹)-γ-Endorphin for schizophrenia: A critical review. *International Pharmacopsychiatry*, 17:147–152, 1982.

Maznina, T.P.; Mikheeva, T.V.; Lavrov, V.E.; Abramova, L.I.; Kolyaskina, G.I.; and Tsutsulkovskaya, M. Ya. T lymphocyte suppressors in juvenile continuously progressive schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 84:1065–1069, 1984.

Mitchell, P.R., and Doggett, N.S. Neuroleptic blockade of stimulus-induced glutamate release from striatal tissue. *Neuroscience Letters*, Supplement 5:S271, 1980.

Mitkevich, S.P. HLA antigens and schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 81:1016–1018, 1981.

Mitkevich, S.P., and Eliava, V.N. Histocompatibility antigens and remissions in schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 83:119–121, 1983.

Morozov, M.A. The virus-like agent in schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 54:735–740, 1954*a*.

Morozov, V.M. On the problem of the viral etiology of schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 54:732–734, 1954*b*.

Moskalenko, V., and Gindilis, V. Twin concordance and partition of phenotypic variance in schizophrenia. *Tsitologiia i Genetica*, 15:70–72, 1981.

Moskvitina, T.; Kamushanskaya, N.; Smirnov, A.; Prilipko L.; and

Gorkin, V. Brain monoamine oxidase in schizophrenia. *Bulletin of Experimental Biology and Medicine*, in press.

Mueser, K., and Dysken, M. Narcotic antagonists in schizophrenia: A methodological review. *Schizophrenia Bulletin*, 9:213–225, 1983.

Murugaiah, K.; Theodorou, A.; Mann, S.; Clow, A.; Jenner, P.; and Marsden, C.D. Chronic continuous administration of neuroleptic drugs alters cerebral dopamine receptors and increases spontaneous dopaminergic action in the striatum. *Nature*, 296:570–572, 1982.

Naber, D.; Ackenheil, M.; Laakman, G.; Fisher, H.; and von Werder, K. Basal and stimulated levels of prolactin, TSH and LH in serum of chronic schizophrenic patients, long-term treated with neuroleptics. *Pharmakopsychiatrie*, 13:325–330, 1980.

Naber, D.; Pickar, D.; Post, R.; Van Kammen, D.; Waters, R.; Ballenger, J.; Goodwin, F.; and Bunney, W.E., Jr. Endogenous opioid activity and β-endorphin immunoreactivity in CSF of psychiatric patients and normal volunteers. *American Journal of Psychiatry*, 138:1457–1462, 1981.

Owen, F.; Bourne, R.; Crow, T.; Fadhli, A.; and Johnstone, E. Platelet monoamine oxidase activity in acute schizophrenia: Relationship to symptomatology and neuroleptic medication. *British Journal of Psychiatry*, 139:16–22, 1981*a*.

Owen, F.; Cross, A.J.; Crow, T.J.; Poulter, M.; and Waddington, J.L. Increased dopamine receptors in schizophrenia: Specificity and relationship to drugs and symptomatology. In: Perris, C.; Struve, G; and Jansson, B., eds. *Biological Psychiatry*. Amsterdam: Elsevier/North-Holland, 1981*b*. pp. 699–706.

Owen, F.; Crow, T.J.; Poulter, M.; Cross, A.J.; Longden, A.; and Riley, G.J. Increased dopamine receptor sensitivity in schizophrenia. *Lancet*, II:223–226, 1978.

Pickar, D.; Vartanian, F.; Bunney, W.E., Jr.; Maier, H.; Gastpar, M.; Prakash, R.; Sethi, B.; Liedeman, R.; Belyaev, B.S.; Tsutsulkovskaja, M.; Jungkunz, G.; Nedopil, N.; Verhoeven, W.; and van Praag, H. Short-term naloxone administration in schizophrenic and manic patients. *Archives of General Psychiatry*, 39:313–319, 1982.

Pivovarova, A.I., and Kolyaskina, G.I. Effect of various doses of phytomitogens on the blood lymphocytes proliferation in schizophrenia, *Bulletin of Experimental Biology and Medicine*, 7:552–554, 1980.

Pusmurova, M.; Hiener, V.; Marek, J.; Filip, V.; and Sramkova, J. Prolactin and somatotropin responses to TRH in schizophrenic patients. *Activitas Nervosa Superior*, 21:171–173, 1979.

Potkin, S.; Weinberger, D.; Linnoila, M.; and Wyatt, R.J. Low CSF 5-hydroxyindoleacetic acid in schizophrenic patients with enlarged cerebral ventricles. *American Journal of Psychiatry*, 140:21–25, 1983.

Prilipko, L.L. Activation of lipid peroxidation under stress and in schizophrenia. In: Kemali, D.; Morozov, M.; and Toffano, G., eds. *New Research Strategies in Biological Psychiatry: New Prospects*. Vol. 3. London: John Libbey, 1984. pp. 175–184.

Prilipko, L.L., and Liedeman, R.R. Lymphocytes of peripheral blood—Biological marker of pathophysiological processes in schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 82:79–86, 1982*a*.

Prilipko, L.L., and Liedeman, R.R. Peroxidation of lipids as a factor modifying membrane-associated proteins of nerve cells in schizophrenia. *Vestnik Akademii Meditsinskih Nauk SSSR*, 1:33–37, 1982*b*.

Pulkkinen, E. Immunoglobulines, psychopathology and prognosis in schizophrenia. *Acta Psychiatrica Scandinavica*, 56:173–182, 1977.

Randrup, A., and Munkvard, I. Evidence indicating an association between schizophrenia and hyperactivity in the brain. *Orthomolecular Psychiatry*, 1:2–7, 1972.

Rao, D.; Morton, N.; Gottesman, I.; and Lew, R. Path analysis of qualitative data on pairs of relatives: Application to schizophrenia. *Human Heredity*, 31:325–333, 1981.

Reveley, M.; Glover, V.; Sandler, M.; and Spokes, E. Brain amine oxidase activity in schizophrenics and controls. *Archives of General Psychiatry*, 38:663–665, 1981.

Reveley, M.; Reveley, A.; Clifford, C.; and Murray, R. Genetics of platelet MAO activity in discordant schizophrenic and normal twins. *British Journal of Psychiatry*, 142:560–565, 1983.

Rimón, R. Viral etiology in schizophrenia? *Annals of Clinical Research*, 15:1–3, 1983.

Rimón, R.; Terenius, L.; and Kampman, R. Cerebrospinal fluid endorphins in schizophrenia. *Acta Psychiatrica Scandinavica*, 61:395–403, 1980.

Rodnight, R. Schizophrenia: Some current neurochemical approaches. *Journal of Neurochemistry*, 41:12–21, 1983.

Roos, B.-E. Schizophrenia and viral and auto-immune issues. *Psycho-*

pharmacology Bulletin, 20:514-518, 1984.

Rosenthal, D.; Wender, P.H.; Kety, S.S.; Schulsinger, F.; Welner, J.; and Ostergaard, L. Schizophrenics' offspring reared in adoptive homes. In: Rosenthal, D., and Kety, S.S., eds. *The Transmission of Schizophrenia*. New York: Pergamon Press, 1968. pp. 377-392.

Sandler, M.; Reveley, M.A.; and Glover, V. Human platelet monoamine oxidase in health and disease: A review. *Journal of Clinical Pathology*, 34:292-302, 1981.

Scarlato, G., and Mastogiovanni, P.D. Ulteriori sull'-azione patogena di liquido allantoideo di embrioni di pollo inoculati con liquor di schizophrenia. *Acta Neurologica*, 11:587-595, 1956.

Semenov, S.F., and Glebov, V.S. Investigations of autoimmune processes related to brain pathology at different stages of schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 69:87-92, 1969.

Shaskan, E.D.; Ballow, M.; Oreland, L.; and Wadell, G. Is there functional significance for dopamine antagonist binding sites on lymphoid cell? *Advances in Biological Psychiatry*, 12:123-141, 1983.

Shields, J., and Gottesman, I.I., eds. *Man, Mind and Heredity. Selected Papers of Eliot Slater on Psychiatry and Genetics*. Baltimore and London: The John Hopkins Press, 1971.

Singer, L.; Wayer, S.; Tongio, M.; Roos, M.; Danion, J.; Bernard, H.; Leclero, P.; Hauptmann, G.; Kafter, M.; Bindler, J.; and Mandel, P. Étude de la frequence des antigenes HLA-A, B, C et B chez des schizophrenes d'origine alsacienne. *Encephale*, 8:9-15, 1982.

Snesarev, P.E. On toxicity in schizophrenia. *Sovetskaya Psikhiatriya*, 5:17-24, 1934.

Stahl, S.; Woo, D.; Wefford, I.; Berger, P.; and Ciaranello, R. Hyperserotonemia and platelet serotonin uptake and release in schizophrenia and affective disorders. *American Journal of Psychiatry*, 140:26-30, 1983.

Stromgren, E. *Statistical and Genetical Population Studies Within Psychiatry: Methods and Principal Results*. Paris: Congr. Intern. Psychiatr. VI, Psychiatr. Soc., 1950. pp. 155-188.

Terenius, L.; Wahlstrom, A.; Lindstrom, L.; and Widerlov, E. Increased CSF levels of endorphins in chronic psychosis. *Neuroscience Letters*, 3:157-162, 1976.

Tissari, A.N. Increase of dopamine synthesis in synaptosomes from rats treated with neuroleptics or reserpine. *Molecular Biology*, 60:38-41, 1982.

Trubnikov, V.; Gindilis, V.; and Shahmatova-Pavlova, I. About anticipation in endogenic functional psychoses. In: *14th International Congress of Genetics. Congress Report*. Moscow: Medizina, 1978. p. 373.

Tyrrell, D.A.J.; Parry, R.P.; Crow, T.J.; Johnstone, E.C.; and Ferrier, I.N. Possible virus in schizophrenia and some neurological disorders. *Lancet*, I:839-841, 1979.

Ungvari, G. Klinikai-genetikai vizsgalatok schizosisokban. *Ideggyogyaszalt Szemble*, 35:481-489, 1982.

Ungvari, G. Validity of the ICD-9 schizophrenia classification. *Acta Psychiatrica Scandinavica*, 68:287-296, 1983.

Vanherweghem, J.; Linkowski, P.; and Mendlewicz, J. Hemodialysis in schizophrenics. *Archives of General Psychiatry*, 40:211-214, 1983.

van Kammen, D.P., and DeLisi, L.E. The viral hypothesis of schizophrenia: Smoke—but is there fire? Summary. *Psychopharmacology Bulletin*, 20:523-525, 1984.

van Praag, H.M. The significance of dopamine for the mode of neuroleptics and the pathogenesis of schizophrenia. *British Journal of Psychiatry*, 130:463-474, 1977.

van Praag, H.M.; Verhoeven, W.; van Ree, S; and de Wied, D. The treatment of schizophrenic psychoses with γ-type endorphins. *Biological Psychiatry*, 17:83-98, 1982.

Vartanian, M.E. Immunological researches of schizophrenia. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 63:457-465, 1963.

Vartanian, M.E. Some biological derangements in schizophrenia and their understanding at the current stage of investigations. *Vestnik Akademii Meditsinskih Nauk SSSR*, 3:11-21, 1966.

Vartanian, M.E. Genetics of psychiatric diseases. In: Snezhnevsky, A., ed. *Handbook of Psychiatry*. Moscow: Medizina, 1983. pp. 115-132.

Vartanian, M.E.; Kolyaskina, G.I.; Lozovsky, D.V.; Burbaeva, G.Sh.; and Ignatov, S.A. Aspects of humoral and cellular immunity in schizophrenia. *Birth Defects. Original Article Series*, 14:339-364, 1978.

Vasiliev, V.N. Effect of small doses of L-dopa on excretion with urine of catecholamines and L-dopa in patients with schizophrenia. *Voprosi Meditsinskoj Khimii*, 26:782-786, 1980.

Wetterberg, L. The genetic control of catecholamine and its possible implication in schizophrenia.

In: Hemmings, G., ed. *Biological Aspects of Schizophrenia and Addiction*, 277:11-19, 1982.

Whitaker, P.; Crao, T.; and Ferrier, I. Tritiated LSD binding in frontal cortex in schizophrenia. *Archives of General Psychiatry*, 38:278-280, 1981.

Winblad, B.; Bucht, G.; Gottfries, C.; and Roos, B. Monoamines and monoamine metabolites in brains from demented schizophrenics. *Acta Psychiatrica Scandinavica*, 60:17-28, 1979.

Winter, H.; Herschel, M.; Propping, P.; Friedl, W.; and Vogel, F.A. twin study of three enzymes (DBH, COMT, MAO) of catecholamine metabolism. *Psychopharmacology*, 57:63-69, 1978.

Wode-Helgodt, B.; Fyrö, B.; Gillberg, B.; and Sedvall, G. Effect of chlorpromazine treatment on monoamine metabolite levels in cerebrospinal fluid on psychotic patients. *Acta Psychiatrica Scandinavica*, 56:129-142, 1977.

Wyatt, R.J.; Potkin, S.; and Murphy, D.L. Platelet monoamine oxidase activity in schizophrenia: A review of the data. *American Journal of Psychiatry*, 136:377-385, 1979.

Zarrabi, M.H.; Zucker, S.; and Miller, F. Immunologic and coagulation disorders in chlorpromazine-treated patients. *Annals of Internal Medicine*, 91:194-199, 1979.

Zayko, S.; Sokolova, N.; Klyushnik, T.; and Burbaeva, G. Isolation and properties of neurospecific proteins 14-3-3, 14-3-3, 10-40-4. *Biokimiia*, 49:355-360, 1984.

Zozulja, A.A.; Patsakova, E.; and Kost, N.V. Study of the interplay between endogenous opiates with human peripheral blood lymphocytes. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 82:700-703, 1982.

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Schizophrenia in Children: A Review of European Research

by John Tsiantis, Iphigenia Macri, and Olga Maratos

Abstract

European literature on childhood schizophrenia is divergent and vast. The topic itself is controversial and complex. The article reviews selected European literature on childhood schizophrenia and presents the most important trends of research from the literature. Extensive work has been devoted in tracing and defining the boundaries of childhood schizophrenia from infantile psychosis, autism, organic psychoses, and borderline states, and of mapping out the condition. The factor of development as an important variable when studying diagnostic criteria and the course of the condition was singled out by many reseachers. The evidence from the research on etiological factors was also reviewed, and most authors seem to agree on a nature-nurture interaction model. The different treatment modalities and approaches are also presented and discussed. Finally, a proposal is made for comparative studies to be carried out that will cover course and followup.

The term "schizophrenia" as originally used by Bleuler is not a clearly defined concept. Classification and diagnosis of adult schizophrenia has always been a controversial issue, and different countries have established varying principles for its recognition at different times. In childhood schizophrenia, the situation has been even more complex. The European literature on the subject is divergent and vast, and the task of integrating it has been both exciting and rather arduous. Collecting the bibliography was particularly difficult, and in some cases it was either impossible to obtain the literature or to have it translated from so many different languages. The present article is therefore a review of *selected* literature and an attempt to present and integrate the major issues of European research on the subject of childhood schizophrenia. The aim has been to present, from the numerous articles available, the most representative rather than to attempt a critical review of the studies. References to the non-European literature are made when indicated. Research on the biochemical and neurophysiological factors in the etiology of childhood schizophrenia has not been included because it represents an extensive area in itself.

Typology and Classification

Historically, research in this area has consisted of an attempt (1) to describe a variety of clinical syndromes of childhood psychoses and (2) to distinguish autism from them. This, in turn, has been followed by attempts to provide a system of classification for childhood psychosis and to work more specifically to develop diagnostic criteria for childhood schizophrenia.

It was de Sanctis (1908) who first described a group of children with abnormalities such as developmental regression, grimacing, and negativism, and applied the term "dementia praecocissima" to them. He distinguished these children from the mentally subnormal and attempted to relate their conditions: to the entity of dementia praecox in adults as described by Kraepelin. This was followed by a number of publications both in Europe and the United States in which various

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clinical syndromes were described and assigned different names coming under the category of childhood schizophrenia. Some examples are early infantile autism (Kanner 1943), symbiotic psychosis (Mahler 1952), and catatonic psychosis of idiocy (Earl 1936). So it appears that childhood schizophrenia has been used as an umbrella term to include a variety of disorders which resemble each other only in their severity, chronicity, and occurrence in childhood.

From the beginning, a great deal of the research centered around the development of objective criteria that would identify different nosological entities within the broad group of disorders classified under the term "childhood schizophrenia." It was Kanner (1943) who, in his now classic article, first described the condition of early infantile autism. During the same period, Asperger (1944) described the condition of autistic psychopathy, while Russian researchers (Gilyarovski 1929; Sukhareva 1937; Simson 1948) were working on a variety of syndromes which they classified under childhood schizophrenia.

In Europe the first real attempts to differentiate among the disorders included under the term "childhood schizophrenia" were made by Anthony (1958*a*, 1958*b*). He was working in the broad field of infantile psychosis, which was considered the earliest form of schizophrenia. Anthony tried to include various syndromes and proposed a classificatory system consisting of the following three groups of childhood psychoses: *Group 1*—Early onset and slow chronic course. The syndromes included here are Kanner's (1943) early infantile autism, Bender's (1947) pseudodefective type, and Despert's (1938) no onset type. *Group 2*—Onset at 3 to 5 years and acute course followed by massive regression. Here, Heller's (1930) disease, de Sanctis' (1908) dementia precocissima, Bender's (1947) pseudoneurotic, Despert's (1938) acute onset type, and Mahler's (1952) symbiotic psychosis are included. *Group 3*—Onset in the middle and late years of childhood and fluctuating, subacute course. This group included Bender's (1947) pseudopsycnopathic type.

Meanwhile, in England, a "working party" was formed to study the schizophrenic syndrome of childhood (Creak 1961). They proposed the following nine characteristics as being specific to this syndrome: (1) A gross and sustained inability to relate normally to people under all circumstances. (2) An apparent unawareness of personal identity to a degree inappropriate to age. (3) A pathological preoccupation with particular objects. (4) A sustained resistance to change in the environment and a striving to maintain or restore sameness. (5) Abnormal responses to sensory experience in the absence of discernible organic abnormality. (6) A tendency to disintegrate into utter confusion everytime the precariously maintained pattern of sameness is interrupted. (7) A relatively high frequency of speech disorders ranging from mutism to echolalic and repetitive speech. (8) Motor disturbances which are not accompanied by clear-cut demonstrable signs, the emphasis being on erratic functioning rather than on any neurological disorder. (9) A general background of low functioning in which islets of normal, near normal, or exceptional intellectual functioning or skill appear.

This list of characteristics, even though comprehensive, does not establish the relative importance of each in making a diagnosis. In addition, these criteria include most types of disturbed behavior observed in children, i.e., emotional, psychomotor, communicative, and developmental, and are therefore not necessarily specific to childhood schizophrenia.

Further research in England (Wolf and Chess 1964, 1965; Rutter 1966; Wing 1966; Rutter, Greenfeeld, and Lockyer 1967) addressed the subject of childhood psychosis and dealt with three basic issues that seemed particularly important. One was the question of intelligence and whether children with mental deficiency, with or without evidence of cerebral disorder, should be classified under childhood psychosis. The second question was whether infantile autism should be considered a subtype of childhood schizophrenia, and the third was whether childhood schizophrenia should be considered an early form of adult schizophrenia.

A considerable number of publications dealt with the second issue, that of autism. The initial opinion that autism is essentially the same condition as adult schizophrenia was based mainly on observations of similarities in the symptomatology of the two conditions (O'Gorman 1958) and on the observation that one of the key features in both is a difficulty with interpersonal relationships. In the United States, however, Eisenberg, as early as 1957, had emphasized the lack of uniformity in criteria for diagnosis and the need for specification of the criteria in future clinical reports. Later, Rutter (1968) cited clinical and research data to argue, rather convincingly, that autism is different from schizophrenia. (A detailed description of these differences is presented below in the section on "Differential Diagnosis.") Many authors, in their attempts to provide a comprehensive classificatory system, emphasized the importance of age of onset in their typologies (Bender 1947; Mahler 1952; Anthony 1958*a*, 1958*b*). It was Kolvin et al. (1971*a*), however, who undertook a systematic study comparing infantile psychosis (IP) and late onset psychosis (LOP) in an attempt to establish differential criteria for the two conditions. For the IP group, age of onset was below 5; for the LOP group, it was 13 years and over, although there were some children with age of onset between 7 and 13 years of age. The findings of this study confirmed earlier reports that early and late childhood psychosis are fundamentally different in age of onset, symptom patterns, family and social background, family history of evidence of schizophrenia, evidence of cerebral dysfunction, and level of intelligence (Kolvin 1971).

Both Rutter (1968, 1972) and Kolvin (1971) argue that children with an early onset of psychotic symptoms belong to a diagnostic category distinct from schizophrenia as seen in later childhood. They also propose that there is discontinuity between early childhood psychosis and childhood schizophrenia, and suggest that schizophrenia may begin with psychotic symptoms in later childhood which, however, are rarely overt before the age of 7 or 8 years. They claim that childhood schizophrenia is not a continuous process that can start in early infancy and continue into adulthood.

French-speaking writers have addressed the issue of childhood schizophrenia under the terms "psychose" and "schizophrenie infantile." Their classification also uses age of onset as a criterion, as well as the fact that the schizophrenic condition is also marked by a *regression* to a previous stage of affective development (Ajuriaguerra 1970; Soulé, Houzel, and Bollaert 1976; Palacio-Espasa 1980). The Kleinian claim that childhood schizophrenia is a form of regressive secondary autism (Tustin 1972) is well accepted by most psychoanalytically oriented French psychiatrists. They state that the confusion in terminology has arisen from the fact that in the classification of childhood psychoses the etiology of the disease is confounded with the symptomatology.

Generally, most French-speaking writers (Michaux et al. 1971; Dopchie 1973; Goudemand et al. 1981) agree on the term "early psychosis" when onset occurs under 5 or 6 years of age and on the term "late psychosis" when the age of onset is between 6 and 10 years. Childhood schizophrenia is classified under the latter. Dopchie (1973) places childhood schizophrenia under the category of "psychoses de structuration."

It is worth mentioning, however, that Soulé, Houzel, and Bollaert (1976), although suggesting five distinct syndromes of childhood psychosis, do not include childhood schizophrenia under any of them.

The work described so far is relevant to the problems of classification of childhood schizophrenia. Rutter (1972) discussed the drawbacks of an etiological classification, a scheme proposed by Eisenberg (1966), and suggested the use of a multi-axial approach to classification that could combine the merits of functional and descriptive diagnosis. This multi-axial system, which was developed at a World Health Organization seminar (Rutter et al. 1969), separated the different dimensions of diagnosis into individual axes and provided specific rules to ensure their use in a systematic and standard manner. The most recent revision of the multiaxial system of classification (Rutter, Shaffer, and Sturge 1975) comprises the following five axes: (1) clinical psychiatric syndrome; (2) specific delays in development; (3) intellectual level; (4) medical conditions including any associated biological or etiological factors; and (5) abnormal psychosocial situations. In this system, childhood schizophrenia comes under the category "psychoses with origin specific to childhood" (299), and it is stated that this category should be used only for psychosis that begins before puberty. This general category includes infantile autism (299.0), disintegrative psychosis (299.1), other psychosis (299.8), and unspecified psychosis (299.9). In this last subcategory, both schizophrenia childhood type (not otherwise specified—NOS) and schizophrenic syndrome of childhood are included. Excluded from this last category is schizophrenia of adult type occurring in childhood (295.0-295.8). This classification system has several advantages because it allows the description of a condition on different axes and allows separate consideration of important factors such as symptomatology, developmental delays, and intelligence level. This approach is particularly useful not only for diagnostic purposes but also for cross-cultural research so that comparisons can be made among groups of children who exhibit differing combinations of those factors.

Diagnostic Criteria and Symptomatology of Childhood Schizophrenia

As early as 1942, Schneider, in his classic work on adult schizophrenia, proposed the following as first-rank symptoms of schizophrenia: hearing thoughts spoken aloud, hearing voices talking to each other that comment on the behavior of the patient, feelings of influence on bodily functions, thought interference, thought stealing, communication of own thoughts to another, and feelings of being influenced from the outside world with regard to emotions, drives, and volition. These symptoms have been proposed to be pathognomonic for adult schizophrenia (Potter 1933; Fish 1962). The International Pilot Study of Schizophrenia (World Health Organization 1973) broadly confirmed their usefulness in differentiating schizophrenia from other disorders. Kolvin (1972) proposed criteria—based on Schneider's first-rank symptoms—that could describe, in terms of symptom frequency, psychotic behavior in children similar to that in adult schizophrenia. Three major groups of symptoms that could be used as diagnostic criteria were identified. The symptoms most frequently described were hallucinations, mannerisms, and an inclination of the ill child to give partial answers to questions. The next group of symptoms comprised disorders of thought (content, association, and thought blocking), and the third group consisted of disorders of affect (incongruity and blunting), drive, and volition.

More specifically, in the major study mentioned earlier (Kolvin et al. 1971*a*), the disorder of thought association and thought blocking was present in 60 percent of the LOP group. In addition, 80 percent of the same group displayed auditory hallucinations, while 48 percent had visual or bodily hallucinations in addition to auditory ones. It is important to note that no child had visual or bodily hallucinations without auditory ones. Blunting of affect was present in 63 percent of the cases, while mannerisms and jerky movements were present in 80 percent and 60 percent of the sample, respectively. In some cases, significant differences in the frequency of the symptoms between the LOP and IP groups emphasized the discontinuity between these conditions. (For a discussion of these differences, see the section on "Differential Diagnosis.")

Most French-speaking writers do not discuss childhood schizophrenia separately from other late onset psychoses of childhood. Although Henny (1970) believes that adult schizophrenia has its genetic roots in childhood, he uses Creak's (1961) nine criteria for the diagnosis of childhood schizophrenia.

In the French literature the criterion that is most discussed in accepting the diagnosis of childhood psychosis is the barrier and difficulty in the child's relation to reality (Kestenbaum 1978). Dopchie (1973) suggests that the diagnosis should be based on (1) the symptomatology, (2) the structure of personality, and (3) the development of the disorder, which encompasses both age of onset and course of illness. Soulé, Houzel, and Bollaert (1976) insist that the diagnosis should be based on the clinical description of symptomatology, while Palacio-Espasa (1980) emphasizes the role of disturbances of the symbolic function.

There is, however, an altogether different trend in European research on childhood schizophrenia in which schizophrenia is considered a *single nosological entity*. One of the main European representatives of this line of thought is Vrono (1974), who has done extensive work on the subject in the U.S.S.R. and is also representative of other writers from Eastern countries. He states that "from the first investigations in this area, Russian psychiatrists . . . have viewed childhood schizophrenia as a *single nosological entity*, defined by the same criteria as other mental illnesses, namely, common etiology, pathogenesis, clinical picture, course and outcome" (Vrono 1974, p. 10; emphasis added).

Vrono's (1974) major study included 200 cases with a followup period of 5–20 years. The age distribution of his sample was as follows: in 21 percent the illness began before the age of 5, in 9 percent of the cases between 5 and 9 years of age, and in 70 percent of the cases between 10 and 16 years of age. He distinguished three types of onset (gradual, subacute, and acute) and emphasized the difficulty in differentiating between premorbid personality features and psychotic features, a difficulty also encountered by Kolvin and his colleagues. In describing the various types of the disease, Vrono considered phenomenology along Kraepelinian lines, age of onset, and course of the development of the disorder as diagnostic criteria.

Vrono examined the clinical features of the illness in the following three age groups: children up to the age of 9, children 10–12 years (prepubertal), and children 13–16 years of age (pubertal). The clinical features described in the first two age groups, which deal with preadolescents, are summarized below:

Age: 0–9 years

1. Episodic progressive course: a combination of continuous and intermittent symptoms with a period of a well-defined "schub." [1]

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[1] The Russian word comes from the German "schub" and denotes those acute events in schizophrenia that gradually abate but leave a lasting change or a schizophrenic defect.

2. Subacute onset: symptoms appear simultaneously or against a background of gradually developing personality symptoms. There is a polymorphism with a characteristic fear syndrome where the child may develop obsessive fears of darkness, solitude, or disease and where catatonic motor and speech disorders are extremely common. Psychotic symptoms such as hallucinations and delusions are rarely encountered in the initial period of childhood schizophrenia.
3. Recurrent course: a remissive or recurrent course which is rarely encountered.
4. Acute onset: the most frequent manifestations are sudden, fleeting states of fear with tension and anxiety, and occasionally attacks of panic, which may be accompanied by terrifying visual hallucinations. In other cases, the acute onset may be manifested by states of motor excitement.

Generally it can be said that various kinds of disorders have been included in this age group, some of which are indistinguishable from early infantile autism, brain syndromes, or severe neurotic problems. Some of the essential features of this group are the absence of hallucinations and delusions, and the presence of negative symptoms. In addition, although Vrono distinguished several subtypes of schizophrenia in this age group and his descriptions are very detailed, it is hard to determine whether the conditions described fall into a homogeneous group of disorders or not.

Age: 10–12 years

1. Continuous sluggish course: characterized by a gradual development of pathological symptoms without marked exacerbations or clear remissions. Early symptoms are marked by a profound change in the child's perception of himself and the world, with signs of a progressive deterioration of personality. The clinical picture is dominated by the emergence of negative symptoms. Positive symptoms appear in indistinct form, and the total picture resembles that of the simple type of schizophrenia.
2. Continuous, progressive course: a predominance of delusional and paranoid thinking where the process progresses moderately throughout the illness.
3. Recurrent course: rare in prepuberty and characterized by an acute onset dominated by disturbances in mood and a variety of positive symptoms. Later in the course of the illness, the attacks become less acute and a "transformation" of the syndrome can take place.
4. Episodic progressive: includes types with polymorphous attacks. The clinical picture is characterized by disturbances of affect such as hypomanic states with delusional fantasies or depressive states with obsessive-compulsive symptomatology.

Vrono's extensive study can be criticized on several grounds. Even though his work is a study on childhood schizophrenia, his sample includes a variety of disorders such as autism, brain syndromes, and severe neurosis. It appears that Vrono's main thesis is that schizophrenia is a single process beginning at an early age in which age differences produce changes in the clinical picture. This position directly opposes that of Rutter and Kolvin, who both maintain that early childhood psychosis is different from schizophrenia. Kolvin (1974), in his review of Vrono's work, notes a lack of clearly defined diagnostic criteria in Vrono's classificatory system. Because Vrono provides a long list of well-described symptoms but no symptom frequency analysis, Kolvin suggests that it is difficult to work out accurately how the various subgroups can be distinguished from each other. Consequently, if one were to compare findings from different cultures and include Vrono's findings, the validity of such a comparison would be questionable. However, Kolvin adds that the descriptive methods used by the Soviet investigators who follow the approach of Kraepelin are complementary to the quantitative methods used by English researchers.

Lebovici (1977) noted the similarities between Vrono's descriptions and his own and those of other authors in France, especially regarding oddities of speech in autistic children and their paradoxical attitudes. He emphasized that even though the severe and bizarre phobic symptoms in childhood described by Vrono may suggest the emergence of a schizophrenic psychosis, it is still difficult to determine pathology solely on the basis of such symptoms. He disagrees with Vrono's claim that the symptoms observed in children are an early or attenuated form of those observed in adult schizophrenics. Lebovici also supports the view of a discontinuity between early childhood psychosis and childhood schizophrenia, especially for the progressive and intermittent forms in which autistic children could be included. However, Lebovici does not seem to consider that early childhood psychosis is altogether different from childhood schizophrenia, even though he mentions in his article that early childhood psychosis does not always develop into schizophrenia. Lebovici also suggests a reclassification that would place all cases of childhood psychosis under a single heading, irrespective of their clinical manifestations, and adds that this classification need not make any presumptions about the child's future. It seems that Lebovici's somewhat contradictory views highlight the uncertainty that still prevails in some areas in the field of childhood psychosis. However, his argument for the definition of objective criteria for diagnostic and research purposes is very important.

In a later French publication, Seabra-Denis (1975), reviewing the Russian approach, emphasized that the form of the development of the disease should be taken into account and distinguished the following three forms of development: (1) progressive-continuous, (2) intermittent, and (3) attack-like.

Along similar lines, and long before Vrono, Ushakov (1965, 1966) had reported on the symptomatology and the clinical peculiarities of childhood schizophrenia. He systematized them by grouping them into the following five categories: (1) compulsive automatisms, (2) coenosthopathic interpretations, (3) affective dissociations, (4) associations of personality reactions, and (5) autism. Ushakov described three forms of the disease: (a) periodic forms with a short initial period of illness, (b) acute and subacute forms lasting for a period of up to 5 years, and (c) forms with a creeping course of development and a long period of onset. Pogády and Hasko (1975) from Czechoslovakia also consider schizophrenia a uniform disease, regardless of age. It is worth noting that in a sample of 114 children and adolescents hospitalized for the first time, 74 percent of the cases were difficult to classify because they presented a less differentiated, atypical psychopathological syndrome. In both studies, the age of onset of the disease was not clearly specified and the diagnostic criteria were not clearly defined. Therefore, Kolvin's criticism of Vrono's work can equally apply to the studies of Ushakov (1965) and Pogády and Hasko (1975).

Similar difficulties in diagnosing schizophrenia have been encountered in other European countries. In Germany, Nowak-Vogl (1979) noted that although there has been an increase in the number of children diagnosed as schizophrenic in German-speaking countries, there is an increased need to establish clearer and more objective criteria for doing so. He also criticized the fact that the main criterion used has been the child's inability to show empathy (Uneinfühlbarkeit). This criterion itself needs to be defined, since the determination of its presence or absence depends on the subjective interpretation of the clinician.

Another major issue in the diagnosis of childhood schizophrenia is that of changes in the clinical picture that occur with advancing age. Kolvin (1972) argues that a child of primary school age exhibits symptoms which are both simple and crude, but that as the child gets older the symptomatology becomes more complex and systematized. Similarly, Van Krevelen (1971*b*) notes that even though in the fully established state the symptoms resemble those of adult schizophrenics, they are somewhat transient, less elaborate, and not systematized. Vrono (1980) also emphasized the need to use a comparative-age approach to the study of schizophrenia.

Other investigators have also studied this issue. Nissen (1971), a German author, in a followup examination of a group of depressive children who grew up under extremely unfavorable conditions, found that 8.6 percent of them were diagnosed as schizophrenic in adult life. Similarly, in a study carried out in Vienna (Friedrich and Leixnering 1980), a comparison of Bleulers', Scheider's, and Berner's criteria for the diagnosis of psychosis was made. Twenty-eight adolescent psychotic patients were observed and supervised for up to 5 years. When Bleuler's criteria were used, a change of diagnosis was noted over time. They found that the 22 cases of schizophrenia and the 6 cases of affective psychosis changed into 16 cases of schizophrenia, 8 cases of affective psychosis, and 4 cases of a borderline condition. When Berner's criteria were used, the 14 cases of endomorphic cyclothymia remained the same, while 13 of the 14 cases of endomorphic schizophrenic psychosis remained the same and only one diagnosis was changed to schizoaffective. Finally, when Schneider's criteria were used, all nine patients diagnosed as schizophrenic remained the same. These findings are somewhat difficult to interpret. They may suggest that Schneider's criteria are more clearly defined so that diagnosis remained the same with advancing age. On the other hand, one might attribute the differences simply to the differences involved in the sizes of the samples (the larger the sample, the greater the probability of a change of diagnosis with time). Problems in diagnosis and the issue of change of diagnosis over time can probably be clarified if adequately defined criteria are used and the necessary reliability studies are carried out.

It appears that the complexity and systematization of symptoms depend on three main factors (age, verbal ability, and intelligence of the child), all of which must be taken into account in further research in the area of diagnosis in childhood schizophrenia.

Incidence and Sex Ratios

It seems that childhood schizophrenia is a rather rare condition: therefore, its incidence is rarely reported in the literature. It took Kolvin (1972) several years to collect 33 cases. Once again, the importance of establishing clear-cut diagnostic criteria cannot be underestimated. It could be argued that as long as all the psychotic disorders of childhood are grouped together, it is difficult to speak of the true incidence of the condition. On the other hand, in the case of conditions that are more clearly defined such as early infantile autism, there have been a number of studies (Lotter 1966; Brask 1970; Wing et al. 1976) reporting the prevalence of the condition.

As far as the sex ratio in childhood schizophrenia is concerned, Kolvin et al. (1971*b*) reported that boys outnumber girls 2.5:1. Similar findings have been reported in studies of adolescents, where boys also seem to outnumber girls (Steinberg 1976). However, Russian researchers such as Sukhareva and Kogan (1933) and Vrono (1974) do not report this difference. This could be due to variations in the definition and diagnostic criteria used.

Differential Diagnosis

A major issue closely related to diagnosis is that of differential diagnosis between schizophrenia and other conditions, and there is a substantial amount of literature related to this problem. The following conditions are dealt with separately in this section: autism, autistic psychopathy, schizophrenia with developmental disorder, progressive disintegrative psychosis, schizoid personality, borderline conditions, and "other."

Seven basic differences between *autism* and *childhood schizophrenia* have been extensively discussed by Rutter (1968) and Kolvin (1971, 1972).

1. A difference in the clinical features, i.e., hallucinations and delusions are common in childhood schizophrenia (Rutter 1968; Kolvin et al. 1971*a*) but are seldom present in autism. On the contrary, gaze avoidance, stereotypies, resistance to change, and insistence on sameness, as well as serious delays in speech and communication, are characteristic of autism but infrequent in childhood schizophrenia.
2. A difference in sex ratio, i.e., in autism boys outnumber girls 4:1 (Rutter 1968), while in childhood schizophrenia boys outnumber girls by a lower 2.5:1 (Kolvin et al. 1971*b*). It should be noted that no such differences are reported in adult schizophrenia, where the sex ratio is approximately equal (Mayer-Gross, Slater, and Roth 1955).
3. A difference in the family's social background. Autism is frequent in the upper socioeconomic class (Lotter 1967), while childhood schizophrenia is frequent in middle or lower socioeconomic classes (Kolvin et al. 1971*b*). However, the findings regarding the social class of the parents of autistic children have recently been challenged by Lorna Wing (1980), who suggests that these results might be due to an artifact of referral policies and diagnosis.
4. A difference in the family history of the children. Schizophrenia is rarely present in the parents or siblings of autistic children (Creak and Ini 1960; Rutter 1967), while its occurrence is significantly higher in the families of schizophrenic children (Kolvin et al. 1971*b*).
5. A difference in the intellectual development of the children. Autistic children have been reported to have a low level of intellectual functioning, with a large percentage having IQs below 70 (Rutter and Lockyer 1967; Kolvin, Garside, and Kidd 1971). Schizophrenic children are reported to have higher IQs than autistic children, even though there is a tendency toward intellectual dullness (Kolvin 1972).
6. Perinatal complications and evidence of cerebral dysfunction such as abnormal electroencephalographic findings. They are present more frequently in autism than in childhood schizophrenia (Kolvin, Dunsted, and Roth 1971).
7. An excess of oddities in the personalities of the parents. They are more common in the parents of schizophrenic children than in the parents of autistic children (Kolvin, Garside, and Kidd 1971).

Some of the differences between autism and schizophrenia could be attributed to the difference in the age of onset in the two conditions, as suggested by Vrono (1980). However, the differences in sex ratio, family history, and social class are somewhat harder to explain.

Two related studies by Cox et al. (1975) and Bartak, Rutter, and Cox (1975) compared a group of autistic boys with a matched sample of boys with severe developmental or receptive language disorders. The findings showed no differences between the two groups with respect to parental warmth, sociability, demonstration of emotions, responsiveness, and presence or absence of parental psychiatric disorder.

Schizophrenia must also be differentiated from the syndrome of *autistic psychopathy*, first described by Asperger (1944, 1968) and later by Van Krevelen and Knipers (1962) and Van Krevelen (1971*a*). Van Krevelen emphasized that this syndrome is a personality trait and not a psychotic process. Autistic psychopathy is a disturbance of contact, and the basic disturbance of these children is their personal unapproachability. Van Krevelen reported the following main features: (1) disorder is manifested in the third year of life or later, (2) child walks late but speaks earlier, (3) child's language aims at communication but remains one-way traffic, (4) child evades eye contact with others, (5) child lives in our world in his own way, and (6) child's social prognosis is good.

Wosiński (1973), in Poland, emphasized the need to differentiate between *schizophrenia* and *schizophrenia with signs of a developmental disorder*. He reviewed the developmental histories of 20 children with a clinical diagnosis of schizophrenia and recognized four types: (1) children with normal development, (2) children with no delay before 3 years of age, (3) children with irregular development before 3 years of age, and (4) children with delayed development from the beginning. He questioned whether a developmental disorder affected the clinical picture in schizophrenia, and suggested the use of developmental criteria in the selection of samples. In the study of childhood schizophrenia, children with similar courses of early development could thus be compared.

Schizophrenia should be differentiated from *progressive disintegrative psychosis*, which includes disorders "in which normal or near normal development for the first few years is followed by loss of social skills and speech together with a severe disorder of emotions, behavior and relationships" (Rutter, Shaffer, and Sturge 1975, p. 16). The condition may follow overt brain disease or encephalitis, but it may also occur in the absence of any known organic brain disease or damage. Corbett and Harris (1977) reviewed the literature on dementias and disintegrative psychosis in childhood, and suggested that there is no reason for retaining the eponymous syndromes of de Sanctis' and Heller's disease. Instead, he suggested the use of the term "progressive disintegrative psychosis" to describe the clinical psychiatric picture in these children.

The problem of differential diagnosis between *schizophrenia of the insidious type* and children with *schizoid* features is addressed by Wolf and Barlow (1979), who described children with schizoid personality. Some of the main characteristics of these children are emotional detachment and solitariness, rigidity or lack of adaptability that at times assumes obsessional proportions, sensitivity with occasional suspiciousness and paranoid ideation, lack of empathy for others that at times amounts to callousness, and odd ideation with metaphorical use of language. These children, predominantly boys, can be differentiated from autistic children because they are of school age and lack the cardinal features of the autistic syndrome such as absent or seriously impaired language development with echolalia, lack of emotional responsiveness with gaze avoidance, and ritualistic and compulsive behavior. They can also be differentiated from schizophrenia because they do not display any of the first-rank symptoms of Schneider. An important question for future research would be to examine whether the described clinical features of these children remain the same with increasing age.

In Sweden, Aarkrog (1973) discussed the problems of differential diagnosis between *infantile borderline cases* and *schizophrenia with a slow and insidious onset*. In Denmark, Brask (1959, 1970) and Aarkrog (1973) have described the borderline condition, which they view as often being a transitional form toward infantile psychosis. In the borderline states, contact is not decidedly autistic, and reality evaluation is not entirely lacking, so that there are more areas of personality intact than there are in infantile psychosis. Brask (1970) indicates that borderline psychoses are characterized by a fluctuating "functional level." In addition, Chilland and Lebovici (1974) describe borderline states in early infancy and during the latency period. In the early period the children are described as agitated, irritable, and hyperkinetic. During later years, the children may be irritable and create a lot of discipline problems at school; or they may be extremely inhibited and reach the level of mutism outside the family. They may also present bizarre phobias (Lebovici and Diatkine 1963). These researchers also suggest, as Aarkrog does, that certain areas of functioning such as intellectual development and satisfactory school performance remain intact in spite of the presence of anxiety and multiple bizarre symptoms that undoubtedly are outside the range of neurotic symptomatology. Kovalev (1980) also discusses difficulties in the differential diagnosis between a slowly progressing schizophrenia and borderline, neurotic, and residual-organic states with pseudoautistic traits. He lists a series of problems of psychopathology and symptomatology, and suggests that these may be related to changes in the clinical picture with advancing age. In addition, French-speaking writers, mostly psychoanalytically oriented (Ajuriaguerra 1970; Wiener 1978; Palacio-Espasa 1980), have been particularly concerned with the early stages of emotional development and its role in differential diagnosis. The syndromes dealt with in their writings are autism, symbiotic psychosis, "psychoses à l'évolution déficitaire," and serious developmental disharmony. Mises and Barande (1963) and Ajuriaguerra (1970) also note the difficulties that arise when there is an organic dysfunction on which psychotic manifestations are organized.

Other relatively rare conditions may present problems in differential diagnosis—for example, acute psychotic-like episodes in childhood which might have been induced by drugs such as amphetamine (Connell 1958) or artane (Black and Woollacott 1974). Differential diagnosis should also be made from celiac disease (Challacambe 1971), which may be accompanied by disorders of affect such as irritability, moroseness, apathy, and depression. These symptoms appear in children aged 1-3 years—the age itself excludes the possibility of schizophrenia--and usually disappear when a glutein-free diet is instituted. The rare acute forms of childhood schizophrenia must also be differentiated from the equally rare manic-depressive psychoses of childhood (Anthony and Scott 1960). Finally, Kolvin (1971) and Rutter (1972) argue that schizophrenia as we know it in adults can begin in childhood. They have gathered a substantial amount of evidence to support the claim that schizophrenia does occur in children and that its onset may be at 7 years of age or earlier. Rutter has even suggested that the term "schizophrenia" is in itself adequate and that there is no need to add the adjective "childhood." This thesis has been disputed mainly by American writers such as Bender (1971) and Miller (1974). However, Rutter (1978) argued that conflicting evidence has derived primarily from studies that either failed to make systematic comparisons by age of onset (Ornitz 1971; Bomberg, Szurek, and Etemad 1973) or used broad definitions of schizophrenia (Bender 1947, 1960). Similar criticisms of Vrono's (1974) work have been made by Kolvin (1974).

Etiological Factors: Pathogenesis

Genetic Studies. Although genetic research and family studies of adult schizophrenics and autistic children abound in the literature, there seem to be very few studies of the families of psychotic children with a late age of onset of psychosis. The idea of a genetic basis for schizophrenia has evoked controversy because of doubts about the validity of twin studies flawed by limitations in sampling and methodology. Although adoption studies conducted in Denmark provide overwhelming evidence of the substantial role of genetic factors in the origin of schizophrenic disorders (Kety et al. 1968; Wender et al. 1974), uncertainty still prevails regarding the mode of transmission and what is actually inherited (Shields, Heston, and Gottesman 1975), i.e., a pathological personality trait or a special vulnerability to the disease. More recently, Kety (1980) has suggested that the findings of the adoption studies give additional credibility to the etiological diversity of schizophrenia. He has proposed a model in which there is a single major locus of intermediate dominance (earlier suggested by Slater 1958) with 15 percent of schizophrenics as phenocopies, 1-2 percent as homozygotes, 84 percent as heterozygotes, and a penetrance of 46 percent and 23 percent in homozygotes and heterozygotes, respectively.

Carlier (1980) has undertaken an extensive description and critique of the studies that have used the method of adoption to isolate the genetic factor involved in schizophrenic psychosis. Her main conclusion is that very few studies have developed an adequate methodology for answering the crucial question. Her most important criticisms of these studies are (1) the lack of control of the age of separation of the child from the ill parent, (2) the method chosen for arriving at the diagnosis, and (3) the failure, in most studies, to report the age at which the diagnosis of mental disturbance in the children was made. She concludes that most studies to date agree that there is a genetic etiology in schizophrenia (adult and childhood), but there is not enough evidence of a direct genetic causal effect. Her conclusions are in agreement with those of other French-speaking writers, who maintain that although there are more disturbed children in families of psychotic parents, there is still no conclusive evidence that childhood schizophrenia is genetically transmitted (Gayral 1973; Bourgeois, Malarrive, and Charruand 1974; Goundemand et al. 1981). In another article, Carlier and Roubertoux (1979) maintain that even if a strong influence from family-environmental factors in childhood psychoses were demonstrated, a genetic analysis would still be useful and complementary. They conclude that psychoses of the schizophrenic type are marked by genetic heterogeneity.

Family Studies. The samples of the studies reviewed in this section are not strictly restricted to children, but the findings are included because they highlight important issues in this area. Many theorists have suggested that schizophrenia arises as a result of disturbed parent-child relationships, and a great deal of research has been done in this field, especially in the United States (Bateson et al. 1956; Lidz 1958; Wynne et al. 1958). In these studies, the deviance of the individual is regarded as a manifestation of disturbed family relationships.

In Finland, Alanen (1958) studied 100 mothers of young schizophrenics and found that these mothers were domineering, possessive, and unable to understand the needs of their children. Furthermore, they behaved differently toward their sons than toward their daughters—being "possessively protective" toward the former and "inimically protective" toward the latter. In a later study, Alanen (1966) examined 30 families of schizophrenics in comparison to a control group of 30 families of neurotics. He distinguished two types of families in the group of schizophrenics: "chaotic" and "rigid." Parents in the rigid families were possessive and restrictive, whereas the boundaries between the different members of the chaotic families were unclear. However, Alanen did not claim that there was a direct cause and effect relationship between parental behavior and schizophrenia in the offspring.

In England, Hirsch and Leff (1975) attempted to replicate the findings of Wynne and Singer (1963), and Wynne (1968), who used a special method of scoring the transcripts of Rorschach tests of parents to study family communication patterns. Wynne and Singer found marked differences between the parents of schizophrenics and the parents of neurotics and normals, but Hirsch and Leff were unable to confirm these findings. Hirsch and Leff reported that the fathers of schizophrenics talked more than the fathers of neurotics. They also observed minor but statistically significant differences in communication deviance, as had been found earlier by Wynne and Singer. However, when Hirsch and Leff took into account the greater talkativeness of the parents of schizophrenics, the difference between the groups disappeared. Hirsch and Leff's results do not confirm Wynne and Singer's "transactional" hypothesis of the etiology of schizophrenia. It seems likely that the differences may be the result of selective referral biases or diagnostic disparity.

Another group of researchers (Blakar, Paulsen, and Sølvberg 1978) studied communication in schizophrenia from a social-cognitive viewpoint. They reported that families containing a schizophrenic member communicated less effectively in conflict situations than their controls.

In a review of the literature on the parental factors involved in schizophrenia, Hirsch (1979) concluded that there is no evidence of a direct causal effect and that the findings are controversial. He considered it important, however, especially for clinical purposes, to take into account the elements of stress-producing behavior in the parents of schizophrenic children.

One of the drawbacks of the studies on family interactions is that observations are made after the first or repeated hospitalizations of the schizophrenic offspring. Thus, it is difficult to determine whether the observed family communication patterns developed in response to the "ill" member or were present before the onset of "illness." This methodological problem might be obviated in future prospective studies.

In a related study in Germany (Schwoon and Angermeyer 1980), the reactions of families whose sons had been hospitalized for the first time with schizophrenia were compared with those whose sons had been hospitalized for surgical treatment. A personality questionnaire was used to assess how three family members (father, mother, and son) viewed themselves and each other. In the families with a schizophrenic son, the father-son relationship differed from that in the control families. This difference was attributed to the reaction of the father to the "deviant" behavior of the son, who did not conform to the father's expectations regarding male sex roles. The responses of the mother partially supported this conclusion.

A series of studies in England (Brown and Birley 1968) explored the role of psychosocial factors in the precipitation of schizophrenia and in the course of its development. Birley and Brown (1970) demonstrated that during the 3 weeks immediately preceding the first onset or an acute relapse with florid schizophrenic symptoms, there is often a marked increase in the frequency of certain social events in comparison to control groups. Some of the events would be expected to have been experienced as pleasant and others as unpleasant. The time relationships suggest that environmental stress could *precipitate* psychosis but would *not cause* psychosis in an otherwise "healthy" individual. This research deals with recent life events that may precipitate schizophrenia in predisposed individuals and does not address the effects parents may have on the children during development.

In Great Britain, the effects of "trauma" in early childhood have been investigated by Stroh (1966, 1974), who studied a group of children with "traumatic psychosis."

These children had been severely traumatized, both physically and mentally, in early childhood. He included them in the category of psychosis because of the intensity and breadth of their disturbance, their faulty adjustment to reality, their grossly distorted or diminished relationships to people, and their sometimes bizarre symptomatology. Even though cruelty and aggression were common in these children, they were able to form a strong bond with an adult caretaker. However, this bond could be easily disrupted. According to Stroh, this was caused by, or at any rate associated with, early episodes of violence which the child had experienced himself, witnessed in his intimate environment, or both. Similar observations have been made by Dockar-Drysdale (1968) in her description of the "frozen" child, and by Rosenfeld (1972). It is possible that these children may fit into the category of borderline psychosis.

The psychoanalytic viewpoint regarding etiological factors in schizophrenia is well represented by the works of Klein (1946), Fairbairn (1952), Winnicott (1952, 1965), and Laing (1967). They view personality and its deviation and disturbances as an outcome of the ways in which we deal with our primitive anxieties during early development. Consequently, the immediate environment—parents, family—plays a considerable role. However, descriptions of systematic investigations testing their hypothesis have not been published.

Most French-speaking writers also discuss the pathogenesis of childhood psychoses in the framework of psychodynamics. Diatkine (1969) stresses the premorbid manifestations of schizophrenia and mentions "affective debility" as an important component of childhood psychoses. Kestenbaum (1978) believes that there is no *single* factor that is more important than others in childhood schizophrenia, and she stresses the importance of psychological factors, along with family, constitutional, neurological, and hereditary factors. Soulé, Houzel, and Bollaert (1976) agree with the pathogenic approach of Klein, Mahler, and Winnicott. Diatkine (1969) gives a good definition of a concept of schizophrenic premorbidity with a psychodynamic structure.

The above descriptions by psychoanalytically oriented authors reflect work stemming primarily from clinical case studies and not from systematic research.

Antecedents or Precursors of Childhood Schizophrenia

There have been many attempts in recent years to identify the precursors (biological and psychosocial) of schizophrenia. It has been suggested that schizophrenia, which may appear in florid forms in late childhood or early adolescence, is often preceded by personality eccentricities and/or oddities. Similarly, there is evidence that individuals who develop schizophrenia as adults have frequently shown abnormalities of a nonpsychotic nature in childhood (Rutter 1972). It is therefore important to try to identify symptoms, personality traits, or other factors such as perinatal complications which may be correlated with schizophrenia.

Bourgeois, Mallarrive, and Charruaud (1974) examined children who were reared by psychotic parents and identified three types of problems: (1) precursors of later adult psychoses (including schizophrenia), (2) parapsychotic or "induced" problems (e.g., hallucinations, delirious states), and (3) neurotic, antisocial, and behavioral disorders. They also found that 5–10 percent of the children were "supernormal," creative, and "colorful." These investigators concluded that it might be better for children to grow up with parents who have been identified as "ill" and are receiving treatment than with parents who are neurotic, borderline, or rejecting.

In studies in Denmark, Mednick et al. (1974) and Schulsinger and Mednick (1975) found that children at high risk for the development of schizophrenia showed a distinctive psychophysiological response to stress: an unusually marked autonomic reaction, which failed to habituate. The risk of psychiatric disorder was enhanced when, in addition to these psychophysiological characteristics, children were of low birth weight and had an excess of early perinatal complications and early stressful separation experiences. Ajuriaguerra (1970) reported studies in which a rather high percentage of perinatal complications, especially neonatal anoxia, and traumatic experiences was found among schizophrenic children.

In a later study, Schulsinger (1976) compared a high-risk group of 207 subjects with severely schizophrenic mothers to a matched low-risk group of 104 subjects with no known mental illness in the family. She followed the two groups for over 10 years and reported marked differences between them with regard to type and degree of psychopathology. Almost 9 percent of the 173 fully reassessed subjects (10 years later) were classified as schizophrenics. In the high-risk group, 15 cases were diagnosed as schizophrenic, while in the low-risk group a particularly large number were diagnosed as neurotic and only one case was diagnosed as schizophrenic.

Finally, Parmas et al. (1982) recently reported the results of a prospective study of the offspring of schizophrenic mothers. They examined the frequency of perinatal complications, as reported in the midwife protocols, of those children who as adults were diagnosed as schizophrenic, borderline schizophrenic, or without evidence of mental illness. The schizophrenics had had the most complicated births, while the borderlines had had the least complicated births. This difference was explained in terms of a "diathesis-stress" model which suggests that schizophrenia is the result of an interaction between adverse environmental conditions and a genetic predisposition (Shields 1978). The latter is in accord with the views of Leonhard (1980, p. 437), who states, "Today, it is generally supposed that both heritability and environment contribute to the origin of schizophrenia, but the kinds of influence of neither are known. . . ."

The findings of the studies cited suggest that it would be important to do preventive work with regard to perinatal complications and psychosocial adverse factors. There is a need for further research to identify the high-risk groups, as well as for research on family interaction and dynamics. Such studies could eventually yield findings which would make it possible to give accurate and helpful advice to parents and others involved in the management and treatment of these children.

Onset, Course, and Prognosis

The onset and course of the development of schizophrenia have been studied extensively by Russian and German researchers. Vrono (1974) describes gradual, acute, and subacute forms of onset. Similarly, Kolvin (1972) describes types with an insidious onset and a rarer acute onset with florid symptoms.

Ushakov (1965), on the basis of an intensive study of 200 schizophrenics aged 13 to 16, was able to establish that for the periodic forms of schizophrenia, the initial period is comparatively short, lasting only days or weeks. For the acute, subacute, paranoid, and hebephrenic forms, it is substantially longer, and it is especially long for the forms characterized by a creeping course. Ushakov suggests that the symptoms are characterized by complications in development which increase with time. Kolvin (1972) also mentions that the content of the symptoms becomes richer with increasing age, and both Vrono (1974) and Kolvin (1974) recognize the difficulty in disentangling premorbid behavioral anomalies from features of a psychotic onset.

Vrono and other Russian writers use four types of course of development of the disorder that are in accord with the classificatory system adopted by the U.S.S.R. Institute of Psychiatry—namely, the continuous sluggish, continuous progressive, episodic progressive, and intermittent recurrent courses of development. Vrono (1980) notes that the continuous sluggish course of development has a favorable prognosis, whereas an unfavorable prognosis is associated with the continuous progressive course, especially when there are exacerbations and protracted episodes without well-defined remissions.

Vrono (1974) distinguishes three broad groups that are related to prognosis by using the rate of personality deterioration as a criterion. First, there is a course of development with gradual and mild personality changes, in which the prognosis is good for the duration of the illness. There is another course of development that is initially favorable but later becomes increasingly unfavorable. Finally, there is a course of development that is unfavorable from the beginning, is characterized by the rapid development of gross personality defect, and has a poor prognosis.

The question of prognosis has also been addressed by other European writers. Eggers (1973), in Germany, undertook a 20-year followup study of 57 schizophrenic children with an age of onset before 14. Eleven children had recovered completely, and about half had improved. The worst prognosis was for children with an onset before 10 years of age and for children with previously abnormal personality characteristics. Above average intelligence and a secure, friendly, and extroverted personality were related to good prognosis. Family history, a disturbed family atmosphere, and type of symptoms were all unrelated to outcome. Albert (1980) reported the followup of six cases of infantile schizophrenia over a 27-year period. One case diagnosed as periodic catatonia was distinguished from the other cases by a normal development until puberty. The other five cases seemed to suffer from "systematic" catatonia, with a slowly progressive course of development characterized by symptoms resembling autism and psychomotor syndromes.

In another study by Lempp and Vogel (1966), 23 children were treated after being diagnosed as having infantile psychosis. The followup period lasted approximately 6¾ years. Eleven of these children developed satisfactorily, while 12 required further institutional treatment. Early psychosis with a slow, gradual onset before school age foreshadowed poor prognosis. On the contrary, prognosis for children with an onset after age 10 was favorable. In the last two studies, it was suggested that the schizophrenic child is frequently an only child.

In a followup study, the Russian writer Bashiva (1980) examined 222 patients with early childhood schizophrenia, including 102 patients with attack-like, slowly progressive schizophrenia. The forms of schizophrenia with onset in early childhood showed a distinct stability. Bashiva suggested that a variation in progression is inherent in each form of the disorder but the greatest variation is found within the range of the attack-like, slowly progressive type of schizophrenia.

From the work cited above, it can be generally concluded that prognosis is better for the acute and florid forms than for the insidiously developing ones.

Treatment

Schizophrenia in children is a complex disorder caused by an interaction between an adverse psychosocial environment and a genetically determined vulnerability. Treatment studies address both of these factors.

In reference to drug treatment, Kolvin (1972) suggests that phenothiazines often lead to the rapid disappearance of florid symptoms and to an arrest of the schizophrenic process. Bovet du Bois (1973) reports that thioridazine is effective in attenuating some of the symptoms found in psychotic children. Michaux et al. (1971) conclude that fluphenazine enanthate is effective in adolescent schizophrenia and partially effective in infantile psychosis, but has no effect on psychotic children in the latency period. It has been suggested (Jørgensen 1979) that the pharmacological treatment of schizophrenia in children should follow the guidelines for treatment of adult patients, but that special care must be taken during the treatment of children in pubertal development because of the well-known influence of phenothiazine derivatives on the excretion of prolactin. On the contrary, O'Gorman (1970) states that tranquilizers are very seldom effective in the treatment of childhood schizophrenia, in contrast to their efficacy in adult schizophrenia.

Kolvin (1972) advocates supportive psychotherapy for schizophrenic children but claims that analytical psychotherapy has made no contribution. However, others such as Winnicott (1967), Lebovici and McDongall (1960), and Kestenbaum (1978) advocate the use of analytical psychotherapy. Soulé, Houzel, and Bollaert (1976), reporting on psychotherapy practiced at the Institut de Puériculture de Paris, state that for early onset psychoses, therapy should be offered within the framework of a day hospital. They believe that the therapist should create an individual relationship with as much continuity as possible, keeping absences and interruptions to a minimum. These authors also recommend that the psychotherapist take physical care of the child (e.g., feeding, bathing). The difficulties that psychotherapists encounter in treating psychosis are compared by Soulé and his colleagues to Winnicott's notion of the mother's "hate" toward her newborn (Winnicott 1947).

Kestenbaum (1978) describes a comprehensive therapeutic intervention as having the following three steps: (1) the child should identify the therapist as a person; (2) work should be done toward amelioration of the use of speech; and (3) after contact has been established, the therapist should go through the early stages of emotional development with the child. In cases of mutism and severe autism, paraverbal techniques should be worked out in therapy. Kestenbaum states that psychotherapy is a mixture of art and science, and she maintains that the therapist should be a real object to the child, not just an object of transference.

Ajuriaguerra (1970) maintains that the results of psychotherapy depend on (1) the quality of the therapist's emotional investment, (2) the absence of changes of therapists, (3) the synchronization of all the components of the treatment, and (4) the support of the parents.

Stroh (1974) has described the essential features in the inpatient assessment and treatment of psychotic children, including schizophrenic children. A specific feature of the functioning of his inpatient unit is the attempt to integrate the social, therapeutic, and educational aspects of care with the day-to-day care of the child. In this therapeutic setting, individual psychotherapy of the child, as well as counseling, support, and, if necessary, treatment of the parents, may be included (Tischler 1971).

However, it should be stressed that the various treatment plans for schizophrenic children, such as the one described above, have not been adequately assessed and there is no acceptable evidence indicating that any one method is better than the others (Rutter 1967). Furthermore, in the description of various treatment modalities, most authors use the term "childhood schizophrenia" in a rather loose manner. This stems from the many difficulties surrounding the use of the concept of childhood schizophrenia. As a result, it is not surprising that one set of researchers, studying children *they* call schizophrenic, may obtain findings different from those of another set of researchers performing similar studies on children *they* designate as schizophrenic. Diagnostic differences make it extremely difficult to draw conclusions about the effects of various methods of treatment. Ideally, clinical impressions should be confirmed, and findings replicated, in adequately controlled evaluations using well-defined and widely accepted diagnostic criteria. However, undertaking such studies may be difficult because of the rarity of childhood schizophrenia.

Summary

The task of reviewing the European literature on childhood schizophrenia, difficult enough in itself, is further complicated by the fact that researchers have yet to agree on a common definition of the disorder. This fact is reflected in the comparative length of the various sections of this article—many pages being taken up by research on the issues of diagnosis, classification, and differential diagnosis. Various authors, under the all-encompassing term "childhood psychosis," have attempted to trace the boundaries of the specific conditions found within it. Boundaries in this "map," however, are often unclear and frequently overlapping. In addition, when one discusses "European" literature on the subject, one must constantly keep in mind the diversity in approach, and the emphasis in some cases on theoretical articles rather than on empirical data.

Extensive research has been devoted to the study of early infantile psychosis (autism, autistic psychopathy, infantile psychosis, and progressive disintegrative psychosis) and late onset psychosis in an attempt to describe the conditions and establish differential diagnostic criteria. The aim was to obtain evidence for the hypothesis that psychosis is a unitary entity.

Another important body of research, in defining the boundaries of childhood psychosis, has been the work on borderline states and on personality disorders, i.e., schizoid children.

Another focal point has been the study of the age of onset, which was used as a criterion for the classification of autism, childhood schizophrenia, and "organic" psychosis.

A major issue that has been extensively discussed was whether childhood schizophrenia is a single disease process which starts at an early age and continues into adulthood, or whether there is, on the contrary, discontinuity between early childhood psychosis and childhood schizophrenia. A basic difference is noted here between the Soviet approach, in which it is argued that there is a continuity of the disorder, and the British viewpoint which argues that children with an early onset of psychotic symptoms represent a diagnostic category distinct from schizophrenia as seen in later childhood.

The developmental factor (i.e., the changes in the clinical picture with age) has been singled out by many writers. They extensively discuss the relative crudeness and simplicity of symptoms at an early age versus the complexity and systematization of symptoms as age progresses.

In the review of the literature, it was noted that using prognosis or the description of the course of the development of the disease for diagnostic purposes has led to considerable confusion, since both prognosis and course can only be assessed after the illness has already progressed.

Finally, the search for etiological factors (i.e., heredity or environment) has so far had inconclusive results. However, most authors agree on a nature-nurture interactional model.

In terms of future research, it seems that comparative studies that cover course and followup are needed. In such research, it is important to use (1) a high degree of selectiveness of subjects, (2) adequately defined diagnostic criteria, (3) reliability studies, and (4) a developmental approach.

It may be useful to consider a European or international pilot study on childhood schizophrenia that would take into account the abovementioned key issues and possibly use a multi-axial classificatory system.

References

Aarkrog, T. Conditions in adolescents who were borderline psychotics as children. *Acta Psychiatrica Scandinavica,* 49:377–385, 1973.

Ajuriaguerra, de J. *Manuel de Psychiatrie de l'Enfant.* Paris: Masson, 1970.

Alanen, Y.O. The mothers of schizophrenic patients. *Acta Psychiatrica et Neurologica Scandinavica,* Supplement 124, 1958.

Alanen, Y.O. The family in the pathogeneses of schizophrenic and neurotic disorders. *Acta Psychiatrica Scandinavica,* Supplement 189, 1966.

Albert, E. Follow-up of early childhood schizophrenias: Progress of 6 cases over 25 years. Psychiatrie, *Neurologie, und Medizinische Psychologie,* 32(1):54–63, 1980.

Anthony, J. An etiological approach to the diagnosis of psychosis in childhood. *Revue de Psychiatrie Infantile,* 25:89–96, 1958*a*.

Anthony, J. An experimental approach to the psychopathology of childhood: Autism. *British Journal of Medical Psychology*, 21:211-225, 1958*b*.

Anthony, J., and Scott, P. Manic depressive psychosis in childhood. *Journal of Child Psychology and Psychiatry*, 1:53-72, 1960.

Asperger, H. Die: "Autistichen Psychopathen." *Kindesalter Archiv fur Psychiatrie und Nervenkrankheiten*. 117:76-136, 1944.

Asperger, H. On the differential diagnosis of early infantile autism. *Acta Paedopsychiatrica*, 35(4):136-145, 1968.

Bartak, L.; Rutter, M.; and Cox, A. A comparative study of infantile autism and specific receptive language disorder: I. The children. *British Journal of Psychiatry*, 126:127-145, 1975.

Bashiva, V. Course and prognosis of childhood schizophrenia in the light of follow-up information. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 80(10):1507-1510, 1980.

Bateson, G.; Jackson, D.D.; Haley, J.; and Weakland, J.H. Towards a theory of schizophrenia. *Behavioral Science*, 1:251-264, 1956.

Bender, L. Childhood schizophrenia: Clinical study of 100 schizophrenic children. *American Journal of Orthopsychiatry*, 17:40-56, 1947.

Bender, L. Treatment in early schizophrenia. *Progressive Psychotherapy*, 5:177-184, 1960.

Bender, L. Alpha and omega of childhood schizophrenia. *Journal of Autism and Childhood Schizophrenia*, 1(2):115-118, 1971.

Birley, J.L.T., and Brown, G. Crises and life changes preceding the onset or relapse of acute schizophrenia: Clinical aspects. *British Journal of Psychiatry*, 116:327-333, 1970.

Black, D., and Woollacott, S. Acute toxic psychosis in two children treated with benzhexol hydrochloride (Artane). *British Journal of Psychiatry*, 125:283-284, 1974.

Blakar, R.M.; Paulsen, O.G.; and Sølvberg, H.A. Schizophrenia and communication: A replication taking ecological variation into consideration. *Acta Psychiatrica Scandinavica*, 58:315-326, 1978.

Bomberg, D.; Szurek, S.A.; and Etemad, J.C. A statistical study of a group of psychotic children. In: Szurek, S.A., and Berlin, I.M., eds. *Clinical Studies in Childhood Psychoses*. London: Butterworth, 1973. pp. 303-347.

Bourgeois, M.; Malarrive, J.; and Charruaud, R. Les enfants des psychotiques (note préliminaire). *Société Médico-Pédagogique. Séance*, June 24, 1974.

Bovet du Bois, N. Le traitement neuroleptique au pédopsychiatrie: Analyse d'une série de 100 cas traités par la thioridazine. *Schweizerische Rundschau fur Medizin*, 62:1556-1564, 1973.

Brask, B.H. Borderline schizophrenia in children. *Acta Psychiatrica Scandinavica*, 34:265-282, 1959.

Brask, B.H. "A Prevalence Investigation of Childhood Psychosis." Presented at the 16th Scandinavian Congress of Psychiatry, 1970.

Brown, G., and Birley, J.L.T. Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behavior*, 9:203-214, 1968.

Carlier, M. La méthode des adoptions dans l'analyse génétique des psychoses schizophréniques: Revue critique. *Psychiatrie de l'Enfant*, 23:507-585, 1980.

Carlier, M., and Roubertoux, P. Psychoses à manifestatious précoses et psychoses à manifestations tardives: Apport de l'analyse génétique. *Psychiatrie de l'Enfant*, 22(2):473-502, 1979.

Challacombe, D. Coeliac disease and schizophrenia. *Lancet*, I:89, 1971.

Chiland, C., and Lebovici, S. Borderline or prepsychotic conditions in childhood. In: Hartocollis, P., ed. *A French Point of View in Borderline Disorders*. International University Press, Inc., 1974. pp. 143-183.

Connell, P.H. *Amphetamine Psychosis*. London: Chapman and Hall, 1958.

Corbett, J., and Harris, R. Progressive disintegrative psychosis of childhood. *Journal of Child Psychology and Psychiatry*, 18:211-219, 1977.

Cox, A.; Rutter, M.; Newman, S.; and Bartak L. A comparative study of infantile autism and specific receptive language disorder: II. Parental characteristics. *British Journal of Psychiatry*, 126:145-159, 1975.

Creak, E.M. Schizophrenia syndrome in childhood: Progress report of a working party. *Cerebral Palsy Bulletin*, 3:501-504, 1961.

Creak, E.M., and Ini, S. Family of psychotic children. *Journal of Child Psychology and Psychiatry*, 1:156-175, 1960.

de Sanctis, S. Folio neuro-bid, 2:9-12, 1908.

Despert, J.L. Schizophrenia in children. *Psychiatric Quarterly*, 12:366-371, 1938.

Diatkine, R. L'enfant prépsychotique. *Psychiatrie de l'Enfant*, 12(2):413-466, 1969.

Dockar-Drysdale, B. Residential treatment of frozen children. In: Dockar-Drysdale, B. *Therapy in Child Care (Collected Papers)*. London: Longman Green, 1968. pp. 14-27.

Dopchie, N. Réflexions sur la théorie des psychoses infantiles. *Acta Psychiatrica Belgica*, 73:527–536, 1973.

Earl, C.J.C. The primitive catatonic psychoses of idiocy. *British Journal of Medical Psychology*, 14:230–251, 1936.

Eggers, C. *Verlanjsweisen kindlicher und propuberoler schizophreniesc.* Berlin: Springer-Verlag, 1973.

Eisenberg, L. The course of childhood schizophrenia. *Archives of Neurology and Psychiatry*, 78:69–83, 1957.

Eisenberg, L. The classification of the psychotic disorders in childhood. In: Eron, L.D., ed. *Classification of Behavior Disorders*. Chicago: Aldine, 1966. pp. 87–114.

Fairbairn, W.R.D. *Psychoanalytic Studies of the Personality*. Tavistock Publications. New York: Basic Books, 1952.

Fish, F.J. *Schizophrenia.* Bristol: John Wright and Sons, Ltd., 1962.

Friedrich, M., and Leixnering, U. Differential diagnosis of schizophrenias in children and adolescents. *Acta Paedopsychiatrica*, 46(1–2): 21–30, 1980.

Gayral, L. L'entourage familial des schizophrènes. *Tunisie Medicale*, 73:5, 1973.

Gilyarovski, V.A. In: *Sovremennye problemy nevrologii*. Moscow, 1929. p. 48.

Goudemand, M.; Goudemand, J.; Goudemand-Joubert, C.; and Parquet, P.J. L'hérédité dans la psychose de l'enfant: Antigènes d'histocompatibilité (HLA A et B) et psychoses infantiles précoces. *Acta Psychiatrica Belgica*, 81:57–71, 1981.

Heller, T. About dementia infantilis. Reprinted in: Howells, J.G., ed. *Modern Perspectives in International Child Psychiatry*. Edinburgh: Oliver and Boyd, 1930. pp. 610–616.

Henry, R. Conceptions de la psychose infantile. *Praxis*, 31:1130–1135, 1970.

Hirsch, S.R. The scientific status of the theory that parents cause schizophrenia. [Author's translation] *Nervenarzt*, 50(6):337–345, 1979.

Hirsch, S.R., and Leff, J.P. *Abnormality in Parents of Schizophrenics: A Review of the Literature and an Investigation of Communication Defects and Deviance.* Maudsley Monograph No. 22. London: Oxford University Press, Inc., 1975.

Jørgensen, O. Psychopharmacological treatment of psychotic children: A survey. *Acta Psychiatrica Scandinavica*, 59:229–238, 1979.

Kanner, L. Autistic disturbances of affective contact. *The Nervous Child*, 2:217–250, 1943.

Kestenbaum, J. Psychothérapie de la schizophrénie infantile. *Psychotherapy and Psychosomatics*, 29:231–234, 1978.

Kety, S. The syndrome of schizophrenia: Unresolved questions and opportunities for research. *British Journal of Psychiatry*, 136:421–436, 1980.

Kety, S.S.; Rosenthal, D.; Wender, P.; and Schulsinger, F. The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In: Rosenthal, D., and Kety, S.S., eds. *The Transmission of Schizophrenia*. Oxford: Pergamon Press, 1968. pp. 345–362.

Klein, M. Notes on some schizoid mechanisms. *International Journal of Psychoanalysis*, 27:99–109, 1946.

Kolvin, I. Psychosis in childhood: A comparative study. In: Rutter, M., ed. *Infantile Autism: Concepts, Characteristics and Treatment.* London: Churchill-Livingston, 1971. pp. 7–26.

Kolvin, I. Emotional problems in childhood and adolescence: Late onset psychosis. *British Medical Journal*, 3:816–817, 1972.

Kolvin, I. Research into childhood psychoses: A cross-cultural comparison and commentary. *International Journal of Mental Health*, 2(3–4):194–212, 1974.

Kolvin, J.; Garside, R.; and Kidd, J.S.H. Studies in the childhood psychoses: IV. Parental personality and attitude and childhood psychoses. *British Journal of Psychiatry*, 118:403–406, 1971.

Kolvin, I.; Humphrey, M.; and McNay, A. Studies in the childhood psychoses: VI. Cognitive factors in childhood psychoses. *British Journal of Psychiatry*, 118(545):415–419, 1971.

Kolvin, I.; Ounsted, C.; Humphrey, M.; and McNay, A. Studies in the childhood psychoses: II. The phenomenology of childhood psychoses. *British Journal of Psychiatry*, 118:385–395, 1971.

Kolvin, I.; Ounsted, C.; Richardson, L. M.; and Garside, R. F. Studies in the childhood psychoses: III. The family and social background in childhood psychoses. *British Journal of Psychiatry*, 118:396–402, 1971.

Kolvin, J.; Ounsted, C.; and Roth, M. Studies in the childhood psychoses: V. Cerebral dysfunction and childhood psychoses. *British Journal of Psychiatry*, 118:407–414, 1971.

Kovalev, V. Current and unresolved problems in the differential diagnosis of schizophrenia and borderline states in childhood. *Zhurnal Nevropatologii i Psikhiatrii imeni*

S.S. Korsakova, 80(10):1503-1506, 1980.

Laing, R.D. The study of family and social contexts in relation to the origin of schizophrenia. In: Romano, J., ed. *The Origins of Schizophrenia.* Amsterdam: Excepta Medica Foundation, 1967.

Lebovici, S., and Chiland, C. Borderline or prepsychotic conditions in childhood. A French point of view. In: Hartocollis, P., ed. *Borderline Personality Disorders.* New York: International University Press, 1977. pp. 143-153.

Lebovici, S., and Diatkine, R. Essai d'approche de la notion de prepsychose au psychiatrie infantile. *Bulletin Psychologique,* 17:20-23, 1963.

Lebovici, S., and McDongall, J. *Un cas de psychose infantile. Etude psychoanalytique.* Paris: Presses Universitaires de France, 1960.

Lempp, R., and Vogel, B. Research in childhood schizophrenia. *Acta Paedopsychiatrica,* 33(10):322-331, 1966.

Leonhard, K. Contradictory issues in the origin of schizophrenia. *British Journal of Psychiatry,* 136:437-444, 1980.

Lidz, T. Schizophrenia and the family. *Psychiatry,* 21:21-27, 1958.

Lotter, V. Epidemiology of autistic conditions in young children: I. Prevalence. *Social Psychiatry,* 1:124-137, 1966.

Lotter, V. Epidemiology of autistic conditions in young children: II. Some characteristics of the parents and children. *Social Psychiatry,* 1(4):163-173, 1967.

Mahler, M.S. On child psychosis and schizophrenia: Autistic and symbiotic infantile psychosis. *Psychoanalytic Study of the Child,* 7:286-305, 1952.

Mayer-Gross, W.; Slater, E.; and Roth, M. *Clinical Psychiatry.* London: Cassell, 1955.

Mednick, S.A.; Schulsinger, F.; Higgins, J.; and Bell, B. *Genetics, Environment and Psychopathology.* New York: American Elsevier, 1974.

Michaux, L.; Dugas, M.; Colomb, F.; and Gueriot, C. Étude sur l'utilisation et l'activité therapeutique de l'oenanthate de fluphenazine, neuroleptique d'action prolongée, dans les psychoses de l'enfant. *Revue de Neuropsychiatrie Infantile,* 19(6):351-359, 1971.

Miller, R. Childhood schizophrenia: A review of selected literature. *International Journal of Mental Health,* 3(1):3-46, 1974.

Misès, R., and Barande, I. Étude clinique de formes précoces intriquant relation psychotique et symptomatologie de type déficitaire. *Psychiatrie de l'Enfant,* 6(1):1-78, 1963.

Nissen, G. Environment factors and subsequent schizophrenic psychosis in depressive children. *Archiv fur Psychiatrie und Nervenkrankheiten,* 21(4):319-323, 1971.

Nowak-Vogl, M. Thoughts on superficial diagnosis of childhood schizophrenia. *Praxis der Kinderpsychologie und Kinderpsychiatrie,* 28(6):199-201, 1979.

O'Gorman, G. The psychosis of childhood: The schizophrenic syndrome. In: Howells, J.G., ed. *Modern Perspectives in Child Psychiatry.* Edinburgh: Oliver and Boyd, 1958. pp. 473-492.

O'Gorman, G. *The Nature of Childhood Autism.* London: Butterworth, 1970.

Ornitz, E.M. Childhood autism: A disorder of sensorimotor integration. In: Rutter, M., ed. *Infantile Autism: Concepts, Characteristics and Treatment.* London: Churchill Livingstone, 1971. pp. 50-68.

Palacio-Espasa, F. Les états psychopatiques infantiles et les relations d'objects précoces. *Psychiatrie de l'Enfant,* 23(2):349-382, 1980.

Parmas, J.; Schulsinger, F.; Teasdale, T.; Schulsinger, H.; Felbman, P.; and Mednick, S. Perinatal complications and clinical outcome within the schizophrenia spectrum. *British Journal of Psychiatry,* 140:416-420, 1982.

Pogády, J., and Hasko, L. Psychopathology of schizophrenia in children and adolescents in relation to psychopharmacotherapy. [Author's translation] *Ceskoslovenska Psychiatrie,* 71(6):353-359, 1975.

Potter, H.W. Schizophrenia in children. *American Journal of Psychiatry,* 89:1253-1270, 1933.

Rosenfeld, H. "Reflection on Borderline and Atypical Cases: Borderline Functioning in a Traumatised Child." Presented at the 20th Anniversary of the Hampstead Clinic, London, 1972.

Rutter, M. Behavioral and cognitive characteristics of a series of psychotic children. In: Wing, J.K., ed. *Childhood Autism: Clinical, Educational and Social Aspects.* London: Pergamon Press, 1966. pp. 51-110.

Rutter, M. Psychotic disorders in early childhood. In: Coppen, A.J., and Walk, A., eds. *Recent Developments in Schizophrenia: A Symposium.* London: R.M.P.A., 1967. pp. 132-158.

Rutter, M. Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry,* 9:1-25, 1968.

Rutter, M. Childhood schizophrenia reconsidered. *Journal of Autism and*

Childhood Schizophrenia, 2(4):315-337, 1972.

Rutter, M. Diagnosis and definition. In: Rutter, M., and Schopler, E., eds. *Autism: A Reappraisal of Concepts and Treatment*. New York and London: Plenum Press, 1970. pp. 1-19.

Rutter, M.; Greenfeeld, D.; and Lockyer, L. A five to fifteen year followup of infantile psychosis: II. Social and behavioral outcome. *British Journal of Psychiatry*, 113:1183-1199, 1967.

Rutter, M.; Lebovici, S.; Eisenberg, L., Snezhnevsky, A.V.; Sadonn, B.; Brook, E.; and Lin, T. A triaxial classification of mental disorders in childhood. *Journal of Psychology and Psychiatry*, 10:41-61, 1969.

Rutter, M., and Lockyer, L. A five to fifteen year follow-up study of infantile psychosis: I. Description of sample. *British Journal of Psychiatry*, 113:1169-1182, 1967.

Rutter, M.; Shaffer, D.; and Sturge, G. *A Guide to a Multi-Axial Classification Scheme for Psychiatric Disorders in Childhood and Adolescence*. London: Department of Child and Adolescent Psychiatry, Institute of Psychiatry, 1975.

Schulsinger, F., and Mednick, S.A. Nature-nurture aspects of schizophrenia. In: Lader, M.H., ed. *Studies of Schizophrenia*. Ashford: Headley Brothers, 1975.

Schulsinger, H. A ten-year follow-up of children of schizophrenic mothers: Clinical assessment. *Acta Psychiatrica Scandinavica*, 53:371-386, 1976.

Schwoon, D.R., and Angermeyer, M.C. Congruence of personality assessments within families with a schizophrenic son. *British Journal of Medical Psychology*, 53:255-265, 1980.

Seabra-Dinis, T. Problèmes méthodologiques posés par la schizophrénie de l'enfant et le concept de schizophrénie en URSS. *Revue de Neuropsychiatrie Infantile*, 23(12):843-853, 1975.

Shields, J. Genetics. In: Wing, J.K., ed. *Schizophrenia: Towards a New Synthesis*. London: Academic Press, 1978. pp. 53-88.

Shields, J.; Heston, L.L.; and Gottesman, I.I. Schizophrenia and the schizoid: The problem for genetic analysis. In: Fieve, R.R.; Rosenthal, D.; and Brill, H., eds. *Genetic Research in Psychiatry*. Baltimore: Johns Hopkins University Press, 1975. pp. 167-197.

Simson, T.P. *Shizofreniya Rannego Detskogo Vozrosta*, Moscow, 1948.

Slater, E. The monogenic theory of schizophrenia. *Acta Genetica et Statistica Medica*, 8:50-56, 1958.

Soulé, M.; Houzel, D.; and Bollaert, Z. Les psychoses infantiles précoces et leur traitement. *Psychiatrie de l'Enfant*, 19(2):341-397, 1976.

Steinberg, D. Psychotic disorders in adolescence. In: Rutter, M., and Hersov, L., eds. *Child Psychiatry: Modern Approaches*. Oxford: Blackwell Scientific Publications, 1976. pp. 749-765.

Stroh, G. Three aspects of research into childhood psychosis. *Rivista Sperimentale di Freniatria*, 90(5):1325-1336, 1966.

Stroh, G. Psychotic children. In: Barker, ed. *The Residential Psychiatric Treatment of Children*. London: Grospy Lockwood Staples, 1974. pp. 175-204.

Sukhareva, G.E. "Trudy 2-go. Vsesoyznoyo s'ezdi." Unpublished scientific article. Neuropatologov. I. Psikhiatrov Moscow, 1937. p. 133.

Sukhareva, G.E., and Kogan, E.I. Prognosis of schizophrenia of childhood and puberty. *Soviet Psikhonevrologia*, 6:120, 1933.

Tischler, S. Clinical work with the parents of psychotic children. *Psychiatria, Neurologia, Neurochirurgia*, 74:225-249, 1971.

Tustin, F. *Autism and Childhood Psychosis*. London: Hogarth Press, 1972.

Ushakov, G. Symptomatology of the initial period of schizophrenia starting in childhood or adolescence. *Psychiatrie, Neurologie und Medizinische Psychologie*, 17(2):41-47, 1965.

Ushakov, G.K. Problems of diagnosing schizophrenia in adolescents. In: Caplan, G., and Lebovici, S., eds. *Psychiatric Approaches to Adolescence*. Amsterdam: Excerpta Medica Foundation, 1966.

Van Krevelen, D.A. Early infantile autism and autistic psychopathy. *Journal of Autism and Childhood Schizophrenia*, 1(1):82-86, 1971.

Van Krevelen, D.A. Psychoses in adolescence in modern perspectives. In: Howells, J.G., ed. *Adolescent Psychiatry*. Edinburgh: Oliver Boyd, 1971. p. 381.

Van Krevelen, D.A., and Knipers, C. The psychopathology of autistic psychopathy. *Acta Paedopsychiatrica*, 29:22-31, 1962.

Vrono, M. Schizophrenia in childhood and adolescence. *International Journal of Mental Health*, 2(3,4):8-116, 1974.

Vrono, M. Comparative age aspect of studies of schizophrenia in children and adolescents. *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova*, 80(10):1510-1514, 1980.

Wender, P.H.; Rosenthal, D.; Kety, S.S.; Schulsinger, F.; and Welner, J. Cross-fostering: A research strategy for clarifying the role of genetic and experimental factors in the etiology of schizophrenia. *Archives of General Psychiatry*, 30:121–128, 1974.

Wiener, P. Autisme infantile et symbiose psychotique: Un carrefour d'idées de la réflexion psychanalytique anglosaxonne. *Psychiatrie de l'Enfant*, 21(1):305–318, 1978.

Wing, J.K. Diagnosis, epidemiology, aetiology. In: Wing, J.K., ed. *Childhood Autism: Clinical Educational and Social Aspects*. London: Pergamon, 1966. pp. 3–49.

Wing, L. Childhood autism and social class: A question of selection. *British Journal of Psychiatry*, 137:410–417, 1980.

Wing, L.; Yeates, S.; Brierley, L.; and Gould, J. The prevalence of early childhood autism: Comparison of administrative and epidemiological studies. *Psychological Medicine*, 6:89–100, 1976.

Winnicott, D.W. Hate in the countertransference. London: Hogarth Press, 1947.

Winnicott, D.W. Psychoses and child care. In: Winnicott, D.W., ed. *Collected Papers*. London: Tavistock, 1952. pp. 219–228.

Winnicott, D.W. *The Theory of the Parent-Infant Relationship in the Maturational Process and the Facilitating Environment*. London: Hogarth Press, 1965.

Winnicott, D. The non-pharmacological treatment of psychosis in childhood. *Concilium Paedopsychiatricum: Proceedings of the Third European Congress of Paedopsychiatry*. Basel: Karger, 1967. pp. 193–198.

Wolf, S., and Barlow, A. Schizoid personality in childhood: A comparative study of schizoid, autistic and normal children. *Journal of Child Psychology and Psychiatry*, 20:29–46, 1979.

Wolf, S., and Chess, S. A behavioural study of schizophrenic children. *Acta Psychiatrica Scandinavica*, 40:438–466, 1964.

Wolf, S., and Chess, S. An analysis of the language of fourteen schizophrenic children. *Journal of Psychology and Psychiatry*, 6:29–41, 1965.

World Health Organization. *Report of the International Pilot Study of Schizophrenia*. Vol 1. Geneva: The Organization, 1973.

Wosiński, M. Early psychomotor development disorders in children with diagnosed schizophrenia. *Psychiatria Polska*, 7(3):277–281, 1973.

Wynne, L.C. *Methodological and Conceptual Issues in the Study of Schizophrenia*. Oxford: Pergamon Press, 1968.

Wynne, L.C.; Ryckoff, I.; Day, J.; and Hirsch, S. Pseudo-mutuality in the family relations of schizophrenics. *Psychiatry*, 21:205–220, 1958.

Wynne, L.C., and Singer, M.T. Thought disorder and family relations of schizophrenics. *Archives of General Psychiatry*, 9:196–206, 1963.

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Family Study of Schizophrenia: Exploratory Analysis for Relevant Factors

by Laura Bellodi, Clara Bussoleni, Raffaella Scorza-Smeraldi, Graziella Grassi, Luisa Zacchetti, and Enrico Smeraldi

Abstract

Two hundred twenty-nine schizophrenic patients, diagnosed according to *DSM-III* criteria, and their first degree relatives (except children) were studied. HLA A1 and CRAG A1 antigens were used as markers of probable dopaminergic pathology and, therefore, as possible indicators of genetic homogeneity that might identify subgroups of families with a specific and recognizable liability. Data were subjected to a logistic analysis in which the dependent variable was the presence of schizophrenia spectrum disorders in relatives, and the independent variables were the presence or absence of HLA A1 and CRAG A1 antigens, the sex of the proband, the sex of the relative, the severity of illness in the proband, and the type of relationship. The results for the entire sample demonstrate that the type of relationship, the sex of the proband, and the sex of the relative have significant effects on the risk of disorder in the relatives. In addition, the presence in the proband of one of the CRAG A1 antigens is a valid classification criterion for identifying a relatively homogeneous subgroup of families of schizophrenic patients.

There now appears to be genetic liability for development of schizophrenia, along with nongenetic factors and specific interactions. However, neither the mode of inheritance nor the biological and/or biochemical equivalents of this liability have as yet been identified (Matthysse and Kidd 1976). In fact, genetic analyses of the illness that might have answered such questions have always been inconclusive, mainly for psychopathological, genetic, or biological reasons.

From a psychopathological point of view, no genetic analysis can successfully define the phenotype if the clinical diagnosis of schizophrenia, which is the experimental component of the whole system, is not valid. In the past, differing clinical conceptions of the disease impeded unequivocal identification of the disease. This problem can be dealt with satisfactorily by use of the *DSM-III* criteria (American Psychiatric Association 1980), especially if information is obtained in semistructured interviews.

For this reason we decided to do genetic analyses of families selected after semistructured interviews based on criteria that would assure reliable psychopathological homogeneity of the probands. However, it should be kept in mind that these procedures do not also guarantee *clinical* homogeneity, because there are no data suggesting that the concept of schizophrenia adopted by *DSM-III* is also reliable genetically and biologically. Therefore, it is necessary to extend the analytical system to include factors and/or criteria for phenotypical homogeneity, which is different from clinical homogeneity. Moreover, from a genetic point of view, it must be remembered that schizophrenia is not inherited as a complete entity, but rather as a combination of risk factors, all of which contribute to genetic liability for the disease. Even if the clinical definition is sufficiently valid and reliable, we must look for those factors that constitute liability (genuinely important variables in the genetic analysis) and differentiate them from those variables that have stochastic meaning and serve only to confuse. For this reason, it is

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necessary to do preliminary exploratory analyses, without any assumptions about the origin or nature of the relevant factors, the results of which can then be used to formulate genetic hypotheses of transmission to be tested. The purpose of the present study was to identify some variables (e.g., sex of relative, sex of proband, severity of illness, and type of relationship) that could be used in genetic models.

Another requisite in exploratory analysis is that criteria for biological homogeneity also be introduced. Biological homogeneity is the hypothetical and identifiable level at which the genetic liability works. Different pathogenetic mechanisms that have proved valid for at least some subgroups of patients have been proposed. We think it most useful to use the most generally accepted hypothesis, dopaminergic hyperactivity (Kidd and Matthysse 1978). Even though there has been no direct demonstration of this in all schizophrenic patients, the hypothesis is based on the undoubted effectiveness of neuroleptics for schizophrenic disorders. However, it is a common experience in clinical practice that not all schizophrenic patients respond in the same way to this therapy, and these differences are not due simply to pharmacokinetic effects (Hollister and Kim 1982).

Although there is no reliable neurobiological way to identify patients who really have dopaminergic hyperactivity, the clinical outcome and the degree of effectiveness of neuroleptic therapy seem to be related to the presence of one of the HLA CRAG (cross-reacting antigens) A1 antigens (Smeraldi et al. 1976*a*; Cazzullo et al. 1979) and are associated with an inability of the dopaminergic receptor to modulate its sensitivity, as assessed by neuroendocrinological strategies (Brambilla et al. 1979; Smeraldi et al. 1980). In fact, these characteristics must be related to the specific binding of neuroleptics with CRAG A1 antigens (Scorza-Smeraldi et al. 1977). For all these reasons, we divided our families into two groups according to the presence or the absence in the proband of one of the HLA CRAG A1 antigens assumed to be a putative marker for dopaminergic pathology. Then, in an exploratory analysis of the risk for schizophrenia in the families, we tried to identify qualitative subgroups of families with specific and recognizable liability systems.

Methods

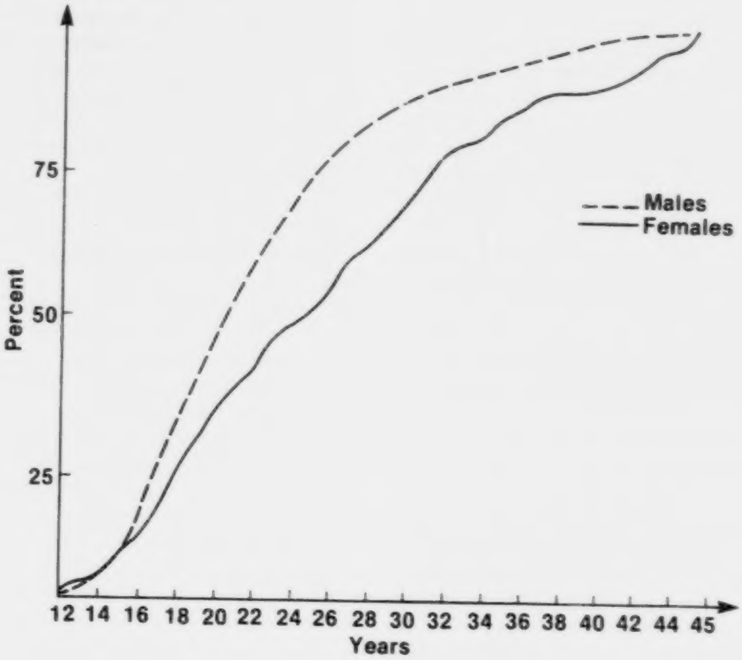
Subjects. We gave 229 consecutively admitted patients (ages 13–72 years; 143 men and 86 women) semistructured Schedule for Affective Disorders and Schizophrenia-Lifetime version (SADS-L) interviews (Spitzer and Endicott 1975). They then were diagnosed according to *DSM-III* as having schizophrenic disorders, paranoid type (60 subjects) or schizophrenic disorders, catatonic, disorganized, undifferentiated type (169 subjects). We have called the first subtype *mild* and all the others *severe*.

We also studied the patients' first degree relatives (457 parents and 588 siblings) except the children, because the children were few (reflecting reduced fertility of schizophrenics; see Vogel 1979) and too young to be at risk. Information about mental disease in the relatives was collected with the Family History-Research Diagnostic Criteria (FH-RDC) method (Andreasen et al. 1977) by specially trained staff, and the relatives were then diagnosed according to *DSM-III*. In our analysis we classified as belonging to the schizophrenia spectrum any relative who met the criteria for schizophrenic disorder, atypical psychosis, paranoid personality disorder, or schizotypal personality disorder. We classified relatives who met the criteria for other psychiatric disorders and relatives who were not mentally ill as not affected.

Since the age of onset for schizophrenic disorders shows a wide range, the data for the unaffected relatives were age-corrected according to Heimbuch, Matthysse, and Kidd (1980). We used different age corrections for males and females, according to the results of our previous work (Bellodi et al. 1982). The age-corrected values used in the present article were derived from the observed age-of-onset distribution of a larger group (317 subjects) of schizophrenics, also diagnosed according to *DSM-III* criteria, which includes subjects who were not typed. Figure 1 shows the observed age-of-onset distributions for males and females.

Statistical Analyses. We submitted our data to a logistic analysis for binary data (Cox 1970), as was done in a previous study (Morabito et al. 1982). With this method, one can detect differences between the independent variables and estimate weights β, which are then inserted into the equation: $\text{logit}\,[p(x|t)] = \beta' x + \text{logit}\,[p_0(t)]$, in which $\text{logit}\,(p) = \log\,[p/(1-p)]$, where $x = \{x_1, x_2, \ldots, x_{10}\}$ is a vector of the independent variables, $p\,(x|t)$ is the probability of onset at a given age t for a subject with x characteristics, $p_0(t)$ is the underlying distribution of onset common to all subgroups, and $\beta' x = \beta_1 x_1 + \ldots + \beta_{10} x_{10}$ is a linear combination of x variables with β weights. The dependent variable was the presence of the

Figure 1. Observed age of onset distribution of schizophrenic probands (n = 317)



schizophrenia spectrum in first degree relatives. The following independent variables were tested as "main effects": (a) sex of the proband, female (0) or male (1); (b) sex of the relative, female (0) or male (1); (c) type of relationship of the relative, parent (0) or sib (1); (d) severity of illness in the proband, mild (0) or severe (1); (e) HLA CRAG A1, presence (0) or absence (1).

HLA Typing. Patients were typed blind to the HLA-A and B loci, as described by Smeraldi et al. (1976*b*), and divided into HLA A1 and CRAG A1 (HLA A3, HLA A10, HLA A11) positive (137 subjects) and HLA A1 and CRAG A1 negative (92 subjects).

Results

The observed risks for schizophrenia spectrum disorders in the members of our families are shown in tables 1*a* and 1*b*, classified according to the HLA typing of the proband (see **Methods**) and listed according to the variables considered. Maximum likelihood estimates of the β values and their significances arrived at by logistic analyses with increasingly comprehensive models are listed in table 2. As can be seen in the table, all main effects and two- and three-way interactions that might explain observed risks were taken into account.

As expected, the most significant parameter was found to be the mean frequency of the disease in relatives. The variable "type of relationship" was also statistically significant, as can be seen from model II in table 2; for the entire sample, the observed risk was higher for the siblings (9.10 percent) than for the parents (4.83 percent). The two variables related to a possible sex effect appear to have different meanings statistically; there is, in fact, a lower risk for relatives of male probands and for male relatives of probands, though the lower risk is statistically significant only for relatives of male probands. The relatives of male probands show a risk of 5.13 percent (5.5 percent in males vs. 4.73 percent in females), and the relatives of female probands show a risk of 9.48 percent (8.87 percent in males vs. 12.23 percent in females).

The variable "severity" of the proband, although not statistically significant when considered by itself, appears to be nearly significant in interaction with the variable "sex of the proband," as can be seen in model IV in table 2; in other words, the risk is lower (3.16 percent) only in the relatives of male mild probands vs. the relatives of male severe probands (6.46 percent). For the relatives of female probands, the trend for the relationship between severity and sex of proband is in the opposite direction.

The last model we applied included classification according to HLA typing of the proband and the interactions involved. With this model, we observed that: (a) The significance of type of relationship and sex of the proband remained essentially the same. (b) The interaction between sex of the proband and the severity of the form of the disease, as described above, became significant. In the relatives of HLA A1 and CRAG A1 positive probands, we observed a systematic trend toward

Table 1a. Age-corrected frequency of schizophrenia spectrum disorders in relatives of schizophrenic probands who are HLA CRAG A1 positive (n = 137)

	Mild (n = 39)		Severe (n = 98)	
Relatives	**Male (n = 19)**	**Female (n = 20)**	**Male (n = 60)**	**Female (n = 38)**
Fathers				
Observed affected	0	0	4	1
Age-corrected total	18.99	20.	60.	36.97
Observed risk			6.66%	2.71%
Mothers				
Observed affected	1	1	2	4
Age-corrected total	18.85	19.88	59.65	37.76
Observed risk	5.31%	5.03%	3.34%	10.59%
Brothers				
Observed affected	1	2	4	6
Age-corrected total	27.83	30.54	43.94	42.57
Observed risk	3.70%	6.55%	9.10%	14.09%
Sisters				
Observed affected	1	7	2	4
Age-corrected total	19.85	30.37	38.33	31.72
Observed risk	5.04%	23.05%	5.21%	12.61%

an increased risk for schizophrenia spectrum disorders in relatives of female vs. male probands and in relatives of severe vs. mild probands. For the relatives of HLA A1 and CRAG A1 negative probands, this trend was found only for sex of proband, while the severity of proband classification did not seem to affect risk. (c) The interaction of the severity of the proband, the sex of relative, and the HLA typing of the proband also appeared to be statistically significant, indicating different risk structures in the two subgroups. In other words, in the relatives of patients positive for HLA A1 and CRAG A1, there was a consensual trend of the risk that depended on both the severity of the proband's illness and the sex of the relative (always greater in females than in males). By contrast, severity of illness in probands negative for HLA A1 and CRAG A1 was not associated with increased risk for the relatives.

Discussion

The existence of a familial concentration of schizophrenia among relatives of schizophrenic probands is one of earliest and most common observations about the disease. Recent studies of susceptibility to schizophrenia that have used the restrictive operative criteria of *DSM-III* to diagnose disorder in the probands and in the relatives have found inconsistent results, eliciting debate on the matter (Pope et al. 1982; Kendler 1983; Weissman et al. 1983). Both the entity of the risk in the relatives and the familial concentration of the disease have been discussed, and there is now some doubt whether the prevalence of the disease is really significantly higher in the relatives than in the general population. Such differences of opinion could be approached more usefully by studying larger, preferably prospective samples and taking into account methodological characteristics, as Abrams and Taylor (1983) recently did. Since we have followed our patients and their families for several years, our study might be considered to have been prospective.

The risk for schizophrenia spectrum disorders that we observed in our sample was 6.48 percent. Since we have no statistically valid estimates of the general prevalence of schizophrenic disorders in the Italian population, we do not know whether this figure represents a risk significantly greater than chance, but it appears to be higher than those generally given for prevalence in the general population. In the United States, Guze et al. (1983) also found a higher risk for the disease, 8.1 percent, when both definite schizophrenia and probable schizophrenia were included as secondary cases.

Sixty-three first degree relatives of our probands were found to have schizophrenia spectrum disorders. Among these, 34 subjects met criteria

Table 1*b*. Age-corrected frequency of schizophrenia spectrum disorders in relatives of schizophrenic probands who are HLA CRAG A1 negative ($n = 92$)

Relatives	**Mild ($n = 21$)**		**Severe ($n = 71$)**	
	Male ($n = 14$)	**Female ($n = 7$)**	**Male ($n = 50$)**	**Female ($n = 21$)**
Fathers				
Observed affected	1	1	2	1
Age-corrected total	14.	6.95	49.83	21.
Observed risk	7.14%	14.38%	4.01%	4.76%
Mothers				
Observed affected	0	0	2	2
Age-corrected total	14.	7.	49.75	20.92
Observed risk			4.02%	9.56%
Brothers				
Observed affected	1	1	2	1
Age-corrected total	21.47	5.96	36.61	25.29
Observed risk	4.66%	16.77%	5.46%	3.95%
Sisters				
Observed affected	0	3	4	1
Age-corrected total	28.40	7.92	24.44	24.34
Observed risk		37.87%	16.37%	4.11%

Table 2. Logistic analysis of observed risk for schizophrenia spectrum disorders in relatives of schizophrenic probands

	Model I		**Model II**		**Model III**		**Model IV**		**Model V**	
	β	***z*-value**	β	***z*-value**	β	***z*-value**	β	***z*-value**	β	***z*-value**
Mean	−2.60	−19.74*	−2.64	−19.26*	−2.63	−18.98*	−2.73	−16.76*	−2.76	−16.07*
Type of relationship			−0.34	−2.48*	−0.32	−2.34*	−0.35	−2.50*	−0.35	−2.53*
Sex of the proband					−0.31	−2.29*	−0.43	−2.74*	−0.45	−2.76*
Sex of the relative					−0.15	−1.12	−0.15	−1.13	−0.16	−1.04
Severity (mild/severe)							−0.14	−0.86	−0.13	−0.77
Sex of the proband × severity							−0.30	−1.91	−0.33	−2.03*
HLA type									−0.07	−0.46
HLA type × sex of the relative									−0.16	−0.99
HLA type × severity									−0.21	−1.30
Severity × sex of the relative									−0.04	−0.28
HLA type × severity × sex of the relative									−0.33	−2.07*
Initial maximal log likelihood	−620.595		−620.593		−620.595		−620.595		−620.595	
Final maximal log likelihood	−225.345		−222.140		−218.839		−216.578		−213.579	

*At $p = .05$, *z*-value is 1.96 since *z*-values are normally distributed.

for schizophrenic disorder, 20 subjects for atypical psychosis (with four cumulative suicides), and five for schizotypal personality disorder. We classified the atypical psychoses identified only by a family history method as schizophrenia spectrum disorders, since the method we used underestimates the frequency of the disease in relatives (Thompson et al. 1982) and it is a psychosis about which there is inadequate information for more specific diagnosis. We have also included as schizophrenia spectrum disorders relatives who met criteria for schizotypal personality disorder, since recent diagnostic reviews of secondary cases among biological relatives of adopted schizophrenic subjects have identified at least two personality disorders (i.e., schizotypal personality disorder and paranoid personality disorder) as components of the schizophrenia spectrum (Kendler, Gruenberg, and Strauss 1981; Kendler and Gruenberg 1982).

We used the traditional, most widely accepted forms of schizophrenia spectrum disorders—those that are symptomatically similar to schizophrenia but of a lesser clinical severity and not always psychotic in nature. For practical purposes, the concept of disease spectrum is useful in genetic studies. It can be taken as an incomplete phenotypic manifestation of susceptibility and, as such, can be used as an index of susceptibility, even without identification of the biological equivalents of the true phenotype. For research purposes, pathologies that do not resemble schizophrenia, but are found in above-chance concentrations in families selected on the basis of a schizophrenic proband, should be included in the analysis. We excluded only alcoholism in this study. However, it has recently been reported that alcoholism is more frequent in monozygotic twins than in dizygotic twins discordant for schizophrenia (Kendler and Robinette 1983). Moreover, the distribution of cases of alcoholism in our sample did not appear to be random, since all except two cases were relatives of HLA A1 and CRAG A1 positive probands. Therefore, the presence of alcoholism might be considered as an index of susceptibility, especially in those studies which include HLA typing of the probands.

We feel that the different risk values we found in the parents and siblings are not due to a methodological bias resulting from different ranges of actual ages of parents and siblings. It might be a generational effect rather than a type of relationship effect. However, the age structure of our sample does not support this idea, since it includes young parents and old siblings. The differences in risk we observed could be explained by the main effect of the "type of relationship" according to previous data (Kety 1983), which might be explained by genetic factors (e.g., the effect of dominance, penetrance defect, assortative mating, and differential fertility), though our descriptive design does not allow us to identify such factors. Finally, in our sample as a whole, the age correction for the relatives unaffected at time of observation affects the siblings more than the parents and might result in an overestimation of the risk in the siblings.

We also found a lower risk for the disease in the relatives of male probands than in the relatives of female probands. This suggests that females could have a greater genetic load whose clinical manifestation is masked and which does not result in a differential prevalence of disease by sex. (Schizophrenia is believed to occur with roughly equal frequency in adult males and females; see Lewine, Watt, and Grubb 1981.) Failure to observe sex differences in disease prevalence could be explained by biological factors, such as the presence in the females of more estrogen, whose antidopaminergic activity is known (Raymond et al. 1978). This same hormonal influence could delay the onset of the disease. Analysis of the age-at-onset distribution curve shows a slight but definite trend toward later ages in the females (Bellodi et al. 1982; Loranger 1984).

The major aim of the present study was to see whether the presence or absence of one of the HLA A1 and CRAG A1 antigens in the proband could identify subgroups of schizophrenics that independently of the clinical similarity of their disease show specific familial risks for schizophrenia spectrum disorders. Our use of HLA typing is substantially different from its use in the pure studies of association in the literature (for review, see Smeraldi et al. 1980), in which investigators attempted to demonstrate that either all schizophrenics or some clinically defined subgroups carried one or more HLA antigens with frequencies different from those in control populations. Because of methodological difficulties, such studies often show weak associations (if demonstrable at all) that involve different antigens depending upon the ethnic origin of the population examined. The antigens examined may not play a specific role in the disease, but are taken into account only because they are closely linked to a disease susceptibility locus and in linkage disequilibrium with alleles at that locus. The ability of the HLA A1 and CRAG A1 antigens to interfere with the neuroleptics (Scorza-Smeraldi et al. 1977), the only drugs able to modulate the disease significantly, and their probable relationship to some pathology of the dopaminergic receptor suggest that they might be involved directly in the pathogenesis or in the biological component of the pathogenesis, at least in those cases in which they are indeed present.

First, we see that the mean risks for the relatives of HLA A1 and CRAG A1 positive and negative probands did not differ, since the variable presence/absence of such antigens was not statistically significant, per se. In this regard, and since there is recent debate about the validity of classifying schizophrenia as familial or sporadic (Kendler and Hays 1982), we should note that in our two groups differentiated by HLA typing of the proband, there were different percentages of families with and without affected relatives. In 28.47 percent of the families of HLA A1 and CRAG A1 positive probands in our sample, there was at least one affected relative, whereas there was an affected relative in only 17.39 percent of the families of negative probands. Because the mean frequencies of disease in the relatives of the groups did not differ significantly, we calculated the probability of finding families with or without affected relatives, including the mean size of the families of the two groups in this calculation (Kendler and Hays 1983). The values obtained were 26.1 percent for the families of the HLA A1 and CRAG A1 positive probands, which is not very different from the 28.47 percent observed, and 26.82 percent for the families of HLA A1 and CRAG A1 negative probands, which appears to be higher than the 17.39 percent observed. We feel that this difference in the latter group was due to a tendency of the secondary cases to group within certain families in such a way as to cause dishomogeneity of the distribution of risk. In the statistical analyses that we are using at present, this information escapes us because our method involves contingency tables constructed from the mean values for each group. Nevertheless, it should be taken into account when doing a genetic analysis that is based on the structure of individual families.

The presence of HLA A1 and CRAG A1 antigens in the probands appears to have a significant effect on the interaction of clinical severity of the form and the possible sex effect identified by the two variables, sex of the proband and sex of the relative. More precisely, only in the families of the HLA A1 and CRAG A1 positive probands were these variables significant and cooperative in increasing the risk in relatives, mild vs. severe and male vs. female. In the relatives of negative probands, only the sex of the proband, per se, appears to be significant. The more probable interpretation of these data is that the classification, *mild/severe*, as defined in our analysis, affects the risk for the disease in the relatives only in families of HLA A1 and CRAG A1 positive probands and interacts with the same effect as sex. Furthermore, the observation that the familial structure (the entity of the risk is not the only factor) of these probands shows peculiar features of the susceptibility that distinguish them from the families of other schizophrenics enhances the hypothesis of a direct antigenic effect.

Certainly, we have not proved any direct effect of HLA A1 and CRAG A1 antigens in a subgroup of schizophrenics similar to the effects for HLA antigens demonstrated in ankylosing spondylitis and hemochromatosis, but we have demonstrated an additional characteristic consistent with such a hypothesis, i.e., HLA A1 and CRAG A1 positive patients can be used, for purposes of genetic research, as a homogeneous subgroup of the disease. A real demonstration of a direct antigenic effect would require the specification of the relationship between the HLA type and susceptibility to the disease. Only the positive results of the linkage studies between the HLA and illness in these families seem to be probative.

To do studies of this kind properly, we will need to identify the formal parameters of the genetic susceptibility for the disease. The usefulness of exploratory analyses like ours consists in detecting possibly related variables for the formulation of genetic hypotheses that can be tested by experimental data. Since the familial structures of the risk of the HLA A1 and CRAG A1 positive and negative probands appear to be different, it would tentatively be useful to subdivide experimental samples according to the HLA typing of the proband. In the HLA A1 and CRAG A1 positive subgroup, when testing the single major locus and multifactorial polygenic hypotheses, as well as even more complex hypotheses which include assortative mating and low fertility (Vogel 1979; Merikangas 1982), we need to consider the severity of the forms of the disease as defined above; we also need to consider the existence of a sex effect. These parameters appear to be meaningless in the second group of negative probands, in which other unidentified factors are probably involved.

We think that the application to genetic studies of operational procedures similar to those we have developed, classifying schizophrenic probands according to HLA typing, and the application of genetic hypotheses derived from the observed familial risk to biologically homogeneous subgroups of the disease can increase our ability to generate new testable hypotheses for even more complicated systems of analysis.

References

Abrams, R., and Taylor, M.A. The genetics of schizophrenia: A reassessment using modern criteria. *American Journal of Psychiatry*, 140:171–175, 1983.

Andreasen, N.C.; Endicott, J.; Spitzer, R.L.; and Winokur, G. The family history method using diagnostic criteria: Reliability and validity. *Archives of General Psychiatry*, 34:1229–1235, 1977.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

Bellodi, L.; Morabito, A.; Macciardi, F.; Gasperini, M.; Benevenuto, M.G.; Grassi, G.; Marzorati-Spairani, C.; and Smeraldi, E. Analytic considerations about observed distribution of age of onset in schizophrenia. *Neuropsychobiology*, 8:93–101, 1982.

Brambilla, F.; Bellodi, L.; Negri, F.; Smeraldi, E.; and Malagoli, G. Dopamine receptor sensitivity in the hypothalamus of chronic schizophrenics after haloperidol therapy: Growth hormone and prolactin response to stimuli. *Psychoneuroendocrinology*, 4:329–339, 1979.

Cazzullo, C.L.; Smeraldi, E.; Bellodi, L.; Negri, F.; and Bonara, P. The HLA system and the clinical response to treatment with haloperidol. In: Saletu, B. et al., eds. *Neuropsychopharmacology*. Oxford and New York: Pergamon Press, 1979. pp. 47–53.

Cox, D.R. *Analysis of Binary Data*. New York: Chapman and Hall, 1970.

Guze, S.B.; Cloninger, C.R.; Martin, R.L.; and Clayton, P.J. A follow-up and family study of schizophrenia. *Archives of General Psychiatry*, 40:1273–1276, 1983.

Hollister, L.E., and Kim, D.Y. Intensive treatment with haloperidol of treatment-resistant chronic schizophrenic patients. *American Journal of Psychiatry*, 139:1466–1468, 1982.

Heimbuch, R.C.; Matthysse, S.; and Kidd, K.K. Estimating age of onset distribution for disorders with variable onset. *American Journal of Human Genetics*, 32:564–574, 1980.

Kendler, K.S. Heritability of schizophrenia. *American Journal of Psychiatry*, 140:131, 1983.

Kendler, K.S., and Gruenberg, A.M. Genetic relationship between paranoid personality disorders and the "schizophrenic spectrum" disorders. *American Journal of Psychiatry*, 139:1185–1186, 1982.

Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia: II. Relationship between schizotypal personality disorder and schizophrenia. *Archives of General Psychiatry*, 38:982–984, 1981.

Kendler, K.S., and Hays, P. Familial and sporadic schizophrenia: A symptomatic, prognostic and EEG comparison. *American Journal of Psychiatry*, 139:1557–1562, 1982.

Kendler, K.S., and Hays, P. Questioning the validity of "familial" versus "sporadic" schizophrenia. *American Journal of Psychiatry*, 140:1266, 1983.

Kendler, K.S., and Robinette, C.D. Schizophrenia in the National Academy of Sciences-National Research Council Twin Registry: A 16-years' update. *American Journal of Psychiatry*, 140:1551–1563, 1983.

Kety, S.S. Mental illness in the biological and adoptive relatives of schizophrenic adoptees: Findings relevant to genetic and environmental factors in etiology. *American Journal of Psychiatry*, 140:720–727, 1983.

Kidd, K.K., and Matthysse, S. Research designs of the study of gene-environment interactions in psychiatric disorders: Report of an FFRP panel. *Archives of General Psychiatry*, 35:925–932, 1978.

Lewine, R.R.J.; Watt, N.F.: and Grubb, T.W. High-risk-for-schizophrenia research: Sampling bias and its implications. *Schizophrenia Bulletin*, 7:273–280, 1981.

Loranger, A.W. Sex difference in age at onset of schizophrenia. *Archives of General Psychiatry*, 41:157–161, 1984.

Matthysse, S., and Kidd, K.K. Estimating the genetic contribution to schizophrenia. *American Journal of Psychiatry*, 133:185–191, 1976.

Merikangas, K.R. Assortative mating for psychiatric disorders and psychological traits. *Archives of General Psychiatry*, 39:1173–1180, 1982.

Morabito, A.; Gasperini, M.; Macciardi, F.; and Smeraldi, E. Possible relationship between outcome in primary affective disorders treated with lithium and family history. In: Costa, A., and Racagni, G., eds. *Typical and Atypical Antidepressants*. New York: Raven Press, 1982.

Pope, H.G.; Jonas, J.M.; Cohen, B.M.; and Lipinski, J.F. Failure to find evidence of schizophrenia in first degree relatives of schizophrenic probands. *American Journal of Psychiatry*, 139:826–830, 1982.

Raymond, V.; Beaulieu, M.; Labrie, F.; and Boisser, J. Potent antidopaminergic activity of estradiol at the pituitary level on prolactin release. *Science*, 200:1173–1175, 1978.

Scorza-Smeraldi, R.; Smeraldi, E.; Fabio, G.; Bellodi, L.; Sacchetti, E.; and Rugarli, C. Interference between anti-HLA antibodies and adrenergic receptor binding drugs. *Tissue Antigens*, 9:163–166, 1977.

Smeraldi, E.; Bellodi, L.; Sacchetti, E.; and Cazzullo, C.L. The HLA system and the clinical response to treatment with chlorpromazine. *British Journal of Psychiatry*, 129:486–489, 1976*a*.

Smeraldi, E.; Bellodi, L.; Scorza-Smeraldi, R.; Fabio, G.; and Sacchetti, E. HLA-SD antigens and schizophrenia: Statistical and genetical considerations. *Tissue Antigens*, 8:191–196, 1976*b*.

Smeraldi, E.; Brambilla, F.; Scorza-Smeraldi, R.; Maffei, C.; Vanzulli, L.; and Bellodi, L. Dopamine receptor sensitivity in chronic schizophrenia: A study on HLA system. In: Racagni, G.; Brambilla, F.; and de Wied, D., eds. *Progress in Psychoneuroendocrinology*. Amsterdam: Elsevier/North-Holland Biomedical Press, 1980. pp. 507–519.

Spitzer, R.L., and Endicott, J. *Schedule for Affective Disorders and Schizophrenia-Lifetime Version*. New York: New York State Psychiatric Institute, 1975.

Thompson, W.D.; Orvaschel, H.; Prusoff, B.A.; and Kidd, K.K. An evaluation of family history method for ascertaining psychiatric disorders. *Archives of General Psychiatry*, 39:53–58, 1982.

Vogel, H.P. Fertility and sibship size in a psychiatric patient population. *Acta Psychiatrica Scandinavica*, 60:483–503, 1979.

Weissman, M.M.; Merikangas, K.R.; Pauls, D.L.; Leckman, J.F.; and Gammon, G.D. Heritability of schizophrenia. *American Journal of Psychiatry*, 140:131–132, 1983.

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Expressed Emotion in Families

Expressed Emotion in Families: Its Significance for Mental Illness, authored by Julian Leff and Christine Vaughn, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). The discovery that discharged schizophrenic patients who returned home to parents or spouses often fared worse than those living alone led researchers to look for conditions within the family that might influence the schizophrenic patient's condition. At the vanguard of this endeavor was George Brown, whose recognition of the debilitating effect of high levels of expressed emotion—such as hostility, criticism, and overinvolvement—has stimulated important insights as well as considerable controversy. In this volume, Leff and Vaughn, together with two other prominent investigators, address some of the confusions and misconceptions that have arisen regarding the measures of expressed emotion and the techniques for obtaining data, and present important new findings which significantly expand Brown's original insights. The book will be of great interest to psychiatric researchers as well as to all mental health professionals who work with schizophrenic patients and their families.

The Inpatient Care of the Chronically Mentally Ill

by John A. Talbott and Ira D. Glick

Abstract

The inpatient treatment of the chronically mentally ill as it has been practiced in the past, is practiced at the present, and may be practiced in the future is discussed. The reasons for admitting the chronically ill to inpatient services; the steps of such treatment and treatment modalities employed; and considerations for discharge are presented. In addition, the indications as well as pros and cons for long-term inpatient hospitalization are included.

The inpatient treatment of the chronically mentally ill has undergone many changes in the last few decades. To understand better where we are now, we will summarize the history of treatment and care of the chronically ill; selectively review the literature about this treatment; summarize length-of-stay studies; discuss the current reasons for admission of chronic patients to inpatient services; review those elements of inpatient treatment that particularly pertain to this population; summarize their long-term treatment; discuss the need for "asylum," as a concept not a building; characterize the discharge planning necessary; and finally forecast some future trends in the treatment and care of this population.

Historical Background

Since America's earliest beginnings, the care of the chronically mentally ill has undergone radical changes in both its locus and method of delivery. In essence, we have moved from custodial care in long-term public settings to active treatment and rehabilitation in a multiplicity of settings.

In colonial times, no specific provision was made for the mentally ill (Dain 1976). With time, counties established poorhouses, almshouses, and workhouses that included the mentally ill among their charges. These were replaced by State hospitals that by 1860 existed in 28 of the then 33 States (Rothman 1971). By the turn of the 19th century a series of alternatives to these public facilities began to appear: psychopathic, private, Veterans Administration, Public Health Service, and general hospitals (Talbott 1983). Beginning in the 20th century, outpatient clinics became commonplace, but hospitals, despite their problems (Deutsch 1949), remained the primary locus for care and treatment of those suffering from chronic illness.

In 1955, State hospitals achieved their highest censuses, and their primacy in delivery of mental health services thereafter began to decline (Kramer 1975). Several forces, then and in the 1960s, resulted in a massive shift from public hospital to community auspices. These forces included (1) the philosophy of community mental health, (2) pharmacological and social therapeutic innovations, (3) pressures from civil libertarians, and (4) Federal assumption of funding for the chronically ill in communities through Medicaid, Medicare and Supplemental Security Income (Talbott 1979).

The net result of this historic shift in locus of care and treatment of the chronically ill was twofold: (1) The mental hospital's role was altered from that of the sole place for

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treatment and care, to but one element in the care spectrum (and that, often, as a last resort). (2) The entire system moved from providing predominately custodial care in asylums to active treatment and rehabilitation in the community.

Paralleling this shift in the locus of delivery has been a striking change in our understanding of the treatment of chronic mental illness, which will be summarized in the following section.

Selective Review of the Literature

There is an extensive literature on the treatment of schizophrenia, but most of it concentrates on the treatment of acute conditions and there are few controlled studies. Recently, however, several reviews that are more applicable to the chronically ill have appeared (Davis 1975). For example, May (1975) concluded that psychotherapy was not essential to the inpatient treatment of chronic schizophrenia, but was for outpatient care; that group psychotherapy was more effective than individual treatment; and that drugs alone were not sufficient to prevent relapses in outpatients.

In addition, Mosher and Gunderson (1979) suggested that a highly structured, organized, expectant milieu that stressed adaptive skills, while suppressing symptoms and maladjustive behavior, was helpful in the hospital treatment of chronic schizophrenia. Finally, Paul and Lentz (1977) demonstrated that a highly organized resocialization-relearning program, with a behavior treatment orientation (e.g., token economy), enabled chronic schizophrenics to stay in the community much longer than those receiving milieu therapy or traditional State hospital care. Patients treated in the behavioral program, which stressed the skills of everyday living essential to community survival, were rehospitalized or never released by the time of final followup less than 10 percent of the time, compared to 30 percent of those in the milieu program and 50 percent of those treated "traditionally."

A series of studies of chronic schizophrenics have compared alternatives to hospitalization with inpatient treatment. In brief, whether this treatment is characterized by home care, family treatment, day hospital care, residential nonhospital treatment, or community-based community support, there is a consistent conclusion (Braun et al. 1981; Kiesler 1982) that alternative care is *as*, if not *more*, effective than inpatient treatment in reducing symptomatology, rehospitalization, interpersonal difficulties, and vocational disablement. While the cost of high quality community care is still quite high, it is a bit less than hospital care plus traditional followup (Weisbrod, Test, and Stein 1980) because of the savings in staff and housing costs. To summarize this literature, although there are strong beliefs, there are no data to support the use of hospitalization instead of outpatient alternatives for most of these patients. Despite this, hospitalization remains a popular treatment in managing the chronic patient—in part, because so many clinicians now practicing were trained in an era when the hospital was seen as the safest and, therefore, most logical treatment for extremely impaired patients. Further, hospitalizing the patient appears to be a way of stabilizing and calming everyone in the patient's social network. There is a tendency to overlook the drawbacks of hospitalization—the financial costs; the fact that the more an individual is hospitalized, the greater the future use of and dependency on the hospital to solve a crisis; the potential harm to the patient's self-esteem as well as role functioning; and the conditioning effect of hospitalization. In view of these problems, a careful differential therapeutic decision should be made before hospitalizing any patient with chronic mental illness and especially before prolonging a hospitalization beyond a relatively brief stay (Glick, Klar, and Braff 1984).

Length-of-Stay Studies

Length-of-stay studies have consistantly demonstrated no advantage for those chronic schizophrenics (defined as having three or more hospitalizations) treated for long (30–90 days) rather than short (under 30 days) periods of hospitalization. In fact, in terms of symptomatology, rehospitalization, and quality of life, short stays seem advantageous.

There are now several controlled studies of outcome of short-versus-long hospitalization. One was undertaken by Herz, Endicott, and Spitzer (1976, 1977), the second by Mattes and his associates (Mattes, Rosen, and Klein 1977; Mattes et al. 1977), and the third by Glick and Hargreaves (1979). All of these studies have been reviewed by Riessman, Rabkin, and Struening (1977).

The study of Herz, Endicott, and Spitzer (1976, 1977) excluded patients who were a danger to themselves or others, as well as patients without families. Patients in their diagnostically mixed sample were randomly assigned to standard inpatient care (60 days), brief hospitalization (11 days) plus day care upon discharge, or brief hospitalization only. After 1 year, they found that those assigned to brief hospitalization plus day care showed the least psychopathology, while those who had received standard care showed the most disturbance. Significant differences between groups in symptom levels had disappeared after the second year. Moreover, patients from the two brief-hospitalization groups were more likely to be working at each followup point. Furthermore, more of the standard-care group were judged as failing to perform any occupational role, including student or housekeeper, as well as wage earner.

The study of Mattes and associates (Mattes, Rosen, and Klein 1977; Mattes et al. 1977) also had a diagnostically mixed sample of patients. Their patients were assigned either to short-term wards with a 90-day stay or to long-term wards with a mean stay of 6 months. They found that short-term patients showed greater improvement at discharge than the long-stay patients. Three years after discharge, there were few between-group differences. Long-term patients were rated by relatives as having less pathology, but since they received more individual psychotherapy, which may have resulted in lower ratings, this finding was discounted. Among patients who were rehospitalized, long-term patients tended to be rehospitalized more often and for longer periods.

Glick, Klar, and Braff (1984) randomly assigned patients either to short-term inpatient care (21–28 days) or to long-term hospitalization (3–4 months). The sample was stratified by diagnosis into patients with schizophrenic disorders, affective disorders, and other nonschizophrenic diagnoses. After 1 year, with no posthospital care being provided, the schizophrenics treated for a longer time were doing significantly better on two global measures than the short-term patients. However, discrete symptom scales failed to differentiate the groups, and there was no difference in rehospitalization data. The long-term group received significantly more psychotherapy after discharge, as well as medication at significantly higher dosage levels, than the short-term group. The authors concluded that their "work with patients and families during the longer hospitalization period effectively encouraged acceptance of the need for continued psychotherapy and medication" (p. 90). After 2 years, the clinical global scale again favored the long-term group, but the relationship was less strong than at 1 year. On further analysis, the investigators found that long-term hospitalization produced a better outcome only in those schizophrenic patients whose prehospital functioning had been relatively good. For those with relatively poor prehospital functioning, longer hospitalization appeared to be detrimental to outcome. In contrast to the findings for the schizophrenic patients, 1- and 2-year followups of the nonschizophrenic patients in long- and short-term treatment revealed no significant differences in global ratings, severity of symptom measures, or social and work adjustment.

"All three studies, when taken together, have generally failed to show important differences in the effects of brief versus standard hospitalization" (Glick and Hargreaves 1979, p. 94). Patients treated by either strategy show a rapid reduction in symptomatology, although the speed with which this occurs may be greater during brief hospitalization. Adult roles in the community are resumed more quickly by short-term patients in several studies, perhaps because continuity in identity is maintained. Nevertheless, these initial disadvantages of long-term patients are made up as soon as they are discharged. Riessman, Rabkin, and Struening (1977) concluded their review of this literature with the opinion that it is incumbent upon the clinician to justify the use of a hospital stay of more than 60 days. We feel that such a blanket conclusion is premature, especially with respect to the kinds of patients not well represented in the available research, and the dependence of this conclusion on the adequacy of nonhospital services. We do agree, however, that the evidence suggests that most psychiatric patients who require hospital treatment can be effectively treated in a relatively short-term setting, so long as day treatment and supportive residential placement are readily available. With adequate outpatient resources, hospitalization can be avoided altogether for many patients who would formerly have been hospitalized (Reibel and Herz 1976; Riessman and Rabkin 1977).

Hospitalization is still indicated in a number of cases, as listed below:

1. Chronic patients sometimes require *reevaluation* of their diagnosis and functioning. Admission to a facility with access to medical technology; careful observation by nursing staff; and 24-hour psychiatric supervision may be indicated when the diagnosis is in question—e.g., organicity or affective illness need to be ruled in or out. In addition, the patient's functioning in the community may indicate he is not at the appropriate level of care (he should be in a more or less structured or supervised setting) or that he is not receiving the appropriate mix of treatment and care services. Finally, the chronically ill require periodic review and reeval-

uation of their treatment plans, and sometimes the inpatient setting is the most logical place in which to perform this task because it is more comprehensive, structured, and equipped with experts.

2. Chronic patients may not be on an optimal *psychopharmacological regimen* and may need readmission to reequilibrate their medication. Either too much medication (indicated by the occurrence of undesirable side effects or the inability to perform the social coping functions necessary to community survival) or too little (indicated by reemergence of psychotic symptoms, inability to function optimally, or behavior that is upsetting or disorganized) may necessitate readmission. Since the milieu affects the "optimal medication level," however, the milieu should be taken into account in assessing this level.

3. Certain chronic mental patients require readmission to effect *changes* in their *treatment plans*. For example, the psychiatrist may want to wash out an ineffective medication and begin a new one, and the hospital structure is invaluable in preventing (or managing) regression during the transition period. Or the person may need to move from one community support element to another, and require the structure of a secure setting for this shift.

4. Some patients require hospitalization because they *cannot be managed* optimally on an outpatient basis. For example, they may be unreliable in taking oral medications and require the initiation of an intramuscular medication regimen. Or they may become overtly psychotic, homocidal or suicidal, and present a danger to themselves or others.

5. The chronically ill may require *treatment that is not available* or easy to administer in community settings. While the administration of electroconvulsive therapy is unusual in chronic schizophrenia, there are definite indications for such treatment in other psychiatric disorders, and in these cases, inpatient admission may be required. In addition, structure may be required during certain periods—for instance, following the death of a close family member or spouse, when other treatment (e.g., increased medication) would not be helpful.

6. Some chronic patients require admission during *transference crises* in their outpatient treatment. At these times, readmission is essential to disentangle the patient from unnecessary complicating ties to the therapist. Conversely, admission may also be helpful when countertransference problems arise. At these times, the hospital serves as consultant to the patient and therapist.

7. *Detoxification* from alcohol or drugs may necessitate hospitalization to guard against the possibility of adverse reactions, e.g., seizures, coma, and death. While this is a little used indication, with the increasing prevalence of polydrug abuse in the "new or young chronic mentally ill" population, it may become more common.

8. Inpatient care can be critical to families and community support systems who cannot care for the chronic patient 365 days a year. Such *"respite care,"* although costly in the short run, has been found to be extremely useful to families of the mentally retarded. However, it is currently an underutilized, underoffered, and underreimbursed service for the mentally ill. Since too few residential facilities offer such "respite care," hospitals, *faute de mieux*, carry on this function.

In all these instances of hospital inpatient admission, it should be explicitly clear (optimally before hospitalization) *what the reasons* for rehospitalization are, *what goals* the admission is intended to achieve, and *what plans* are envisioned if the target goals are not reached. Admissions that constitute fishing expeditions or hope for magical solutions for "tough cases" are irresponsible and bound to fail.

We also wish to stress that there are specific *contraindications* to admission for this population. These include (Glick, Klar, and Braff 1984):

- Repeating a treatment that has already failed.
- Using the hospital because nothing else works.
- Attempting to make major characterological changes.
- Attempting to try a new medication, absent any positive indications for its success.
- Trying to convince patients to change their living situations,
- Treating the patients' unwillingness to follow treatment plans, and
- Sheltering malingerers or patients facing legal charges.

Treatment of Chronic Patients in the Hospital

Many treatment and care elements are provided in the modern inpatient hospital ward.

Evaluation/Assessment. Before specific treatment modalities are discussed, it is necessary to consider the evaluation and assessment of chronic illness. Primary to the establishment of an effective treatment program is the concurrence on a specific diagnosis. Since withdrawal, depression, and lack of spontaneity, for example, are common to chronic schizophrenia, chronic affective disorders, chronic institutionalization, and organic mental conditions, this is one series of differential diagnoses that must be considered from the start. The required interventions in such cases flow from the diagnosis: e.g., socialization for those suffering from institutionalization; appropriate medication for affective disorders; and elimination of organic factors, if present, for organicity (Talbott 1981).

In addition, many chronically mentally ill persons have received diagnoses, years before, in public institutions, and regardless of either current thinking about nomenclature or changes in presentation (e.g., affective symptoms), the diagnoses are reapplied thoughtlessly year after year. Also, Axis II or III diagnoses may be more "treatable" and thus provide the critical wedge in a seemingly unbudgeable psychotic condition.

Not only psychiatric assessment, but psychological evaluation, as well as functional assessment, should be conducted. The focus should be on an assessment of everyday functioning, social functioning, and vocational functioning, as well as an assessment of the variety of networks used or needed by the patient, instead of on a personality or psychodynamic profile.

Another critical factor frequently forgotten with the chronically ill is evaluation and *reevaluation* of what treatment and care has or has not worked in the past. This should include identification of the medication(s) to which the patient and his family members have responded in the past, since members of the same family often are responsive to the same medication.

Medication. There is no question that a primary inpatient treatment modality for the chronic patient is psychopharmacologic. Antipsychotic medication should be considered in most chronic patients exhibiting symptoms of psychosis. Medication is of great value in symptom suppression in the hospital, but it may impede community adaptation on discharge, and thus must be reduced at some point after discharge (Segal and Aviram 1981). Equally important, if the patient has no active symptoms and only residual symptomatology, discontinuance (or low doses) should be actively considered.

However, since we know, from the previously mentioned studies, that the two ingredients that prevent or forestall exacerbation of illness and readmission to hospital are medication and some form of talking therapy, i.e., counseling or psychotherapy, medication is critical to the ongoing treatment of this population. Adequate doses, e.g., those that achieve the optimal balance between symptom suppression and side effects, should be the goal in all chronic patients.

Psychotherapy. It appears that psychotherapy is not additive to medication among chronically ill inpatients but *is* critical in preventing rehospitalization among outpatients. Group therapy is more effective than individual therapy in outpatients (May 1975).

Because of both patient desires and professional prejudices (including the wish not to withhold any treatment that *may* be beneficial), individual psychotherapy remains common in the inpatient treatment of chronic schizophrenia. If conducted, however, it should be goal-directed, task-oriented, and combine supportive and clarifying elements (Lamb 1976; Talbott 1981).

Although group therapy is more effective than individual therapy with outpatients, there are no data about its superiority with inpatients. When used, group therapy also needs to be targeted to specific goals (e.g., socialization and reduction of anxiety). Along with any other treatment, it should never become a rote, automatic part of a hospital treatment cocktail.

Milieu treatment, as stated previously, is also helpful for the chronic patient, but only if highly structured, well-designed, and adequately monitored. There is currently substantial doubt whether a short-term inpatient unit can provide the sort of milieu therapy available in longer stay settings.

Family Treatment/Psychoeducation. Until relatively recently, the families of the chronically ill were seen primarily as noxious agents and important contributors to patients' illnesses. After almost 30 years of deinstitutionalization, we are beginning to see their potential contribution as therapeutic allies and are shifting from a psychoanalytic or interpersonal treatment model to a systems or psychoeducational one that stresses information about chronic illness, help in handling the behavior of the patient, and peer-support among families of chronic patients (Anderson et al. 1980; Anderson 1981; Boyd, McGill, and Falloon 1981; Falloon et al. 1981). Most of current psychoeducational approaches are based on the recognition that the identified patient with schizophrenia and the family are exquisitely sensitive to each other. This sensitivity, when expressed in a form of high expressed emotion (high criticism, hostility, and emotional overinvolvement), can lead to poor outcome (Goldstein 1981). These newer techniques mobilize strengths of the family and educate the family on what they don't know about the illness rather than what they do know or fantasize.

There are now four studies reporting positive results of family psychoeducational techniques in the management of chronic patients. All show positive results, although their details vary. Anderson (1981) gives her treatment in a clinic, while Falloon (Falloon et al. 1981) does interviews at home. Anderson does not include the patient, while Falloon does. What they have in common is an emphasis in crisis management and instruction in problem-solving skills.

What this body of work suggests is that there needs to be a major change in the delivery of inpatient treatment services with a new form of intervention with families of the chronically ill as a necessary, but not sufficient, component of the treatment package. The modern treatment of chronic mental illness in the hospital includes intensive involvement with families, to ensure their optimal contribution to the treatment of their relatives, and to reduce the stress of their experience dealing with them. This is equally true of those without interested or accessible families, in which case, social networks, as they exist, must be used.

Finally, it should be mentioned that the emergence of the National Alliance for the Mentally Ill (NAMI) offers mental health providers an enthusiastic organizational ally. Time is well spent speaking to local chapters and offering whatever assistance or information is requested to both groups and individuals (Hatfield 1981). The same usefulness and mutual need for cooperation applies to ex-patient groups.

Skills of Everyday Living. At the beginning of the deinstitutionalization movement, patients were discharged from public mental hospitals without adequate training in how to survive in modern society. Today, it is critical to know whether patients can truly survive outside a sheltering institution before they are discharged. Therefore, an adequate inpatient hospitalization both evaluates and trains patients in the skills of everyday living. These skills include self-care (hygiene, grooming, and dressing), transportation, banking, shopping, purchasing and preparing food, and washing and cleaning clothes.

Instruction in skills of everyday living can be carried out in a hospital, but both direct supervision of the patients' ability to perform the activities and actual observation of patients while negotiating community systems is essential. In addition, it should never be taken for granted that the chronic patient, once discharged, will continue to perform these activities without difficulty, and some form of followup, continuing assessment, and ongoing interest is critical.

Vocational Rehabilitation. (For further details on this and other topics, see Talbott 1980.) While provision of a full vocational rehabilitation program is not possible as part of a short-term hospitalization, both vocational evaluation and prevocational guidance are. After an adequate assessment of the level of functioning of the chronic patient is carried out, an assessment of the patient's ability to work in sheltered or competitive employment should be made. Sometimes merely brushing up on specific skills (e.g., typing) and/or assistance in generic work areas (e.g., interviewing, resumé preparation, and personal appearance) will facilitate transition either to sheltered or competitive work situations.

Socialization. Critical to the community retention of chronic patients is their ability to communicate, get along with others, and develop networks of support. Again, while short-term hospitalization does not allow for full development of socialization skills, it does provide the opportunity to target specific deficits and areas that need improvement, and begin remediation of those areas. Referral to ongoing socialization programs can then follow.

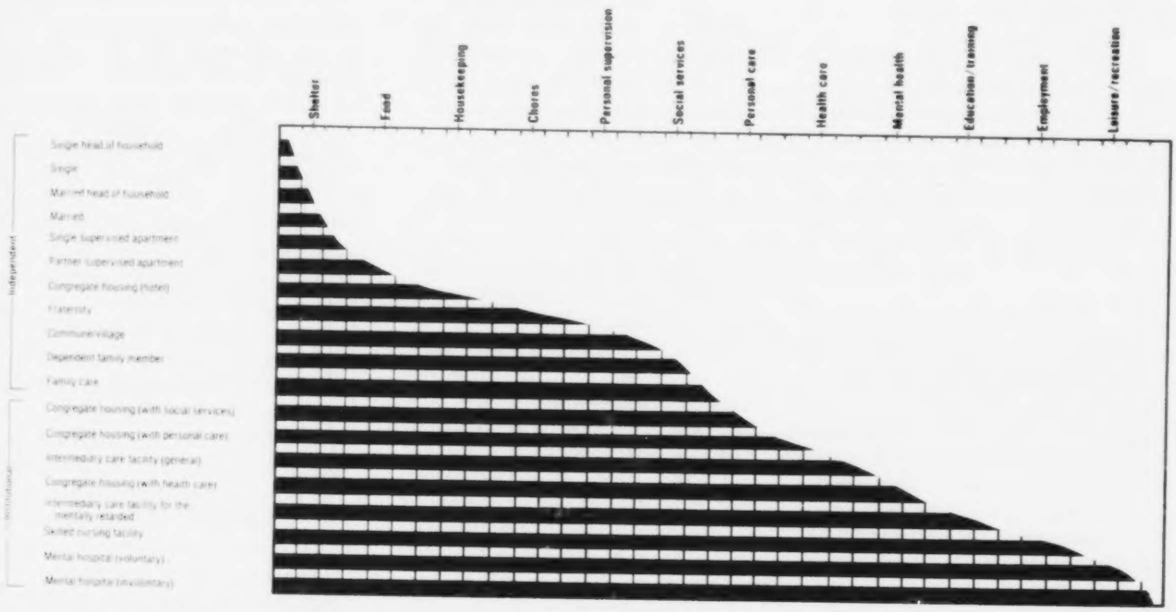
Discharge Planning

Planning for discharge from inpatient status is probably the most important process in the inpatient hospitalization of persons suffering from chronic illness. *It involves planning the management of the patient's illness over a lifetime rather than for the mere 21 or 30 days of hospitalization.* This shift in thinking has been difficult in psychiatry, and hospital staffs still persist in thinking of the hospital as central to the patient's treatment and care, instead of simply one small portion of it.

Needless to say, such planning should begin at admission, not near or at discharge. Discharge planning with the chronically ill must be based primarily upon an understanding of what functions need to be performed to enable the patient to return to community-based care (see figure 1). As patients move through graded levels of settings and services, one must bear in mind all the services that are required by them.

Psychiatric Care. One of the most critical aspects of discharge planning is the psychiatric care of chronic patients' posthospitalization. Two ingredients of care that forestall or prevent exacerbation of illness and rehospitalization are continued

Figure 1. Needs and settings for the chronically ill [1]



[1] Reprinted, with permission, from Talbott (1982).

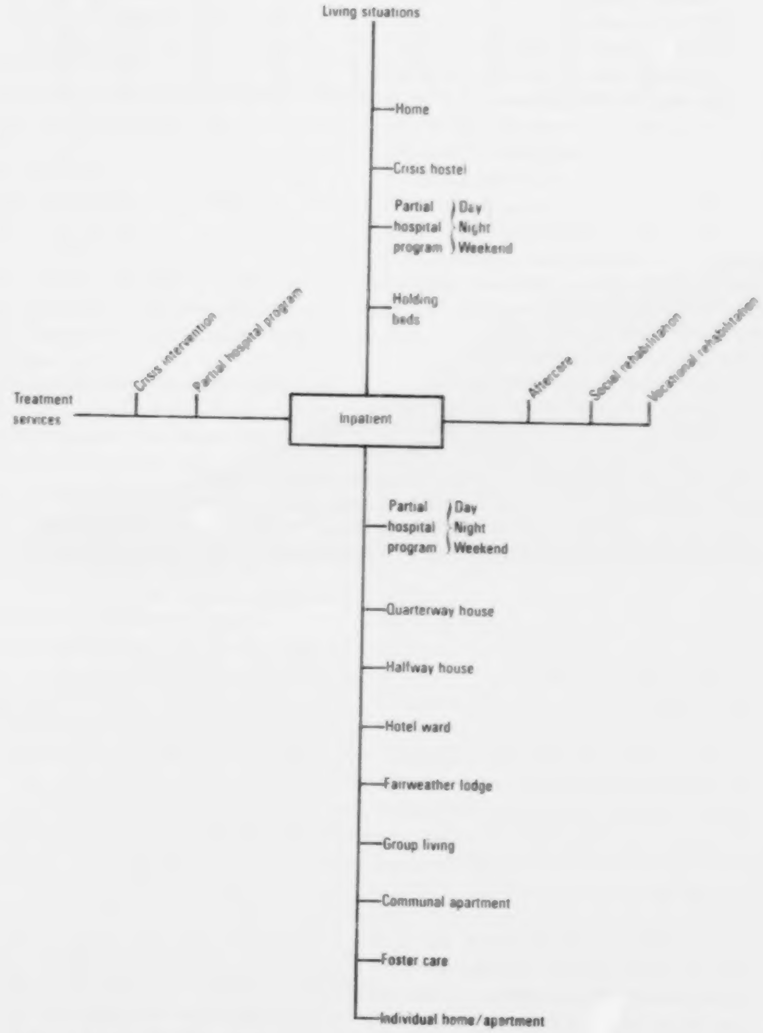
medication and psychotherapy or counseling, but these are carried out less than 50 percent and 25 percent of the time, respectively (Minkoff 1978). Provision of each is critical. In addition, provision for crisis intervention and crisis stabilization must be built into any plan. Otherwise, patients will merely spin around in the revolving door or bounce from community to hospital settings and back (Talbott 1974).

Medical Care. Persons suffering from chronic mental illness have three times the mortality and morbidity from chronic medical illnesses as their age-peers (Amdur 1981). Therefore, provision must be made for continuing medical care. This must involve ease of access to medical care, as well as sensitization of mental health personnel to medical issues, because the chronically ill have a seemingly high tolerance for pain and a low level of complaint (Talbott and Linn 1978). The former is difficult to achieve because general physicians often see psychiatric patients as having only mental illness, neglecting concurrent medical illness.

Housing. Figure 2 demonstrates the wide range of housing opportunities needed by the chronic patient population. Such a spectrum is rare in any American community, but does provide the optimal range. The guiding principle of placement should be to find the setting that is optimally therapeutic—rather than least restrictive—as it has been shown that the two are not identical (Bachrach 1980). Here, as in other parts of discharge planning, patient collaboration is key. One must always balance the needs of the patient against the services provided by each setting (figure 1), choosing the best match. In addition, the concept of allowing patients the opportunity to progress, but not at the expense of being challenged unrealistically, must be borne in mind.

Socialization and Social Rehabilitation. Provision must also be made for some form of socialization or social rehabilitation for most chronic patients. Whether this is in a structured (e.g., day hospital) or unstructured (e.g., lounge program) setting depends to a large degree on patients' functional levels and personal desires. An ideal way of conceptualizing the social needs of chronic patients is offered by Cutler and

Figure 2. Spectrum of optimal treatment services and living situations [1]


Living situations
Home
Crisis hostel
Partial hospital program
Day
Night
Weekend
Holding beds
Treatment services
Crisis intervention
Partial hospital program
Inpatient
Aftercare
Social rehabilitation
Vocational rehabilitation
Partial hospital program
Day
Night
Weekend
Quarterway house
Halfway house
Hotel ward
Fairweather lodge
Group living
Communal apartment
Foster care
Individual home/apartment

[1] Reprinted, with permission, from Talbott (1982).

Tatum (1983) who propose five different kinds of social networks, each of which should be considered in planning the posthospitalization life of chronic patients.

Vocational Rehabilitation and Work. Some constructive activity must be planned for most persons leaving the hospital. This includes vocational rehabilitation, sheltered workshops (both transitional and terminal), and competitive employment (often part-time, which chronic patients can handle more easily than full-time, at least at the start). Constructive activity is important to self-esteem, and is therefore most critical to eventual recovery.

Income. If competitive employment does not produce sufficient income for economic self-support, provision must be made for continuing economic support through home relief, Supplemental Security Income, or Social Security Disability Income. Currently, governmental policy discourages graded assumption of employment, but it is to be hoped that this antitherapeutic practice will eventually be eliminated.

Continuity of Care. As is obvious from the above, in moving from the single institutional umbrella under which all services were provided (e.g., the public mental hospital) to community-based care, we have forced patients into the position of "scrounging" for services. Continuity of care has many dimensions, but we are concerned here primarily with two—vertical continuity (pulling together all services needed at one time) and longitudinal continuity (pulling together all services needed over time). To ensure the provision of both these types of continuity of care, some person and agency must be designated as the glue that holds services together and enables the patient to have access to them. The concept of the resource or case manager is critical to continuing community survival of chronic patients (Lamb 1976; Lourie 1978). It is important to emphasize that the personal relationship between patient and case manager or case management team is critical.

Community Programs. Optimally, all the sorts of services required by the discharged chronic patient will be offered as part of one community program. Examples of such programs are those offered by psychosocial rehabilitation centers such as Fountain House and its over one hundred similar models (Glasscote 1978); the Madison, Wisconsin, Training in Community Living Program (Stein and Test 1977); and the Southwest Denver Program (Stein and Test 1975). The recent initiative by the National Institute of Mental Health to establish Community Support Programs (CSPs) throughout the country is another model; while inadequately funded to date, it offers additional promise of providing the glue with which needed services can be stuck together. The growing number of psychosocial rehabilitation programs across the country is an important and critical development in the move to genuine "community care" of the chronically ill.

Long-Term Inpatient Treatment of Chronic Mental Illness

There is little evidence that long-term (e.g., over 30 days) inpatient treatment of the chronic patient is superior to shorter treatment, but it may be indicated in special instances (Group for the Advancement of Psychiatry 1978).

Indications. The primary indication for long-term inpatient hospitalization of chronic patients is *diagnostic*, e.g., to determine what aspect of the patient's personality or family interactions plays a strong role in his current presentation. For example, despite the fact that a patient exhibits all the signs and symptoms of chronic mental illness, the predominant issue in his life may be a *characterological* one, e.g., passive-aggressive behavior or paranoid ideation. In these cases, it may be beneficial to use the structure and milieu of a long-term setting to ameliorate the patient's condition.

Another indication is *behavioral*, i.e., when the patient's behavior cannot be controlled or modified in outpatient settings. One example is chronic antisocial or incompetent behavior that either endangers the patient or others. In these cases, longer treatment, usually in a highly structured behavioral modification or social learning program, may be warranted.

Treatment of a sort not available in outpatient settings is another indication. Here, treatment, whether psychotherapeutic, somatic, or behavioral, is sought within the structure of and opportunity for supervision available in inpatient settings. Sometimes, when long-term psychotherapeutic hospitalization has not been attempted in a patient suffering from chronic illness, exposure to this modality of treatment may be warranted.

Rehabilitation of or social learning in severely impaired chronic patients is another indication for long-term hospitalization. For instance, persons who have been institutionalized for many years have severe ego deficits, or cannot function at even a minimal level because of their mental disability may require a long-term structured experience like that described by Paul and Lentz (1977), before progressing to another facility or setting (even a halfway house or group home).

Finally, some persons are hospitalized for long periods because they *cannot be treated* and their symptoms *cannot be controlled* in outpatient programs. Examples are those with resistant or unremitting symptoms, usually psychotic. Here, however, one must ask whether the hospital is being used for treatment (e.g., to provide a structure in which medication and psychotherapy can effectively control symptoms) or merely for custody (e.g., because the person cannot survive outside a structured, supervised setting). In the latter case, hospitalization may not be warranted, and care could equally well be provided in a locked facility in the community, an adult home, or foster care).

Advantages of Long-Term Hospitalization. Long-term hospitalization does offer several advantages over treatment in alternative settings. These include structure, a therapeutic milieu, 24-hour supervision, use of special medications or combinations of medications, 24-hour behavioral modification or social learning, and availability of all services under one roof (e.g., housing, therapy, vocational therapy, and social rehabilitation).

Disadvantages of Long-Term Hospitalization. There are serious reservations about long-term inpatient treatment of chronic patients. These include the following: induction into a role of "institutionalized" patient, overreliance on the hospital to solve social (e.g., housing) problems, suppression of the development of coping skills, and high cost.

In conclusion, whereas a few decades ago, long-term inpatient treatment was the preferred (if not the only) treatment for chronic mental illness, current thinking holds that such treatment should be reserved for a very few specific cases. Short-term inpatient treatment should be the rule for the majority of chronic patients, as opposed to the acutely ill, for whom a trial of long-term inpatient treatment is warranted (Glick and Hargreaves 1979).

The Need for Asylum

Radical shifts in the care of mentally ill patients have taken place in the 300 years of American history. Until the 1950s, hospitals were the locus of treatment and care; but subsequently, especially during the late 1960s, some theorists maintained that there was no need for *any* hospitals for the mentally ill. Only recently has it been realized that there is a need for asylum for some small percentage of our chronic patient population.

By asylum, we do not mean a building or institution, but rather a function. Certainly, in California, it has been demonstrated that both board and care homes and "L-facilities" have performed the function of asylum, custody, or supervised, sheltered care. Such asylum functions should have State funding, but not be operated by the State. In fact, because of their tertiary role in the mental health system, they are probably best operated by local governmental units and located in the community.

While the percentage or number of such beds is unknown, largely because no system has ever been funded sufficiently and equipped with the entire range of alternative settings, several estimates are available (Stein and Test 1975). Wing estimates the number at 15-24 per 100,000, with an additional 300 per 100,000 for the elderly infirm; Beeson and Ford at 18 per 100,000; and the California Model at 40 per 100,000 (Elpers and Crowell 1982).

Consensus, then, among the estimates is in the 15-40 per 100,000 range (1.5-4 percent).

The Future

The future of inpatient hospital treatment for chronic patients depends in large part on two factors: reimbursement and scientific research. With the beginning understanding of differential therapeutics (Frances, Clarkin, and Perry 1984), it is becoming increasingly obvious that the practices of both professionals and third parties will be governed more and more by answers to the question, "what treatment works for which patients in what settings?" The current fad of lumping medical diagnoses into large groupings (diagnosis-related groups) is inapplicable to chronic mental illness, and more sophisticated methods of delineating different subgroups must be used.

It is clear that progress has been made in the care and treatment of chronic illness, but there is no cure in sight for the basic disease or diseases. As has often been pointed out, scientific advances take place in spurts, and just as no one could have predicted the current advances in treating many formerly devastating cancers, no one can predict which mental illnesses will come under control next or when.

However, waiting around for the millennium is an inadequate response to the current problem. At present, we are not implementing what little we know about the treatment of chronic mental illness (Talbott 1979; Frances, Clarkin, and Perry 1984); e.g., continuity of care, use of short-term hospitalization, combination of psychosocial and psychopharmacological interventions, necessity of seeing a hospital episode as just one part in the patient's lifetime vulnerability to illness, and need to forge a true mental health system. While we await the "cure" for those suffering from chronic illnesses, we have plenty of work to attend to in simply bringing about their "care."

References

Amdur, M.A. Death in aftercare. *Comprehensive Psychiatry,* 22:619-626, 1981.

Anderson, C.M. The psychoeducational family treatment of schizophrenia. *New Directions in Mental Health Services,* 12:79-94, 1981.

Anderson, C.M.; Hogarty, G.E.; and Reiss, D.J. Family treatment of adult schizophrenic patients: A psychoeducational approach. *Schizophrenia Bulletin,* 6:490-505, 1980.

Bachrach, L.L. Is the least restrictive environment always the best? Sociological and semantic implications. *Hospital & Community Psychiatry,* 31:7-102, 1980.

Boyd, J.L.; McGill, C.W.; and Falloon, I.R.H. Family participation in the community rehabilitation of schizophrenics. *Hospital & Community Psychiatry,* 32:629-632, 1981.

Braun, P.; Kochansky, G.; Shapiro, B.; Greenberg, S.; Gudeman, J.E.; Johnson, S.; and Shore, M.F. Overview: Deinstitutionalization of psychiatric patients—A critical review of outcome in American studies. *American Journal of Psychiatry,* 138:736-749, 1981.

Cutler, D.L., and Tatum, E. Networks and the chronic patient. In: Cutler, D.L., ed. "Effective Aftercare for the '80's." *New Directions in Mental Health Services,* 19:13-22, 1983.

Dain, N. From Colonial America to Bicentennial America: Two centuries of vicissitudes in the institutional care of mental patients. *Bulletin of the New York Academy of Medicine,* 52:1179-1196, 1976.

Davis, J.M. Overview: Maintenance therapy in psychiatry—I. Schizophrenia. *American Journal of Psychiatry,* 132:1237-1245, 1975.

Deutsch, A. *The Mentally Ill in America: A History of Their Care and Treatment from Colonial Times.*

2nd ed. New York: Columbia University Press, 1949.

Elpers, J., and Crowell, A. How many beds? An overview of resource planning. *Hospital & Community Psychiatry*, 33:755–761, 1982.

Falloon, R.H.; Boyd, J.L.; McGill, C.W.; Strang, J.S.; and Moss, H.B. Family management training in the community care of schizophrenia. *New Directions in Mental Health Services*, 12:61–77, 1981.

Frances, A.; Clarkin, J.; and Perry, S. *Differential Therapeutics in Psychiatry: The Art and Service of Treatment Evaluation*. New York: Brunner-Mazel, 1984.

Glasscote, R. What programs work, and what programs don't work to meet the needs of the chronic mental patients? In: Talbott, J.A., ed. *The Chronic Mental Patient: Problems, Solutions and Recommendations for a Public Policy*. Washington, DC: American Psychiatric Association, 1978. pp. 75–85.

Glick, I.D., and Hargreaves, W.A. *Psychiatric Hospital Treatment for the 1980's*. Lexington, MA: Lexington Books, 1979.

Glick, I.D.; Klar, H.; and Braff, D. When should chronic patients be hospitalized? *Hospital & Community Psychiatry*, 35:934–936, 1984.

Goldstein, M.J. Short and long-term effects of combining drug and family therapy. *New Directions in Mental Health Services*, 12:5–26, 1981.

Group for the Advancement of Psychiatry. *The Chronic Mental Patient in the Community*. New York: GAP, 1978.

Hatfield, A.B. Families as advocates for the mentally ill: A growing movement. *Hospital & Community Psychiatry*, 32:641–642, 1981.

Herz, M.I.; Endicott, J.; and Spitzer, R.L. Brief vs. standard hospitalization: The families. *American Journal of Psychiatry*, 133:795–801, 1976.

Herz, M.I.; Endicott, J.; and Spitzer, R.L. Brief hospitalization: A two-year follow-up. *American Journal of Psychiatry*, 134:502–507, 1977.

Kiesler, C.A. Mental hospitals and alternative care: Noninstitutionalization as potential public policy for mental patients. *American Psychologist*, 37:349–360, 1982.

Kramer, M. *Psychiatric Services and the Changing Institutional Scene*. Bethesda, MD: National Institute of Mental Health, 1975.

Lamb, H.R.N. *Community Survival for Long-Term Patients*. San Francisco: Jossey-Bass, 1976.

Lourie, N.V. Case management. In: Talbott, J.A., ed. *The Chronic Mental Patient: Problems, Solutions and Recommendations for a Public Policy*. Washington, DC: American Psychiatric Association, 1978. pp. 159–164.

Mattes, J.A.; Klein, D.F.; and Millian, D. Comparison of the clinical effectiveness of "short" vs. "long" stay psychiatric hospitalization: II. Further results of a 3-year posthospital follow-up. *Journal of Nervous and Mental Disease*, 165:395–402, 1977.

Mattes, J.A.; Rosen, B.; and Klein, D.F. Comparison of the clinical effectiveness of "short" vs. " long" stay psychiatric hospitalization: II. Results of a 3-year posthospital follow-up. *Journal of Nervous and Mental Disease*, 165:387-394, 1977.

May, P.R.A., and Simpson, G.M. Schizophrenia: Overview of treatment methods. In: Freedman, A.M.; Kaplan, H.I.; and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry/II*. Baltimore: The Williams & Wilkins Company, 1975. pp. 923–938.

Minkoff, K.L. A map of the chronic mental patient. In: Talbott, J.A., ed. *The Chronic Mental Patient: Problems, Solutions and Recommendations for a Public Policy.* Washington, DC: American Psychiatric Association, 1978. pp. 11–37.

Mosher, L.R., and Gunderson, J.G. Group, family, milieu, and community support systems treatment for schizophrenia. In: Bellak, L., ed. *Disorders of the Schizophrenic Syndrome*. New York: Basic Books, 1979. pp. 399–452.

Paul, G.L., and Lentz, R.J. *Psychosocial Treatment of Chronic Mental Patients: Milieu Versus Social-Learning Programs*. Cambridge, MA: Harvard University Press, 1977.

Reibel, S., and Herz, M.I. Limitations of brief hospital treatment. *American Journal of Psychiatry*, 133:518–521, 1976.

Riessman, C.K., and Rabkin, J.G. "Hospital vs. Home Care Programs for Psychiatric Patients: Summary of Published Findings." Unpublished manuscript, 1977.

Riessman, C.K.; Rabkin, J.G.; and Struening, E.L. Brief versus standard psychiatric hospitalization: A critical review of the literature. *Community Mental Health Journal*, 2:1–10, 1977.

Rothman, D.J. *The Discovery of the Asylum: Social Order and Disorder in the New Republic*. Boston: Little Brown, 1971.

Segal, S.P., and Aviram, U. *The Mentally Ill in Community-Based Sheltered Care*. New York: Wiley, 1981.

Stein, L.I., and Test, M.A. *Alternatives to Mental Hospital Treatment*. New York: Plenum Press, 1975.

Stein, L.I., and Test, M.A. Use of

special living arrangements: A model for decision-making. *Hospital & Community Psychiatry*, 28:608-610, 1977.

Talbott, J.A. Stopping the revolving door. *Psychiatric Quarterly*, 48:159-168, 1974.

Talbott, J.A. *Toward a Public Policy on the Chronically Mentally Ill.* Washington, DC: American Psychiatric Association, 1979.

Talbott, J.A., ed. *The Chronic Mentally Ill: Treatment, Programs, Systems.* New York: Human Sciences Press, 1980.

Talbott, J.A. Therapy of the chronic mentally ill. In: Masserman, J.H., ed. *Current Psychiatric Therapies.* New York: Grune & Stratton, 1981. pp. 347-355.

Talbott, J.A. Trends in the development of psychiatric services. In: Talbott, J.A., and Kaplan, S.R., eds. *Psychiatric Administration: A Comprehensive Text for the Clinician-Executive.* New York: Grune & Stratton, Inc., 1982. pp. 7-14.

Talbott, J.A. Twentieth century developments in American community psychiatry. *Psychiatric Quarterly*, 54:207-219, 1983.

Talbott, J.A., and Linn, L. Reactions of chronic schizophrenics to life-threatening disease. *Psychiatric Quarterly*, 50:218-227, 1978.

Weisbrod, B.A.; Test, M.A.; and Stein, L. An alternative to mental hospital treatment: III. Economic benefit-cost analysis. *Archives of General Psychiatry*, 37:400-405, 1980.

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Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work, in cooperation with several community and mental health organizations, has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside."

For more information about the rental or purchase of these videotapes, please contact: **Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.**

Bibliography and Abstracts on Schizophrenia

The abstracts that appear below are drawn from the computer-based information storage and retrieval system operated by the National Clearinghouse for Mental Health Information. They were selected for inclusion here on the basis of their direct relevance to schizophrenia: judgments about the quality of the article or book abstracted have been left to the reader. Foreign titles have been translated and the city of the journal's origin noted. Languages that use neither the Latin nor Fraktur (German) alphabets (e.g., the Slavic languages) have been transliterated. Titles of articles, books, and infrequently cited journals have been translated from these languages, and all other identifying information appears in English in their citations.

The articles described in these abstracts are not available from either the Clearinghouse or the Schizophrenia Research Branch.

Attention, Perception, Cognition

6485. Broga, M.I. (University of Western Ontario, London, Ontario, Canada) An information processing approach to the study of schizophrenic memory and thought. Dissertation Abstracts International, 42(6):2517-B, 1981.

Deficits in memory and thought processes among samples of paranoid and nonparanoid schizophrenic subjects were investigated. On the tachistoscope letter detection task, all groups appeared to be able to filter out irrelevant stimuli and to focus on designated information when attention was directed by a physical cue; however, neither the paranoid schizophrenic (PS) nor the nonparanoid schizophrenic (NPS) groups showed a decay function associated with a temporal loss of information from the iconic store. On the Posner letter-matching task, all three groups showed comparable encoding strategies, although the PS and NPS groups were slower in performing these operations than the normal group. Results are also reported from the Peterson-Peterson task, the Sternberg task, an overt rehearsal task, and free recall of categorized words. Seventeen measures from the battery were also analyzed by multiple discriminant function analysis and two significant dimensions extracted. Data from the 17 information-processing measures were also subject to modal profile analysis. Five underlying modal types of information processors are identified. The nature of the empirically formed subgroups which loaded on each distinct modal profile are investigated, and the capabilities and limitations in processing of each type are presented.—Journal abstract, modified.

6486. Callahan, D.F. (No address given) Laterality, schizophrenia and the guided imagery technique. Dissertation Abstracts International, 42(8):3410-B, 1982.

The differential functioning of the right and left hemispheres and the effect of guided imagery on laterality were investigated in 24 schizophrenic outpatients tested on a dichotic listening task and assigned to guided imagery or traditional therapy conditions. The hypothesis that guided imagery subjects would make more responses based on emotional than verbal cues on the listening task at posttest was not substantiated. Both groups made significantly more content/verbal, or left hemisphere, responses than tone/emotional responses from pre- to posttesting, and the control group made more content than tone responses from pre- to posttesting. Both groups showed improvement in symptomatology, but did not differ significantly from each other. Both groups also showed improvement at 10-month followup. No sex differences were found on the dichotic listening task.—Journal abstract, modified.

6487. Cook, M., and Simukonda, F. (Dept. of Psychology, University College of Swansea, Singleton Park, Swansea SA2 8PP, Wales) Anhedonia and schizophrenia. British Journal of Psychiatry, 139 (December):523–525, 1981.

The relationship between anhedonia and symptoms of apathy and withdrawal in schizophrenia was studied. Subjects were 52 schizophrenic inpatients and 102 hospital staff controls, who were administered Chapman's anhedonia and perceptual aberration scales. Differences between the two groups were found for both the physical and social anhedonia scales and for the perceptual aberration scale, but the difference for physical anhedonia was only marginally significant. It is concluded that the anhedonia scales do not differentiate between the two groups sufficiently well to be a useful diagnostic tool. (11 references)

6488. Crider, A.; Solomon, P.; and McMahon, M.A. (Dept. of Psychology, Williams College, Williamstown, MA 01267) Disruption of selective attention in the rat following chronic d-amphetamine administration: Relationship to schizophrenic attention disorder. Biological Psychiatry, 17(3):351–361, 1982.

Kamin's (1968) blocking paradigm was used to examine further an animal model of schizophrenic attention disorder, using chronic *d*-amphetamine administration in the rat. In the blocking paradigm, prior training to one conditioned stimulus (CSA) blocks the ability to attend to a second conditioned stimulus (CSB) when the two form a compound (CSAB) in subsequent training. Experiment 1 shows that *d*-amphetamine disrupted rats' ability to ignore the irrelevant (CSB): the animals responded equally to both elements of the CSAB compound following five daily administrations of 4 mg/kg *d*-amphetamine. In experiment 2, the disruption of blocking by *d*-amphetamine was eliminated by a concomitant administration of .02 mg/kg haloperidol. These results are consistent with previous research showing that *d*-amphetamine disrupts rats' ability to ignore repeated presentations of a single nonreinforced stimulus in the latent inhibition paradigm. The inability of anphetamine-treated animals to ignore one element of a dual element compound bears some resemblance to selective attention deficits observed among schizophrenic patients. (38 references)—Author abstract, modified.

6489. Faber, R., and Reichstein, M.B. (Audie Murphy Veterans Hospital, San Antonio, TX 78284) Language dysfunction in schizophrenia. British Journal of Psychiatry, 139 (December):519–522, 1981.

Similarities between language disorders in aphasia and formal thought disorder in schizophrenia were explored in 24 schizophrenic, 5 manic, and 5 depressed psychiatric inpatients and 28 normal controls. Eight subtests from the Boston Diagnostic Aphasia Examination, a picture-naming test, and the Token Test were administered. Schizophrenic subjects with formal thought disorder showed significant abnormalities compared to all other groups, particularly on the Token Test and the repetition of phrases test. These deficits are suggestive of language comprehension and repetition dysfunctions in a substantial minority of rigorously defined schizophrenic subjects. (19 references)—Author abstract.

6490. Frumkin, I.B. (No address given) Information processing in schizophrenia: An attentional dysfunction. Dissertation Abstracts International, 42(8):3419–B, 1982.

Information processing of dichotically presented lists of digit pairs was investigated in 10 nonparanoid schizophrenics, 10 affective disorder patients, and 10 normals. Digit lists were presented orally under a fast or slow presentation rate. Under successive report, subjects were to report the first three digits spoken in one ear, and then to report the digits heard in the other ear. Under alternating report, subjects were instructed to report digits in an order approximating order of receipt. Recall performance was calculated by counting the total number of relevant digits correctly reproduced in each list, taking order and position into account. Results show significant main effects for diagnosis, presentation rate, and recall strategy. A significant three-way interaction of these factors supports Yates' (1966) account of schizophrenic attention dysfunction as a result of a slowness in switching back and forth between acoustic or contextual channels. All subjects performed best under successive recall at the fast presentation rate. Only the schizophrenic patients had better performance at the slow rate with the successive, as opposed to the alternate, recall condition.—Journal abstract, modified.

6491. Johnson, O., and Crockett, D. (2605 W. 37th Ave., Vancouver, British Columbia, Canada V6N 2T3) Changes in perceptual asymmetries with clinical improvement of depression and schizophrenia. Journal of Abnormal Psychology, 91(1):45–54, 1982.

The hypothesis of altered lateral asymmetries with clinical improvement in depression and schizophrenia was investigated with two dichotic listening tests and two cognitive tests. Tests were administered at intervals ranging from initial hospitalization to recovery. Repeated measures analysis of variance found that severely depressed patients failed to obtain normal ear superiority in either dichotic test, but normal superiorities emerged with recovery from depression. Schizophrenic subjects initially failed to show normal right ear superiority on a dichotic words test but did obtain the expected left ear advantage on a dichotic chords test. Following treatment, schizophrenics shifted from a left ear advantage to a right ear advantage in dichotic chords and also increased their right ear advantage in dichotic words. Both patient groups showed normal word fluency but impaired spatial ability, which did not improve with recovery. Results are interpreted as suggesting that both depression and schizophrenia are associated with a breakdown in the processes of interhemispheric inhibition that mediate perceptual asymmetry. In depression, the effect of treatment is a return to normal patterns of asymmetry, whereas in schizophrenia the result of treatment is an abnormal pattern of asymmetry that may reflect the allocation of both verbal and nonverbal material to the left hemisphere. (42 references)—Author abstract, modified.

6492. Kay, S.R. (Dept. of Psychology, Bronx Psychiatric Center, 1500 Waters Place, Bronx, NY 10461) Conceptual disorder in schizophrenia as a function of encoding orientation. Journal of Nervous and Mental Disease, 170(3):154–163, 1982.

The hypothesis that schizophrenic conceptual disorder derives from abnormal verbal encoding, geared to salient affective and physical cues at the expense of conceptual attributes, was evaluated. Subjects were 63 adult psychotic inpatients, including 42 schizophrenics and 21 nonschizophrenics of similar age, sex, race, and chronicity. They underwent 12 trials on Wickens' release from proactive interference (PI). The release effect for schizophrenics was significantly weaker on the conceptual dimension and stronger on the affective dimension. Schizophrenic subjects contrasted controls by their diminished PI release with less salient concepts. The results suggest that encoding by schizophrenics is uniquely oriented to stimulus salience rather than semantic relevance. (66 references)—Author abstract, modified.

6493. Koukkou, M. (Research Dept., Psychiatric University Hospital, P.O. Box 68, CH-8029 Zurich 8, Switzerland) EEG states of the brain, information processing, and schizophrenic primary symptoms. Psychiatry Research, 6(2):235–244, 1982.

Brain states underlying information processing in 22 acute and recovered medication-free schizophrenic subjects and 20 matched controls were examined using electroencephalographic (EEG) reactivity to auditory information, the central component of the orienting reaction. EEG reactivity was assessed as information-induced changes of parameters extracted from power spectral frequency analysis of the ongoing EEG. Results indicate that EEG reactivity in controls and schizophrenics in remission was largely similar. Acute schizophrenics show deviant ectropic reactivity, which consists of changes within the 2–8 Hz α band. These results indicate an ectropic brain state during cognitive processing of received information during the period of acute schizophrenic symptomatology. (33 references)—Author abstract, modified.

6494. Kukla, F. (Frankfurter Allee 272, DDR-1130 Berlin, German Democratic Republic) Disturbed information processing of schizophrenics in the recognition of semantic relationships between concepts. Zeitschrift für Psychologie mit Zeitschrift für Angewandte Psychologie, 189(3):269–288, 1981.

Information processing in recognizing semantic relationships was studied in two groups of 16 subjects each. One group was normal; the other was composed of schizophrenics who had not yet begun therapy. The two groups were matched on the basis of premorbid and actual intelligence, age, education, and sex. Subjects were required to solve verbal analogy problems for 12 different concepts to test ability to define, classify, and use discrimination with these concepts. The schizophrenic subjects demonstrated that activation of concept features from long-term memory in schizophrenia is influenced more by the number of features to be compared between two concepts than by task specificity and context. This indicates that not only long-term memory (meaning of concepts often forgotten), but also short-term memory (forgetting features during concept comparison) is impaired in schizophrenic subjects. (59 references)

6495. Matsue, Y., and Okuma, T. (Dept. of Psychiatry, Tohoku University School of Medicine, Sendai, Japan) Flickering light spot as a tracking target for the study of smooth pursuit eye movements in schizophrenics—A new method. Folia Psychiatrica et Neurologica Japonica, 35(4):437–445, 1981.

Smooth pursuit eye movements (SPEM) were studied in 26 schizophrenics and 20 normal controls. A light spot on the screen of a cathode ray oscilloscope was used as a tracking target. In order to induce SPEM disorder easily, the subjects were required to track a light spot which flickered in various frequencies. The new method devised has clearly demonstrated that the SPEM in schizophrenics was more profoundly disturbed by the flickering of the tracking target than that in a normal control. It is suggested that poor tracking of a flickering light spot in schizophrenics may be due to the inadequate concentration of attention to visual stimuli and/or inability to predict the movements of a tracking target. (9 references)—Author abstract, modified.

6496. Moyaert, P. (Universitaire Faculteit Sint Ignatius, Antwerp, Belgium) Language, body, and affect in schizophrenia. Tijdschrift voor Psychiatrie, 24(1):49–69, 1982.

Schizophrenia is described in relation to the structure of language (word and thing presentations) and the vicissitudes of the instinct (affect), in accordance with the metapsychological reflections of Freud and Vergote. The schizophrenic's dialectic between the preconscious word presentations and the unconscious thing presentations is explained, and the vicissitudes of the affect are further defined. (11 references)—Journal abstract, modified.

6497. Portnoff, L.A.; Yesavage, J.A.; and Acker, M.B. (Neuropsychology Laboratory, Eastern State Hospital, Vinita, OK 74301) Visual search performance by paranoid and chronic undifferentiated schizophrenics. Perceptual and Motor Skills, 53(2):411–418, 1981.

Recent observations that some schizophrenics have difficulty with visual tracking and that this cognitive abnormality is suggestive of attentional deficits were investigated. Visual search performance was assessed in 15 chronic undifferentiated, 15 paranoid schizophrenics, and 15 normal controls on two tests which measure search in a systematic and an unsystematic stimulus mode. Chronic schizophrenics showed difficulty with both kinds of search tasks. Paranoids had only a deficit in the systematic task, and their ability for visual search in the unsystematized stimulus array was equivalent to that of normals. Although replication and cross-validation is needed to confirm these findings, it is concluded that the two tests of visual information processing may provide a useful ancillary method for differential diagnosis between these two types of schizophrenia. (27 references)—Author abstract, modified.

6498. Saccuzzo, D.P.; Braff, D.L.; and Sprock, J. (Psychology Clinic, San Diego State University, San Diego, CA 92182) The effect of mental retardation and schizophrenia on information processing. Journal of Nervous and Mental Disease, 170(2):102–106, 1982.

A forced-choice letter discrimination task was used to examine the relationship between schizophrenia and retardation from the standpoint of information-processing theory. The subjects consisted of eight schizophrenic persons of average or better intelligence, eight mentally retarded schizophrenic persons, and eight minor depressive individuals who were matched with the nonretarded schizophrenic group for intelligence. The groups did not differ significantly on the minimum exposure duration needed to identify an unmasked target stimulus at criterion levels of accuracy. When masked stimuli were used, however, the depressive group obtained significantly more correct detections than did both schizophrenic groups. More importantly, the performance of the two schizophrenic groups did not differ significantly. Data indicate that schizophrenic deficits in information processing are independent of intellectual factors. (31 references)—Author abstract, modified.

6499. Sandhu, J.S. (Punjab Mental Hospital, Amritsar, Punjab, India) Relationship between attentional capacity and abstract thinking in schizophrenics. Indian Journal of Clinical Psychology, 8(2):85–94, 1981.

Abstract thinking deficit in schizophrenic patients was investigated in a study of the attentional capacity of chronic schizophrenic patients. Two groups of 23 chronic nonparanoid schizophrenic patients separated at two levels of abstraction and 23 normal controls matched for various demographic variables were compared for their performance on four tasks of attention and an abstraction task. Input dysfunction was confirmed as a major source of abstract thinking deficit in schizophrenics, but results do not support the same conclusion for normal subjects. Results suggest that attentional dysfunction in schizophrenics may determine the nature of errors made in the process of abstraction. It is concluded that different measures of attention correspond with each other differently for normal and schizophrenic subjects, suggesting that primary mental functioning of normal subjects may be radically different from primary mental functioning of schizophrenic patients. (39 references)—Author abstract, modified.

6500. Stanikowska, I. (I Klinika Psychiatryczna, Instytut Psychoneurologiczny, Al. Sobieskiego 1/9, 02-957 Warszawa, Poland) The diagnostic and prognostic significance of disorders of conceptual thinking in schizophrenic patients. Psychiatria Polska, 15(3):279-284, 1981.

Studies of 202 patients were conducted to determine if the phenomenon of overinclusion is specific to schizophrenia and if it is related to clinical pattern, time of duration, and prognosis. Reed's modification of Epstein's test of concept subordination and Payne's test of solid classification were studied in groups of acute schizophrenics (35), chronic schizophrenics in the period of exacerbation (33), chronic schizophrenics in the period of remission (34), depressives (40), and normal subjects (60). The ability to form and use abstract concepts was found undiminished in schizophrenic patients; however, the hypothesis that their thinking involves overinclusion has been verified. It has been found that the most characteristic symptoms of schizophrenia are unusual responses and the frequency of their occurrence differentiates schizophrenics from normal subjects and from depressive patients. (20 references)—Journal abstract, modified.

6501. Stuss, D.T.; Benson, D.F.; Kaplan, E.F.; Weir, W.S.; and Della Malva, C. (Dept. of Medicine (Neurology), Ottawa General Hospital, 501 Smyth Road, Ottawa, Ontario, K1H 8L6, Canada) Leucotomized and nonleucotomized schizophrenics: Comparison on tests of attention. Biological Psychiatry, 16(11):1085-1100, 1981.

Deficits in attention have been strongly linked with both schizophrenia and pathology in the prefrontal cortex. This observation was tested by administering a battery of commonly used tests of attention, sustained mental activity, and tracking to 16 patients who had undergone prefrontal leucotomy approximately 25 years earlier. Presurgical diagnosis in each patient was schizophrenia. The 16 patients were divided into three groups based on their recovery after surgery. A control group of nonleucotomized schizophrenics was established to control for psychiatric symptomatology. A second control group consisted of subjects without a history of psychiatric or central nervous system disorder. In general, there was no statistically significant impairment of performance on attention tests between the patients with prefrontal psychosurgery and the normal control subjects. The nonoperated schizophrenic control group performed most poorly. Lesion chronicity, interaction of leucotomy and presurgical psychiatric state, and conditions of test administration are suggested as possible explanations for the unexpected results. (64 references)—Author abstract.

6502. Weiner, H. (Hoffman Division of Research, St. Elizabeths Hospital, Washington, DC 20032) Increasing slowed operant responding of schizophrenics under a fixed-ratio schedule of reinforcement. Perceptual and Motor Skills, 53(2):579-582, 1981.

Two procedures were evaluated in terms of their ability to modify the slowed, operant responding of schizophrenics under a fixed-ratio (FR 40) schedule. FR 40 response rates increased when schizophrenics were instructed to earn a minimum number of reinforcements or to imitate the rate of responding of someone shown earning that minimum number of reinforcements. The greater increase in FR 40 responding occurred under the latter procedure. The data suggest that merely providing schizophrenics with information on response/reinforcement relationships is not as effective as telling them how many reinforcers to earn. (7 references)—Author abstract, modified.

Biology

6503. Baron, M.; Perlman, R.; Levitt, M.; Meltzer, H.Y.; Gruen, R.; and Asnis, L. (New York State Psychiatric Institute, 722 West 168th St., New York, NY 10032) Plasma copper and dopamine-beta-hydroxylase in schizophrenia. Biological Psychiatry, 17(1):115-120, 1982.

Copper and dopamine-β-hydroxylase levels were studied in plasma samples from schizophrenic and control subjects in light of their relevance for the dopamine hypothesis of schizophrenia. Although the results were not statistically significant, they were consistent with previous reports of elevated plasma copper in schizophrenia. In addition, sex differences—attributable to endocrine factors— similar to those of earlier research were noted. The finding of elevated plasma copper in drug-free schizophrenic subjects and the reproducibility of copper levels over time regardless of clinical state indicate that neuroleptics do not play a major role in altering copper concentration and that copper levels are a state-independent characteristic. Several hypotheses are suggested which link copper to the biology of schizophrenia, and suggestions are made for further research using acute and catatonic patients. (23 references)

6504. Cutler, N.R.; Jeste, D.V.; Karoum F.; and Wyatt, R.J. (Adult Psychiatry Branch, Div. of Special Mental Health, Intramural Research Program, St. Elizabeths Hospital, Washington, DC 20032) Low-dose apomorphine reduces serum homovanillic acid concentrations in schizophrenic patients. Life Sciences, 30(9):753–756, 1982.

The postulated dopaminergic autoreceptor regulatory effect in man of low-dose apomorphine was investigated. Behavior and serum homovanillic acid (HVA) concentrations following low-dose apomorphine were investigated in five chronic schizophrenic patients. Serum HVA concentrations were measured by mass fragmentography before and after 0.005 mg/kg of apomorphine or saline placebo. Results demonstrate significant reductions in serum HVA concentrations in all five subjects following apomorphine as compared with placebo. These results constitute direct evidence of a specific dopamine autoreceptor effect of low-dose apomorphine in schizophrenic patients. (22 references)

6505. Darchiya, N.Sh.; Zurabashvili, Z.A.; and Dzamashvili, M.G. (No address given) Some aspects of the mechanism of action of chlorpromazine. Soobshcheniya Akademii Nauk Gruzinskoy SSR, 102(3):725–727, 1981.

The mechanism of action of chlorpromazine was studied in elements of the white blood cells of patients with schizophrenia during an acute period of the illness (clear catatonic manifestations). Before the administration of chlorpromazine, there was little clasmatosis in the regular elements of the white blood cells of the subjects. Fifteen minutes after the administration of chlorpromazine, clasmatosis increased sharply, and it reached its maximum within an hour of administration. Results show that chlorpromazine strengthens the lysosomal membranes, facilitates the emergence of lysosomes, and thus intensifies clasmatosis; it converts cells to pentose phosphatic metabolism, reduces the quantity of heterochromatin, and increases that of eucromatin.—Journal abstract, modified.

6506. Davis, B.A.; Yu, P.H.; Carlson, K.; O'Sullivan, K.; and Boulton A.A. (Psychiatry Research Division, University Hospital, Saskatoon, Saskatchewan, S7N OXO, Canada) Plasma levels of phenylacetic acid, m- and p-hydroxyphenylacetic acid, and platelet monoamine oxidase activity in schizophrenic and other patients. Psychiatry Research, 6(1):97–105, 1982.

Blood from chronic schizophrenic patients in two hospitals and from institutional and noninstitutional controls was analyzed for platelet monoamine oxidase (MAO) activity and trace acid levels. MAO activity was assessed on three different substrates: tryptamine, phenylethylamine, and *p*-tyramine. The trace acids studied were conjugated and unconjugated phenylacetic acid (PAA), *m*-hydroxyphenylacetic acid (*m*HPA), and *p*-hydroxyphenylacetic acid (*p*HPA). Compared to controls, schizophrenic patients had significantly reduced MAO activity toward all substrates. Compared to controls, schizophrenics showed significantly reduced unconjugated PAA in one hospital, increased *p*HPA in the other hospital, and increased conjugated PAA in both hospitals. However, the most consistent significant finding was a reduced unconjugated *m*HPA in both groups of schizophrenics compared with both control groups. (98 references)—Author abstract, modified.

Author abstract, modified.

6507. DeLisi, L.E.; Weinberger, D.R.; Potkin, S.G.; Neckers, L.M.; Shiling, D.J.; and Wyatt R.J. (Adult Psychiatry Branch, Intramural Research Program, Saint Elizabeths Hospital, Washington, DC 20032) Quantitative determination of immunoglobulins in CSF and plasma of chronic schizophrenic patients. British Journal of Psychiatry, 139(December):513–518, 1981.

Certain immunoglobulins—IgG, IgA, and IgM—were quantified in cerebrospinal fluid (CSF) and plasma from 35 chronic schizophrenic patients and 33 neurological and normal controls using an immunofluorescent antibody technique. A generalized reduction in immunoglubulin levels was observed in the schizophrenic patients compared with controls. While this result supports other reports of abnormal immune functioning in schizophrenia, it fails to replicate previous findings of elevations in CSF IgG and elevations in serum IgA. It is possible that although subjects were drug free, the schizophrenic patients' previous long-term neuroleptic treatment affected the immune system, or that abnormal immunity plays a role in the development of schizophrenia. (64 references)—Author abstract, modified.

6508. Dunlop, S R.; Sattin, A.; Shea, P.; and Hendrie, H.C. (Dept. of Psychiatry, Indiana University Medical Center, Indianapolis, IN 46223) Comparison of MAO, D-beta-H, and COMT activities in chronic schizophrenics selected on the basis of nailfold capillary pattern. Acta Psychiatrica Scandinavica, 64(5):409–414, 1981.

The activity in blood of three enzymes involved in the metabolism of catecholamines was measured in 17 white male chronic schizophrenic subjects divided on the basis of plexus visualization score (PVS). PVS is a measure of the visibility of a venous plexus in the skinfold proximal to the fingernail, and has been suggested as a marker to distinguish subgroups of schizophrenics. High and low PVS subjects were compared to each other and to low PVS controls on measures of platelet monoamine oxidase (MAO), red blood cell catechol-O-methyltransferase (COMT), and plasma dopamine-β-hydroxylase. There were no differences between high and low PVS subjects on any biochemical variable. Schizophrenic subjects had lower platelet MAO activity than controls. Platelet MAO and red blood cell COMT were significantly correlated in schizophrenic subjects. It is concluded that whatever the significance of the PVS feature to the presence of schizophrenia, it apparently cannot be explained in terms of biochemical variables. (19 references)—Author abstract, modified.

6509. Fulton, A.; Norman, T.R.; and Burrows, G.D. (Dept. of Psychiatry, University of Melbourne, Melbourne, Australia) Dopamine and schizophrenia—Biochemical and receptor studies. Advances in Human Psychopharmacology, 2:33–50, 1981.

Studies of the relationship of schizophrenia and dopamine, which investigate the formation of an endogenous psychotoxin by an abnormal biochemical transformation of catecholamines, dopamine metabolism in patients with schizophrenia, and dopamine receptor function, are reviewed. The data suggest that it now seems unlikely that the sumptoms usually grouped under the term "schizophrenia" are caused by excessive levels of dopamine (DA) in the central nervous system. The concept of the schizophrenic state as being generated from disturbances to the entire transmitter network would probably be closer to the true situation than a study of DA transmission abnormalities in isolation. Suggestions are presented for testing this concept. (160 references)

6510. Gattaz, W.F., and Beckmann, H. (Beckmann: Zentralinstitut für Seelische Gesundheit, Psychiatrische Klinik, Postfach 5970, D-6800 Mannheim, West Germany) The HLA-system in psychiatric research. Fortschritte der Neurologie, Psychiatrie und Ihrer Grenzgebiete, 49(4):145–151, 1981.

Studies of the action of human leukocyte antigens (HLA) on manic-depressive and schizophrenic patients are considered. In eight studies of HLA in manic-depressive patients, 17 antigens were recognized, and this occurred with a different frequency in patients when compared with controls. In nine studies of HLA in schizophrenic patients, 28 antigens showed a different frequency in patients when compared with controls. In studies with schizophrenic patients, there was relatively greater homogeneity, e.g., the HLA-A appeared with an increased frequency in patients in six out of nine studies; an increased frequency of HLA-A9 was found in five out of nine studies; and the HLA-B27 frequency was decreased in three studies. However, contradictory results were found as well. The contradictory findings may be accounted for either by assuming that there is no association between HLA and endogenous psychoses or that there are methodological biases. (38 references)—Journal abstract, modified.

6511. Golden, C.J.; MacInnes, W.D.; Ariel, R.N.; Ruedrich, S.L.; Chu, C.C.; Coffman, J.A.; Graber, B; and Bloch, S. (University of Nebraska Medical Center, Nebraska Psychiatric Institute, 42nd and Dewey Ave., Omaha, NE 68105) Cross-validation of the ability of the Luria-Nebraska Neuropsychological Battery to differentiate chronic schizophrenics with and without ventricular enlargement. Journal of Consulting and Clinical Psychology, 50(1):87–95, 1982.

An attempt was made to cross-validate discrimination rules derived from a previous study on the Luria-Nebraska Neuropsychological Battery for use in predicting ventricular enlargement and to replicate the correlations found between the Luria-Nebraska scales and ventricular-brain ratio. A group of 43 schizophrenic patients between the ages of 20 and 30 were given a computed tomography (CT) scan and the Luria-Nebraska Battery. Ventricular-brain ratio was calculated, and the Luria-Nebraska rules previously suggested were applied to predict the absence or presence of ventricular enlargement. All 15 of the patients with ventricular enlargement were correctly classified by the Luria-Nebraska. In the 28 patients with normal ventricles, the Luria-Nebraska classified 18 as normal and 10 as brain-damaged, for a combined hit rate of 33 out of 43, or 77 percent. The multiple correlation between the Luria-Nebraska scales and ventricular-brain ratio was .76, comparable to the results of the previous study. Differences between the studies and the implications of objective CT measurement for detecting cerebral atrophy are discussed. (25 references)—Author abstract, modified.

6512. Goldman, Z.; Ebstein, R.P.; Lerer, B.; Zohar, J.; Hermoni, M.; and Belmaker, R.H. (Dept. of Research, Jerusalem Mental Health Center-Ezrath Nashim, P.O. Box 140, Jerusalem, Israel) Haloperidol blood levels during dosage reduction in chronic schizophrenic patients. Neuropsychobiology, 7(6):281–284, 1981.

Haloperidol levels were measured by radioreceptor assay in 12 schizophrenic patients during gradual dosage reduction from 60 mg/day to 0 mg/day. Serum levels were highly correlated with the oral dose in each patient, but poorly correlated across patients. Clinical deterioration appeared only 10 to 20 days after total cessation of the oral drug; it was not possible to determine an optimal blood level for chronic maintenance. Below oral dosage of 10 mg/day of haloperidol, many patients' blood levels are undetectable by the radioreceptor method. (12 references)—Author abstract, modified.

6513. Goode, D.J.; Manning, A.A.; and Middleton, J.F. (Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, NC 27109) Cortical laterality and asymmetry of the Hoffman reflex in psychiatric patients. Biological Psychiatry, 16(12):1137–1152, 1981.

Cortical laterality and asymmetry of the Hoffman reflex was studied in 36 hospitalized schizophrenic or schizoaffective disorder patients judged unresponsive to conventional treatment who were treated 5 days after last receiving medication and retested 4 weeks after treatment with thiothixene, loxapine, or molindone. Results indicate that cortical laterality measures of patients were not significantly different from those of control subjects and were not related to measures of clinical symptomatology. Recovery curve height was significantly correlated with number of schizophrenic symptoms recorded for diagnosis based on the Research Diagnostic Criteria. Recovery curve height was significantly correlated with right-sided preference as measured by both visual half field and dichotic listening in medicated patients. In unmedicated patients, lateralization of the recovery curve was correlated with clinical symptomatology. Left hemispheric or subcortical dysfunction was related to schizophrenic symptomatology. Right hemispheric or subcortical dysfunction was related to depression symptomatology. Findings are compatible with asymmetries of subcortical amine function in schizophrenia and schizoaffective disorder. (53 references)—Author abstract, modified.

6514. Goudemand, M.; Goudemand, J.; Parquet, Ph.-J.; and Fontan, M. (Service de Psychiatrie Générale, C.H.U. de Lille, Unités de Soins Normalisées, 6, rue du Pr-Laguesse, F-59037 Lille Cedex, France) Schizophrenic psychoses and HLA antigens: Personal data and a review of the literature. Encephale, 7(5):609–622, 1981.

The relation between HLA antigens and schizophrenic psychoses was studied. The frequency of 33 HLA alleles in 51 paranoid schizophrenics was compared to the alleles in a control population of 94 subjects. The frequency of HLA-A 29 was lower in the schizophrenics, and the frequency of HLA-B 15 was higher, but these data were not statistically significant. However, a review of all results published between 1974 and 1980 indicates some significant associations which vary depending upon whether the schizophrenia is considered as a clinical entity or a subtype. It is concluded that the correlation between schizophrenia and HLA antigens is uncertain but that it may be related to the genetic heterogeneity of schizophrenic disorders. (55 references)—Journal abstract, modified.

6515. Guelfi, J.D., and Mialet, J.-P. (Clinique des Maladies Mentales et de l'Encephale, 100, rue de la Santé, F-75674 Paris, France) Current trends in biological psychiatry research on

psychoses. Encephale, 7(5):569-600, 1981.

Some of the biological, electrophysiological, and neurophysiological research being done on schizophrenia and affective psychoses is discussed. Most current biological hypotheses of schizophrenia—transmethylation and the dopaminergic theory—are based on disorders of the biogenic amines. The norepinephrine, serotonin, and endorphin systems may also play a role. Quantitative electroencephalographic studies have shown a dysfunction in arousal and cerebral dominance. Other electrophysiological studies have concurred with this and indicate an impairment in arousal level which may relate to the attention and adaptation disorders seen in schizophrenia. The major thrust of research on affective disorders has been on norepinephrine and serotonin abnormalities; however, acetylcholine, dopamine, electrolytic modifications of cell membranes, or immunological or neuroendocrine disturbances may also be involved. (216 references)—Journal abstract, modified.

6516. Hempel, K.; Ullrich, H.; and Philippu, G. (Institut fur Medizinische Strahlenkunde, Wurzburg, West Germany) Quantitative investigation on the urinary excretion and metabolism of 3,4-dimethoxyphenylethylamine in schizophrenics and normal individuals. Biological Psychiatry, 17(1):49-59, 1982.

A quantitative method for detecting 3,4-dimethoxyphenylethylamine (DMPEA) in the urine of schizophrenics, using the fluorometric determination of DMPEA in the form of its phosphopyridoxyl derivative, is described. The limit of detection is 2 mg DMPEA per 1 g creatinine. The DMPEA content was measured in urine from healthy persons, schizophrenic subjects, and psychiatric patients without other diagnoses who were hospitalized with the schizophrenics. From each person, 5 to 10 (24-hour) urine samples were tested, and no DMPEA was found in schizophrenic subjects, in controls, or in healthy subjects. The urinary excretion of parenterally applied ^{14}C-DMPEA was determined in three healthy volunteers and in three rats. In man, about 25 percent of the label was excreted as DMPEA. The main metabolite in urine was homoveratric acid. Both compounds were excreted as conjugates. (13 references)—Author abstract, modified.

6517. Iacono, W.G. (Dept. of Psychology, University of British Columbia, #154-2053 Main Mall, Vancouver, British Columbia, Canada V6T 1Y7) Bilateral electrodermal habituation-dishabituation and resting EEG in remitted schizophrenics. Journal of Nervous and Mental Disease, 170(2):91-101, 1982.

Bilateral electrodermal responding, heartrate, and resting electroencephalogram (EEG) were compared for 24 carefully diagnosed nonpsychotic, remitted schizophrenic subjects and 22 medical outpatient controls. Subjects were exposed to 17 pure tones; the 16th differed in frequency and duration from the others. Before the tone series, subjects engaged in 2 minutes of respiratory maneuvers, followed by a 5-minute rest period during which EEG was recorded. Consistent with past studies of chronic inpatients, the schizophrenics divided evenly into responding and nonresponding groups, and skin conductance responding was associated with a higher rate of spontaneous activity, elevated tonic conductance levels, and more responding during respiratory exercises. No group differences between controls and schizophrenic responders appeared with respect to number and amplitude of tone-elicited responses; there were no differences in dishabituation between these two groups. No evidence of any electrodermal or cerebral asymmetry was found. There were no differences between the two schizophrenic groups in measures of heartrate or resting EEG, although schizophrenics as a group produced less EEG alpha and more delta than did normals. Results are consistent with the hypothesis that electrodermal and EEG phenomena identified in psychotic hospitalized patients represent stable traits characteristic of schizophrenia. (66 references)—Author abstract, modified.

6518. Jones, G.H., and Miller, J.J. (Dept. of Psychological Medicine, Welsh National School of Medicine, Whitchurch Hospital, Cardiff CF4 7XB, Wales) Functional tests of the corpus callosum in schizophrenia. British Journal of Psychiatry, 139(December):553-557, 1981.

The notion was tested that poor links exist between the two cerebral hemispheres in schizophrenics. Subjects were 12 consecutive admissions with schizophrenia to acute wards and 12 hospital staff and student controls. The interhemispheric conduction time across the corpus callosum, as measured by the ipsilateral/contralateral latency differences of the early somatosensory evoked response, was found to be effectively zero in the schizophrenics, giving no overlap with controls. It is suggested that schizophrenia is a split brain condition akin to agenesis of the corpus callosum, but unrecognized because of the use of compensatory ipsilateral sensory pathways. (23 references)—Author abstract, modified.

6519. Kokkinidis, L., and Anisman, H. (University of Saskatchewan, Saskatoon, Saskatchewan, S7N OWO, Canada) Amphetamine psychosis and schizophrenia: A dual model. Neurosciences and Biobehavioral Reviews, 5(4):449–461, 1981.

Behavioral and neurochemical consequences of acute and chronic amphetamine treatment are reviewed. It is suggested that decreased noradrenergic activity after long-term amphetamine treatment influences stimulus sampling. Enhanced dopaminergic activity appears to be responsible for the progressive augmentation of stereotypy and self-stimulation behavior after long-term exposure to amphetamine. It is hypothesized that amphetamine-induced psychosis and the symptomatology associated with schizophrenia are related to alterations in norepinephrine and dopamine activity. (173 references)—Author abstract, modified.

6520. Kovaleva, Ye.S., and Prilipko, L.L. (Lab. obshchey patofiziologii, Institut psikhiatrii AMN SSSR, Moskva, USSR) The role of the blood serum of schizophrenic patients in the altered osmotic resistance of their lymphocytes. Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 81(7):1018–1021, 1981.

The resistance of peripheral blood lymphocytes was studied in normal people and schizophrenic patients in relation to hypo-osmotic effects. Results show that in the normal subjects about 20 percent of the lymphocytes were slightly resistant; in 60 percent of the schizophrenic subjects the proportion of slightly resistant lymphocytes was much lower; and the content of those cells in the blood of schizophrenic subjects with different forms of the disease varied—patients with the periodic form had minimal, and patients with the continuously progressive form had maximal deviations from the norm. (7 references)—Journal abstract, modified.

6521. Kushner, S.G., and Maznina, T.P. (No address given) Classes of immunoglobulins responsible for the antithymocytic activity of the blood serum in normal and mentally ill people. Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 81(7):1014–1015, 1981.

The antithymocytic activity of the blood serum was studied in schizophrenic patients and in mentally healthy control subjects using fluorescent antibodies. Results show that the antithymocytic activity of both the schizophrenic and healthy subjects was due to immunoglobulins G and M; the number of thymocytes showing fluorescence under the action of IgG was approximately the same in both groups; when fluorescein-labeled IgM was used, the number of fluorescent thymocytes was three times greater in the schizophrenic subjects than in the healthy subjects. These results suggest that these differences may be associated with increased avidity of the IgM antibodies, or with disturbances of the synthesis of immunoglobulins in the schizophrenic subjects. (5 references)—Journal abstract, modified.

6522. Landowski, J., and Wontrobski, Z. (Klinika Chorob Psychicznych, Akademia Medyczna, ul. Debiniki 7, 80-211 Gdansk, Poland) The ratio of blood platelet monoamine oxidase to aldehyde reductase activity in paranoid schizophrenia. Psychiatria Polska, 15(2):133–137, 1981.

The ratio of monoamine oxidase activity to blood platelet aldehyde reductase was determined in 47 schizophrenic patients (10 with simple schizophrenia and 37 with paranoid schizophrenia) and 20 control subjects. The value of this ratio was found to be significantly lower in paranoid schizophrenics than in simple schizophrenics and in controls. The results of the study may be related to the hypothesis suggesting a relationship between paranoid syndromes and the hyperfunction of the central noradrenergic system. These results also serve as a basis for attempts at a biochemical interpretation of the development of paranoid syndromes. (14 references)—Journal abstract, modified.

6523. Loga, S.; Curry, S.; and Lader, M. (Lader: Dept. of Pharmacology, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, England) Interaction of chlorpromazine and nortriptyline in patients with schizophrenia. Clinical Pharmacokinetics, 6(6):454–462, 1981.

Seven male inpatients suffering from acute schizophrenia were treated with 100 mg of chlorpromazine elixir for 9 weeks. Nortriptyline (50 mg) was added during weeks 4, 5, and 6. Plasma chlorpromazine concentrations, antipyrine plasma half-life, blood pressure, pulse rate, pupil size, salivation, handwriting, and clinical state were measured at weekly intervals. Plasma chlorpromazine concentrations rose when nortriptyline was added, and the antipyrine plasma half-life was prolonged. Blood pressure dropped on institution of chlorpromazine and dropped further with the addition of nortriptyline. The pulse rate rose in a parallel fashion. Pupil size, salivation, and handwriting were diminished by chlorpromazine, but hardly affected further by nortriptyline. The addition of nortriptyline dramatically reversed the therapeutic actions of chlorpromazine, mainly through pharamacodynamic interaction. It is concluded that this combination is potentially deleterious, and must be used with care. (13 references)—Author abstract.

6524. Manowitz, P.; Goldstein, L.; and Nora, R. (Dept. of Psychiatry, College of Medicine and Dentistry, Rutgers University Medical School, Piscataway, NJ 08854) An arylsulfatase A variant in schizophrenic patients: Preliminary report. Biological Psychiatry, 16(11):1107–1113, 1981.

To determine if an abnormal arylsulfatase A (ASA), an enzyme that catalyzes the degradation of sulfatides, is present in some schizophrenics, leukocyte lysates from 90 adult male schizophrenics were analyzed for ASA specific activities and electrophoretic mobilities. Results indicate that there are some schizophrenics who have ASA specific activities which are intermediate between those of adult metachromatic leukodystrophy (MLD) patients and heterozygotes for MLD. These same subjects showed an ASA with abnormal electrophoretic mobility. A proposed mechanism is that the intermediate ASA specific activities are due to an inherited amino acid substitution in the protein backbone of the enzyme which results in its abnormal mobility and lower activity. (8 references)

6525. Mitkevich, S.P. (No address given) HLA antigens and schizophrenia. Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 81(7):1016–1018, 1981.

The distribution of HLA antigens was studied in 87 schizophrenic patients with various forms of the disease, and in 130 healthy people. HLA-A10 antigen was found among patients with continuous schizophrenia, and more HLA-B12 antigen was found among patients with the attack-like form of the disease. There were no differences in the distribution of HLA antigens between healthy people and the total group of schizophrenic subjects. (17 references)—Journal abstract, modified.

6526. Moriguchi, I. (Dept. of Neurology and Psychiatry, Gifu University, School of Medicine, Gifu, Japan) A study of schizophrenic brains by computerized tomography scans. Folia Psychiatrica et Neurologica Japonica, 35(1):55–72, 1981.

The brains of 55 schizophrenics and 65 controls were examined using computerized tomography. The width of the interhemispheric fissure and the third ventricle, and the volume of the lateral ventricles in the schizophrenic patients were significantly larger than those obtained in the controls. The extension of the width of the interhemispheric fissure and the increase in volume of the lateral ventricles in the schizophrenic patients were assumed to be caused not by aging but by the schizophrenic illness itself. The increase in the volume of the lateral ventricles and the area of the anterior horns of the lateral ventricles in the schizophrenic patients was assumed to be related to the schizophrenic pathogenesis. (29 references)—Author abstract.

6527. Okasha, A., and Madkour, O. (Dept. of Psychiatry, Ain Shams University, Cairo, Egypt) Cortical and central atrophy in chronic schizophrenia. Acta Psychiatrica Scandinavica, 65(1):29–34, 1982.

Computed tomography brain scans were performed on 43 chronic schizophrenic subjects of more than 5 years' duration and under age 60, and in 39 normals matched for age, sex, and education. There was no statistical difference between controls and schizophrenics regarding cortical atrophy as measured by the interhemispheric and Sylvian fissures and mean sulcal width. Highly significant differences were found regarding central atrophy. Third ventricle measurements were normal in 9.3 percent; there was slight enlargement in 37.2 percent, moderate in 48.8 percent, and extreme in 4.7 percent. The cella media index denoting lateral ventricle dilation showed changes in 46.5 percent, slight changes in 9.3 percent, and the remaining 44.2 percent were normal. The study shows that a large number of chronic schizophrenics have an associated central atrophy. (17 references)—Author abstract.

6528. Pandey, R.S.; Gupta, A.K.; and Chaturvedi, U.C. (Dept. of Psychiatry, National Institute of Mental Health and Neuro Sciences, Bangalore, Karnataka, India) Autoimmune model of schizophrenia with special reference to antibrain antibodies. Biological Psychiatry, 16(12):1123–1136, 1981.

The prevalence of antibrain antibodies in schizophrenic patients and nonschizophrenic controls, and the relationship between the antibrain antibody titer and the duration of illness, family history, past history, and subtype of schizophrenia were investigated. Antibrain antibody titers were determined by hemagglutination technique in sera and cerebrospinal fluid (CSF) of 54 schizophrenic patients and 27 nonschizophrenic controls. Results indicate that antibrain antibodies were detected in sera and CSF of 26 schizophrenics but in none of the controls. They were significantly more often present in those schizophrenics who had a past history and family history of schizophrenia. Antibrain antibodies had no significant relationship with the subtype and the duration of schizophrenia. (60 references)—Author abstract, modified.

6529. Pavlova, Ye.V. (No address given) Microtubules and microfilaments in the embryonic human brain (normal and pathological). Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 81(7):969–974, 1981.

Microtubules and microfilaments in the embryonic human brain were studied by electron microscopy in fetuses taken from healthy and schizophrenic women in medical abortions. Results show that the brain cells of fetuses obtained from the schizophrenic women had greater numbers of the organelles of both types than cells of fetuses taken from healthy women. Results also show that in the fetuses taken from schizophrenic women the organelles formed accumulations and lost their correct orientation, the microfilaments contained compact inclusions, and the microtubules were convoluted. (22 references)—Journal abstract, modified.

6530. Rinieris, P.; Stefanis, C.; Lykouras, E.; and Varsou, E. (Dept. of Psychiatry, Athens University Medical School, Eginition Hospital, 74 Vas. Sophias Ave., Athens 611, Greece) Subtypes of schizophrenia and ABO blood types. Neuropsychobiology, 8(2):57–59, 1982.

Determination of ABO blood types was carried out in 430 (171 female and 259 male) patients with hebephrenic schizophrenia and in 340 (155 female and 185 male) patients with paranoid schizophrenia. Data were compared with a random sample of 600 individuals (268 female and 332 male) drawn from the general population. No significant association was detected between any of the blood phenotypes and affliction either with hebephrenic or paranoid schizophrenia. (6 references)—Author abstract.

6531. Rosofsky, I.; Levin, S.; and Holzman, P.S. (Levin: Mailman Research Center, McLean Hospital, 115 Mill St., Belmont, MA 02178) Psychomotility in the functional psychoses. Journal of Abnormal Psychology, 91(1):71–74, 1982.

The fine psychomotor functioning of schizophrenics, manic-depressives, hospitalized nonpsychotic patients, and normal control subjects was examined. The psychomotility measures tested were: simple reaction time (RT), traverse RT, tapping speed, dexterity, and strength of grip. Results show generalized slowing among patient groups on simple RT, traverse RT, tapping speed, and manual dexterity. Findings are interpreted with reference to other evidence involving motor impairments in psychosis. Findings of clear psychomotor slowing in psychosis suggest that psychosis implicates processes controlled in the central nervous system. These processes may reflect a more or less enduring condition of inadequate integration at the neural level that disrupts reality attunement of attention and motor behavior. (10 references)—Author abstract, modified.

6532. Schreiber, W.E.; Gentry, R.; and Fischer, E.H. (Gentry: Dept. of Psychiatry and Behavioral Sciences GI-20, University of Washington, Seattle, WA 98195) Erythrocyte calmodulin in chronic schizophrenia. American Journal of Psychiatry, 139(1):106–108, 1982.

To determine whether the protein, calmodulin, is present in excess in schizophrenia, the erythrocyte calmodulin levels of 17 schizophrenic and 18 normal subjects were measured. The calmodulin levels of the two groups were not significantly different. (10 references)—Author abstract.

6533. Senitz, D., and Winkelmann, E. (Bezirkskrankenhause für Psychiatrie und Neurologie, Olga-Benario Str. 16/18, DDR-4350 Bernburg, German Democratic Republic) Morphological findings in the orbitofrontal cortex of schizophrenics. A golgi and electron microscopic study. Psychiatrie, Neurologie und Medizinische Psychologie, 33(1):1–9, 1981.

The structure and function of nerves, their pathological changes, and variation in structures of neurons were studied using the Golgi method. Parts of the orbitofrontal cortex of eight deceased schizophrenics, 30 to 50 years old, were impregnated and fixated with neutral formal solution. Pyramidal cells with thick and partly sinuous dendrites covered heavily with spines were observed under the microscope. Also observed were irregularly arranged triangular cells in Lamina VI with thickened dendrites and increased density of spines, as well as splitting of the myelin lamellae. It is concluded that by using the Golgi method routinely, pathological specifics of changed nerve cells can be diagnosed more easily, and it is recommended that further research be done, especially in larger quantities in normal and schizophrenic cells. (18 references)—Journal abstract, modified.

6534. Siris, S.G.; Cooper, T.B.; Rafkin, A.E.; Brenner, R.; and Lieberman, J.A. (KCC 1 South, Mount Sinai Hospital, One Gustave L. Levy Place, New York, NY 10029) Plasma imipramine concentrations in patients receiving concomitant fluphenazine decanoate. American Journal of Psychiatry, 139(1):104-106, 1982.

Plasma imipramine/desipramine levels were monitored in four schizoaffective depression and residual schizophrenia with depression patients who were receiving concomitant fluphenazine decanoate and oral imipramine. High plasma levels were found, and combined tricyclic plasma levels continued to increase between days 21 and 42. Results suggest that the high plasma levels of imipramine and desipramine were the result of impaired metabolism. Findings raise concerns for potential toxicity; caution in dosing is recommended. (10 references)—Author abstract, modified.

6535. Snider, S.R. (Dept. of Neurology, University of Arizona Health Sciences Center, Tucson, AZ 85724) Cerebellar pathology in schizophrenia—Cause or consequence? Neuroscience and Biobehavioral Reviews, 6(1):47-53, 1982.

The clinical and experimental literature on cerebellar pathology in schizophrenia is reviewed in relation to the question of whether such pathology is cause or consequence of schizophrenia. Atrophy of the cerebellar vermal cortex has been reported to occur in 10 percent or more of patients with schizophrenia. Data from studies on experimental animals indicate that a functional relationship exists between the cerebellum and parts of the forebrain involved in emotion, and that the cerebellum may influence some types of behavior. Cerebellar abnormality in schizophrenic patients, although of uncertain cause, could contribute to the symptomatology of the disease. (103 references)—Author abstract, modified.

6536. Stevens, J.R. (No address given) Receptor supersensitivity: Relationships to cerebral anatomy and histopathology of schizophrenia. Biological Psychiatry, 16(12):1119-1122, 1981.

Results of altered dopaminergic binding sites in the striatum of schizophrenic patients are discussed in terms of the relationship of receptor supersensitivity to cerebral anatomy and histopathology of schizophrenia. If schizophrenia is related, even secondarily, to altered numbers or sensitivity of striatal dopaminergic binding sites, assaying samples of striatum that receive projections from relevant brain regions is advocated. Two reasons to compare medially and laterally disposed striatal structures in the study of schizophrenia are given. If there is a real difference between medial and lateral ligand binding or amine content in schizophrenia material but not in controls, the findings are suggested to be related to the clinical and anatomic pathology of schizophrenia. (12 references)

6537. Walker, E., and Green, M. (Cornell University, Ithaca, NY 14850) Soft signs of neurological dysfunction in schizophrenia: An investigation of lateral performance. Biological Psychiatry, 17(3):381-386, 1982.

Lateral differences in neurological soft signs were investigated in 20 schizophrenic subjects, 20 depressed subjects, and 20 normal controls, bilaterally administered measures of haptic sensitivity and neuromotor function. Earlier findings that schizophrenic subjects exhibited more neurological soft signs than psychiatric and normal controls were confirmed; however, there was no evidence of lateral asymmetry in abnormalities for any of the groups. Findings are discussed in terms of their implications for theories of hemispheric dysfunction in schizophrenia. (10 references)

6538. Yu, P.H.; Bowen, R.; Carlson, K.; O'Sullivan, K.; and Boulton, A.A. (Psychiatric Research Division, University Hospital, Saskatoon, Saskatchewan, S7N OXO, Canada) Comparison of biochemical properties of platelet monoamine oxidase in mentally disordered and healthy individuals. Psychiatry Research, 6(1):107-121, 1982.

Some biochemical properties of platelet monoamine oxidase (MAO) isolated from chronic schizophrenics, institutionalized nonschizophrenics, and healthy controls were investigated. The enzyme activity level in the healthy population was reasonably constant over at least a 6-week period. Correlations were found for all groups between MAO activity assessed for different substrates: *p*-tyramine, β-phenylethylamine, and tryptamine. Some heterogeneity of the platelet MAO may exist, however, at least in some of the chronic schizophrenics, since the substrate specificities were changed and the K_m values were reduced. The half-life of the enzyme at 58°C was 2 to 3 minutes and the transition temperature derived from Arrhenius plots was 16 to 17°C with respect to β-phenylethylamine. Platelet MAO from chronic schizophrenics was not significantly different from control values with respect to temperature effects. SES-polyacrylamide gel electrophoresis of the ^{3}H-pargyline-MAO adduct revealed that the subunit of platelet MAO is a single band protein with a molecular weight of about 65,000. No apparent difference was observed between individuals. The adjacent structure of the flavine site of the platelet MAO was chromatographically identical to the pentapeptide isolated from MAOs from other tissues. The response of platelet MAO to thimerosal, a new differential type-A MAO inhibitor, could be distinguished not only from type-A MAO isolated from human placenta, but also from type-B MAO isolated from bovine liver. (38 references)—Author abstract, modified.

6539. Zarifian, E.; Scatton, B.; Bianchetti, G.; Cuche, H.; Loo, H.; and Morselli, P.L. (Service Hospitalo Universitaire de Santé Mentale et de Therapeutique, Hôpital St. Anne, 1 Rue Cabanis, F-75014 Paris, France) High doses of haloperidol in schizophrenia. Archives of General Psychiatry, 39(2):212-215, 1982.

The effects of high doses of haloperidol on clinical status, plasma neuroleptic and prolactin concentrations, and cerebrospinal fluid (CSF) levels of homovanillic acid (HVA) and γ-aminobutyric acid (GABA) were investigated in three paranoid schizophrenic patients over 6 weeks. Oral dosages were increased at weekly intervals from 10 mg/day to 200 mg/day and then reduced to 10 mg/day. The increase did not affect paranoid symptoms. Neurological side effects were slightly increased in two patients and were moderately reduced in one. Plasma prolactin levels, initially high, increased when the dosage was increased to 100 mg/day, but did not increase further. The CSF levels of HVA and GABA increased to day 7 but returned to initial values on day 28 in two patients; they were decreased to day 28 in one patient. (22 references)—Author abstract, modified.

Case Studies

6540. Bienenfeld, D., and Hartford, J.T. (Hartford: 7206 Medical Sciences Building, 231 Bethesda Avenue, Cincinnati, OH 45267) Pseudodementia in an elderly woman with schizophrenia. American Journal of Psychiatry, 139(1):114-115, 1982.

The case of a 71-year-old woman is described who was hospitalized with symptoms suggesting dementia: confusion, memory problems, slovenliness, and delusions. The psychiatric history, however, confirmed the existence of a functional psychiatric disorder and a pattern of reversible cognitive impairment. Unmedicated, the patient showed no change in cognitive function, affect, or delusion. Initiation of treatment with fluphenazine hydrochloride resulted in steady improvement, and the patient was discharged on maintenance fluphenazine. The history and presentation of the patient fulfilled *DSM-III* criteria for undifferentiated schizophrenia. The association of pseudodementia with schizophrenia emphasizes the need to search for the particular features of this syndrome in all patients presenting with cognitive deficits. (5 references)—Author abstract, modified.

6541. Kiejna, A.; Piss, T.; and Leszek, J. (Klinika Psychiatryczna, Akademia Medyczna, ul. Kraszewskiego 25, 50-229 Wroclaw, Poland) A pharmacogenic type of cataleptic reaction in the course of treatment for schizophrenia. Psychiatria Polska, 15(1):91-93, 1981.

A pharmacogenic type of cataleptic reaction in the course of treatment for schizophrenia is described in a case study. On admission, the patient had difficulties in making verbal contact, behaved eccentrically, was autistic, and had a slovenly appearance. A catatonic state was diagnosed, and he was given 10 mg of haloperidol. Following the appearance of hypertonic hypokinetic dyskinesias, haloperidol was replaced with thioridazine in doses up to 600 mg. The patient's state deteriorated. In relation to contemporary administration of neuroleptics, it has been observed that cataleptic reactions appear during the treatment of catatonic schizophrenia and may be the result of the blocking of dopaminergic receptors. The possibility of other predisposing factors, such as physical constitution or a function of brain cells, cannot be excluded. (8 references)

6542. Nanko, S. (Institute of Brain Research, School of Medicine, University of Tokyo, Tokyo, Japan)

Schizophrenia-like psychosis in a 46,XX male. Folia Psychiatrica et Neurologica Japonica, 35(4):461-463, 1981.

A case of a schizophrenia-like psychosis in a 46,XX male found among 4,129 male inpatients in Japanese mental hospitals is presented. It is noted that the XX male is rather rare among this population. The main clinical feature was a formal thought disorder that included delusions, auditory hallucinations, and poor reality testing. This is the first report of a 46,XX male with schizophrenia-like psychosis. (12 references)—Author abstract, modified.

6543. Rovera, G.G.; Angelini, G.; and Possamai, L. (Istituto di Clinica Psichiatrica, Università di Torino, Torino, Italy) Two cases of transsexual schizophrenia. Minerva Psichiatrica, 22(2):99-103, 1981.

The literature on transsexuality is reviewed, with emphasis on biological, psychopathological, and therapeutic research. Adult sexual manifestation is defined as dependent on: (1) the prenatal hormonal mix; (2) the prepubescent psychosocial structuring; (3) hormonal activity from puberty to adulthood; and (4) structure and sexual disposition of partners. Two cases are analyzed. (8 references)—Journal abstract, modified.

6544. Sharma, S.K.; Garg, A.R.; and Pamecha, R. (136 Ashok Nahar, Udaipur (Raj.), 313 001, India) Lawrence-Moon-Biedl syndrome with schizophrenia. Australian Journal of Developmental Disabilities, 7(4):193-194, 1981.

A case of Lawrence-Moon-Biedl syndrome with schizophrenia is reported in an 18-year-old female. The patient displayed all the physical features of this syndrome, which includes obesity, mental retardation, retinitis pigmentosa, hypogenitalism, and polydactyly with an autosomal recessive mode of inheritance. In addition, she showed a gradual onset of psychiatric symptoms, beginning with insomnia, and followed by behavioral abnormalities including talking to herself, refusal to eat, running away, hallucinatory behaviors, and paranoid delusions. Treatment with oral chlorpromazine (800 mg/day) resulted in gradual improvement of mental disturbances over a 4-week period. (8 references)

Cross-Cultural Studies

6545. Dean, G.; Walsh, D.; Downing, H.; and Shelley, E. (Medico-Social Research Board, 73 Lower Baggot St., Dublin 2, Ireland) First admissions of native-born and immigrants to psychiatric hospitals in South-East England 1976. British Journal of Psychiatry, 139 (December):506-512, 1981.

In 1976, over 91 percent of all first admissions to psychiatric hospitals in southeast England were analyzed by birthplace, sex, age group, and marital status. First admissions for schizophrenia were five times the expected number of immigrants from New Commonwealth America (the West Indies), four times the expected number for immigrants from New Commonwealth Africa (mostly ethnic Asians), and three times the expected number from India. Immigrants from Pakistan and the remaining New Commonwealth Asian countries did not show a significantly higher than expected number of admissions for schizophrenia, and their first admissions for alcoholic psychosis and alcoholism, psychoneuroses, and personality and behavior disorders were significantly fewer than expected. First admissions for schizophrenia were also significantly more than expected among immigrants from Ireland, Germany, and Poland, but not from Italy. Females had a higher first admission rate than males, and the highest first admission rates for both males and females occurred in those aged 20 to 34 and in those over 55 years old. Cultural factors influencing differential admission rates among immigrants are discussed. (17 references)—Author abstract, modified.

6546. El-Islam, M.F. (Faculty of Medicine, P.O. Box 24923, Kuwait) Rehabilitation of schizophrenics by the extended family. Acta Psychiatrica Scandinavica, 65(2):112-119, 1982.

Patterns of care by extended and nuclear families for their relatives were studied in 540 Arab schizophrenic outpatients in Qatar. The extended family differs from the nuclear family in its larger size and in the greater emotional commitment of its members to each other's well-being. The extended family is more tolerant of patients' minor behavioral abnormalities and temporary protective withdrawals. The extended family is more helpful in supervising patients' medication, social adjustment, and use of leisure time. The more traditionally oriented extended family helps patients understand their illness in terms of cultural belief systems once they lose the components that cannot be contained in these systems. Findings show that extended family members are less likely than nuclear family members to tax the patients' emotional resources and limited repertoire of social skills. (14 references)—Author abstract, modified.

Description

6547. Adebimpe, V.R.; Klein, H.E.; and Fried, J. (Northern Communities Mental Health & Mental Retardation Center, River Ave. and Alcor St., Pittsburgh, PA 15212) Hallucinations and delusions in black psychiatric patients. Journal of the National Medical Association, 73(6):517-520, 1981.

The incidence of hallucinations and delusions was compared in a sample of 129 black and 146 white schizophrenic patients to determine if black patients experience hallucinations and delusions more frequently than whites. Results from standardized research rating scales—in contrast to previous reports based on hospital records—indicate hallucinations are more frequent among blacks, but no racial differences are found in the frequency of delusions. Current ignorance regarding the content of hallucinations in normal and nonschizophrenic, and in schizophrenic black subjects is noted as a source of diagnostic confusion which may have dire consequences for many individuals. (23 references)—Author abstract, modified.

6548. Bellodi, L.; Morabito, A.; Macciardi, F.; Gasperini, M.; Benvenuto, M.G.; Grassi, G.; Marzorati-Spairani, C.; and Smeraldi, E. (Institute of Clinical Psychiatry, via F. Sforza 35, I-20122 Milano, Italy) Analytic considerations about observed distribution of age of onset in schizophrenia. Neuropsychobiology, 8(2):93-101, 1982.

In light of possible use of age correction in future genetic studies of schizophrenia, the existence of factors influencing a frequency distribution of age at the onset of the disease was evaluated in data for 217 patients. Factors examined included sex, diagnostic subtype, marital status, present age, duration of illness, existence of affected parents at patient's time of onset, number of siblings, birth order, and a variable which separates patients with siblings from those without. Of these variables, sex, diagnosis, number of siblings, and birth order were significant according to the Cox's regression model used. (17 references)—Author abstract, modified.

6549. Biebel, D. (Helmtrudenstr. 1, D-8000 Munchen 40, West Germany) The schizophrenically reacting mother. Dynamische Psychiatrie, 14(4/5):187-201, 1981.

The genetic explanation of schizophrenia that is used to justify abortion, sterilization, and adoption is challenged by a case study and a related pilot study. In the case study, a schizophrenically reacting patient in the therapeutic process gradually developed the social energetic deficits of her childhood, and thus became more able to be a mother to her child. The pilot study on the group dynamics of pregnancy and birth demonstrates that schizophrenically reacting mothers do not have schizophrenically ill children. (15 references)—Journal abstract, modified.

6550. Boucharlat, J. (Service de Psychotherapie, Hôpital Sud, Centre Hospitalier Regional Universitaire de Grenoble, F-38130 Echirolles, France) Schizophrenic conversation—Mythic conversation: A diary. Psychologie Medicale, 13(13):2103-2104, 1981.

Excerpts from a young schizophrenic patient's diary are presented, and their significance is discussed. The diary is of interest because it touches on universal themes which are reflected in religious myths, such as abandonment, death, mutilation, and love. It is suggested that present day psychology, which emphasizes psychopathology and biological discoveries, should also consider the metaphysical aspect of existence. (5 references)—Journal abstract, modified.

6551. Carrico, K.L., Jr. (Michigan State University, East Lansing, MI 48823) An examination of the social networks of normals and schizophrenics. Dissertation Abstracts International, 42(8):3490-B, 1982.

The social networks of normal subjects and schizophrenics were compared. The comparison was made along four classes of social network variables: structure, content, function, and emotion. In the first phase, it was found that the schizophrenic patients' perception of social relationships was as reliable as was the perception of the normals. Self-report data were found to represent solely the subjects' perceptions regarding social networks, not the actual status of the social networks. In the second phase—the comparison between social networks of the normals and schizophrenics—differences were found in perceived network size and reciprocity. Schizophrenic subjects perceived themselves to be surrounded by fewer important persons than did normals. Normals perceived their relationships as reciprocal in terms of support, whereas schizophrenic patients perceived the relationships as lacking in reciprocity.—Journal abstract, modified.

6552. Darchiya, N.Sh., and Zurabashvili, Z.A. (No address given) Certain types of clasmatosis in schizophrenia. Soobshcheniya Akademii Nauk Gruzinskoy SSR, 103(3):729-731, 1981.

Some types of clasmatosis occurring in schizophrenia were studied. In attack-like progressive schizophrenia, clasmatosis formations are bright, optically empty, and elongated (ellipsoidal). Clasmatosis in paranoid schizophrenia takes the form of very large pyriform formations of optically heterogeneous density. The total number of clasmatosis formations in paranoid schizophrenia is greater than in attack-like progressive schizophrenia. These results show that the inclusions found in clasmatosis formations differ in shape, content, nature, quantity, and other characteristics, depending on the nature of the illness.—Journal abstract, modified.

6553. Destounis, N. (12 Lykavitton Street, Athens 136, Greece) Schizophrenia—A psycho-social approach. Dynamische Psychiatrie, 14(4/5):301-305, 1981.

It is suggested that the field of psychiatry should be approached not only from the viewpoint of already known disciplines, but also from an existential and phenomenological point of view. Cultural psychiatry and social psychiatry have opened new horizons into the understanding of the etiology and treatment of mental diseases such as schizophrenia. Social psychiatry is primarily a field of research, and cultural psychiatry, as its branch, deals with the sociocultural aspects of mental disorders in a given cultural milieu. The present technological revolution decreases man's capacity for adaptation due to physical, social, and psychological stresses. The confrontation of these stresses requires continuous transcultural research in order to increase our knowledge of the genesis and treatment of mental illness and, in particular, of schizophrenia.

6554. Freire, M.; Roy, A.; Blake, P.; and Seeman, M.V. (University of Toronto, Ontario, Canada) Rejection in parents of schizophrenic outpatients correlates with time spent together. Comprehensive Psychiatry, 23(2):190-193, 1982.

Parental rejection was measured in the parents of 16 schizophrenic outpatients by a self-report questionnaire. Parental expression of hostility was positively correlated with time spent with the patient. This was true in a relatively stable patient population protected by neuroleptics. It is concluded that if further studies confirm that parental rejection is partly reactive, that reaction may be amenable to therapeutic intervention. The findings are discussed in the context of previous work with expressed emotion and schizophrenic relapse. (8 references)—Author abstract, modified.

6555. Fujinawa, A. (Psychology and Psychopathology Inst., Kyoto University, Nihonmatsu-Cho, Yoshida, Sakyo-ko, Kyoto 606, Japan) Schizophrenic patients and self-portraits. Dynamische Psychiatrie, 14(4/5):250-261, 1981.

A study is presented of schizophrenic patients able to draw their inner self-image, but unable to draw their self-portraits from a mirror. The problem is considered from the perspective of observing and analyzing the changing of the patient's attitudes toward portraying himself. Lacan's concept of the mirror phase is the theoretical background for dividing the patient's attitude toward self-portraiture into three phases. These are: (1) the transient phase when the patient begins to come out of the pathological state, (2) the phase in which the patient develops his inner self-image, and (3) the phase in which the patient is able to draw his image as it appears in the mirror. It is suggested that a schizophrenic patient, in the course of therapy, undergoes these phases several times and in an increasingly differentiated manner. Through the self-portraits, this process can be analyzed visually.—Journal abstract, modified.

6556. Galle, G. (Universitats-Nervenklinik mit Poliklinik, Schwabachanlage 6+10, D-8520 Erlangen, West Germany) Transitory initial schizophrenic episodes. Psycho: Psychiatrie und Medizinische Psychologie für die Praxis, 7(1):24-27, 1981.

Initial schizophrenic episodes of young people who are not diagnosed as acute schizophrenics are examined. Transitory initial schizophrenic episodes may be recognized as schizophrenic only in retrospect. Five case histories are reported of people under age 25 who had received 8 days of psychiatric inpatient treatment and then were released. Reported symptoms included weakness, nervousness, apathy, feelings of paranoia, mistrust, and anxiety. The diagnosis of transitory schizophrenia proved to be correct in three cases. It is concluded that the terms "pubertal crisis" and "general depression" often are used inappropriately to describe schizophrenic phenomena. (6 references)

6557. Gerard, K. (Zaklad Psychiatrii Spolecznej, Instytut Psychoneuro-

logiczny, Al. Sobieskiego 1/9, 02-957 Warszawa, Poland) Changes in the sociodemographic structure of populations of patients first hospitalized for schizophrenia. Psychiatria Polska, 15(1):29-36, 1981.

The sociodemographic characteristics of schizophrenic patients first hospitalized during the period 1927 to 1968 were studied. There was a decrease in the percentage of farmers and poorly educated subjects, with a simultaneous increase in the percentage of white-collar workers and better educated subjects; there were also fewer patients from large families. Patients differed from the general population in that they were mainly single and childless. Initially, the number of single and childless subjects was similar for both sexes; with the passage of time it increased for men and decreased for women. The mean age at the time of hospitalization remained the same for men, but increased for women. These changes suggest a trend toward a mitigation of the morbid process in women and the opposite tendency in men. (13 references)—Journal abstract, modified.

6558. Glazer, W.; Sholomskas, D.; Williams, D.; and Weissman M. (34 Park Street, New Haven, CT 06519) Chronic schizophrenics in the community: Are they able to report their social adjustment? American Journal of Orthopsychiatry 52(1):166-171, 1982.

Data obtained from the administration of the Social Adjustment Scale II were compared for 56 schizophrenic subjects and their significant others. It is noted that deinstitutionalization of schizophrenic patients makes it imperative that we develop reliable and valid methods to assess social adjustment. High agreement was found between the reports of schizophrenics and their significant others. The findings suggest that the reports of schizophrenic patients about their social adjustment are a reasonable indicator of status. (15 references)—Author abstract, modified.

6559. Grunewald, U.; Korabelnikov, K.; and Pink, E. (Bezirksfachrankenhaus für Psychiatrie/Neurologie, DDR-9706 Rodewisch, German Democratic Republic) Social development conditions in schizophrenics. Results of data analysis of rehabilitation questionnaires. Psychiatrie, Neurologie und Medizinische Psychologie, 33(1):39-47, 1981.

One-hundred schizophrenic patients, 17 to 55 years old, were studied at the Karl Marx University in Leipzig to determine their premorbid disabilities. A survey of the literature on family research and the social environment of schizophrenic patients shows that patients develop paranoid syndromes, neurosis, and depression in their youth. Premorbid distress was noted more frequently in marriage partners and less frequently in parents. It is concluded that the social-psychological problems and peculiarities of schizophrenics are the consequences of a disturbed premorbid development in social communication. It is recommended that family members and other people close to the schizophrenic patient undergo personality-centered therapy to overcome difficult emotional problems and to enable them to face reality in family as well as on the job. (19 references)—Journal abstract, modified.

6560. Hubschmid, T. (Sozialpsychiatrische Universitatsklinik, Murtenstr. 21, CH-3010 Bern, Switzerland) Psychiatric thinking—Systemic thinking. Familiendynamik, 6(4):366-378, 1981.

Three theories of schizophrenia which influence the procedures in psychiatric institutions are reviewed: medical/somatic, psychoanalytic, and family-oriented approaches. Examples from a social/psychiatric clinic which illustrate a possible approach based on a systemic model also are examined. (17 references)—Journal abstract, modified.

6561. Hunca-Bednarska, A.; Olajossy, M.; Chlewinski, Z.; and Bednarski, M. (Klinika Psychiatryczna, Akademia Medyczna, ul. Abramowicka 2, 20-442 Lublin, Poland) The emotional significance of selected concepts in a group of patients with early and chronic schizophrenia (a comprehensive study). Psychiatria Polska, 15(1):1-7, 1981.

Forty patients with early and chronic schizophrenia were studied using the Osgood Semantic Differential. Concepts evaluated by the patients had a positive, a negative, an aggressive, and an indifferent emotional character. The emotional importance of emotionally indifferent and positive concepts was found to vary significantly, due mainly to the influence of such factors as valuation, stability, and sensitivity. The results obtained for patients with chronic schizophrenia reveal more intense emotional associations, and may suggest that there is no simple loss of emotionality in these patients. (20 references)—Journal abstract.

6562. Jeste, D.V.; Kleinman, J.E.; Potkin, S.G.; Luchins, D.J.; and Weinberger, D.R. (Adult Psychiatry Branch, Division of Special Mental

Health Research, NIMH, St. Elizabeths Hospital, Washington, DC 20032) Ex uno multi: Subtyping the schizophrenic syndrome. Biological Psychiatry, 17(2):199–222, 1982.

Ninety-three chronic schizophrenic inpatients who met the Research Diagnostic Criteria for schizophrenia were studied. Data on a number of historical, epidemiologic, phenomenologic, biochemical, neuropathological, and treatment/response variables were analyzed. Patients were classified into pairs of subgroups as follows: (1) ventricle/brain ratio (VBR) assessed on computed tomography (normal vs. abnormal); (2) premorbid adjustment (good vs. poor); (3) therapeutic response to neuroleptics (good vs. poor); (4) platelet monoamine oxidase (MAO) activity (low vs. high); (5) paranoid features (present vs. absent); (6) tardive dyskinesia (present vs. absent); and (7) hemispheric asymmetry on computed tomography (normal vs. abnormal). Results suggest that two biological variables, VBR and platelet MAO activity, might be useful in identifying two distinct subgroups among chronic schizophrenic patients. A subgroup with large VBR was associated with poor premorbid adjustment, neurological impairment, and poor therapeutic response to neuroleptics, while the subgroup with low platelet MAO activity displayed paranoid features and tardive dyskinesia. (47 references)—Author abstract, modified.

6563. Julien, R.A.; D'Agostino, N.; and Balzamo, E. (Service de Psychiatrie, Secteur VI des Bouches du Rhône, CHS Valvert, Boulevard des Liberateurs, F-13011 Marseille, France) Dream recall and primary delusional experience in schizophrenics. Psychologie Medicale, 13(13):2107–2112, 1981.

Dream recall of five schizophrenics who were either in an acute phase or in remission was studied. Subjects spent three consecutive nights in the sleep laboratory. Their first night's sleep was uninterrupted; during the second and/or third nights they were awakened three or four times 10 minutes after the electroencephalogram indicated that rapid eye movement sleep had begun, and were asked to tell their dreams. All the dreams were closely related to the patients' lives, but contrasted with their waking states. The mood in the dream often contrasted with the mood of the previous day, and subjects often made semantic modifications when recalling the dream. (18 references)

6564. Kobal, M. (Psychiatrische Universitatsklinik Ljubljana, YU-61260 Ljubljana Polje, Yugoslavia) Some characteristics of schizophrenia in adolescence. Dynamische Psychiatrie, 14(6):332–344, 1981.

Factors in the development of adolescent schizophrenia are discussed, including genetics, individual peculiarities in the workings of the nervous system, and early childhood development. Fifty-five patients of the Adolescent Unit of the Mental Health Center in Ljubljana were observed. Patients' symptoms which occurred most often were introjection, problems in abandoning childish behavior patterns, preoccupation with eschatological problems and transcendental phenomena, and shift in thinking toward details. It is emphasized that all of the above categories in and of themselves are normal preoccupations of adolescents, but that the persistence of these symptoms may indicate the onset of schizophrenia. (15 references)

6565. Kramer-Dover, S.L. (George Washington University, Washington, DC 20006) The similarity in psychopathology between schizophrenic mothers and their children. Dissertation Abstracts International, 48(2):3428–B, 1982.

Similarity in psychopathology was investigated in 115 Danish schizophrenic mother-child dyads. A total of 37 symptom syndromes, scored as present or absent for mothers, were compared with symptom syndrome scores of adult offspring. Contrary to expectations, schizophrenic mothers and their offspring were not found to be similar in terms of psychopathological symptoms manifested by each, and mother-daughter dyads exhibited the same degree of similarity as mother-son dyads. Thus, the social learning model of symptom transmission was not substantiated. Proximity in time of ratings was not a factor determining similarity, but proximity of age of mothers and children at respective times of rating was positively associated with degree of symptom similarity.—Journal abstract, modified.

6566. Leff, J., and Abberton, E. (MRC Social Psychiatry Unit, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, England) Voice pitch measurements in schizophrenia and depression. Psychological Medicine, 11(4):849–852, 1981.

A laryngograph was used to attempt to distinguish between the monotonous voice produced by schizophrenic patients and that of severely depressed patients. Voice pitch was studied in a series of emotionally blunted and nonblunted schizophrenic patients, and retarded and nonretarded depressive patients. The frequency distributions of the laryngographic recordings were analyzed to yield kurtosis scores. The group of retarded depressive patients had a significantly higher kurtosis score than the group of blunted schizophrenic patients. Hence this technique allows an objective distinction to be made between two kinds of monotonous voices produced by psychiatric patients. It is noted that blunted schizophrenic patients had a higher mean kurtosis score than nonblunted schizophrenic patients. (5 references)—Author abstract, modified.

6567. Majczak, A.; Olajossy, M.; Nagay, J.; and Perzynski, J. (Klinika Psychiatrii, Akademia Medyczna, ul. Abramowicka 2, 20-442 Lublin, Poland) Analysis of selected data concerning patients first hospitalized for schizophrenia between 1971 and 1975 in the city and voivodship of Lublin. Psychiatria Polska, 15(1): 17-22, 1981.

Case histories of 272 schizophrenic patients first hospitalized between 1971 and 1975 in the city and voivodship of Lublin were studied. More men than women had been hospitalized, particularly among those in the 21- to 25-year-old age group. Rehospitalization was more frequent in patients from the city of Lublin. Patients from the city did not differ from those from the voivodship (province) of Lublin with regard to social status or number of children. Analysis of the sources of income for 1975 revealed that a greater percentage of professionally active patients were in the group first hospitalized between 1971 and 1975, as compared to a group of those first hospitalized between 1960 and 1970. (10 references)—Journal abstract, modified.

6568. Meya, U. (Abt. Neurologie, Medizinische Fakultat, RWTH Aachen, Goethestrasse 27/29, D-5100 Aachen, West Germany) The problem of the attitude adopted by psychotic patients toward their own disease, seen from the aspect of its semantic (definitional significance) development. Fortschritte der Neurologie-Psychiatrie, 49(11): 395-405, 1981.

Studies on the way schizophrenic patients view their illness are presented, and a model to visualize the complex social network in which patients find themselves while assimilating the experience of their psychosis is presented. The model is based on advances in social psychology during the past 25 years. The most important elements with characteristic influences on the process of assimilation are: (1) typical judgments by the public about mental disease; (2) judgments of the social primary group and the way of obtaining the social role of a patient; (3) interaction with close friends and relatives; (4) the experience of a mental hospital, the treatment, and the therapist, nurses, and other patients; and (5) previous experience as a patient. The model presents the following conclusions: (1) a high degree of harmony between the elements listed facilitates the assimilation of the experience of a psychosis and thus leads to a better prognosis; and (2) the information gained in a social setting of an investigator exploring a patient represents just a small part of the complex process of assimilating the experience of a psychosis. (64 references)—Author abstract, modified.

6569. Neufeldt, A.H. (Dept. of Psychology, York University, Downsview, Ontario M3J IP3, Canada) ". . .Move over!": A message from self-help groups. Canada's Mental Health, 29(4):3-4, 33, 1981.

The history, purpose, and accomplishments of two Canadian self-help groups, On Our Own and Friends of Schizophrenics, are described. Founded in 1977, On Our Own is an organization composed of deinstitutionalized psychiatric patients, providing friendship, understanding, and support through outreach to those still in institutions and those just released. Features of the organization include a democratic, self-governing, nonprofit structure with monthly business meetings and annual meetings for members. Friends of Schizophrenics, initiated in 1978 by parents of schizophrenics, provides a forum for support, discussion, and information for the families of schizophrenic patients. In addition to supporting families, the group has devoted much energy to educating the public. (3 references)

6570. Perzynski, J. (Klinika Psychiatrii, Akademia Medyczna, ul. Abramowicka 2, 20-442 Lublin, Poland) Studies of the structure and dynamics of neurotic syndromes initiating schizophrenic processes. Psychiatria Polska, 15(1):9-15, 1981.

Two groups of 22 patients each underwent clinical analysis. The first group was composed of patients in whom schizophrenia had begun with neurotic disorders; the other group was composed of neurotic patients. The two groups were found to differ in a statistically significant way with regard to age at onset of the illness, duration of the disorder, and number and frequency of the various symptoms. Great variations were found in the neurotic syndromes of early schizophrenia with respect to the occurrence, intensity, and season-dependent intensification of symptoms as well as the occurrence of symptoms characteristic of this group of patients.—Journal abstract.

6571. Pull, C.B. (Centre Hospitalier de Luxembourg, 4, rue Barble, Luxembourg, Belgium) Depression and schizophrenia. Encephale, 7:343–346, 1981.

The classification of affective psychoses and schizophrenic psychoses is discussed. Although schizophrenia and depression represent two valid and distinct classifications of disorders, the boundary between the two is vague and it is difficult to classify illnesses with both schizophrenic and affective elements. It is uncertain whether schizoaffective psychoses should be classified under the schizophrenias or affective psychoses, or if they are distinct from both of these classifications. It is concluded that although they probably do not constitute a homogeneous grouping, affective psychoses and schizophrenic psychoses should be classified separately at present, if only to facilitate further research. (15 references)—Journal abstract, modified.

6572. Ramsay, R.A.; Ananth, J.; Engelsmann, F.; Krakowski, A.J.; Wittkower, E.D.; and Ghadirian, A.M. (McGill University, P.O.B. 6070, Montreal, Quebec H3C 3GI, Canada) Schizophrenia and psychosomatic illness. Journal of Psychosomatic Research, 26(1):33–42, 1982.

To explore the possibility of a mutually exclusive relationship between schizophrenia and certain psychosomatic disorders, 354 adult schizophrenic patients, both acute and chronic, were studied with regard to lifetime prevalence of peptic ulcer, bronchial asthma, neurodermatitis, and rheumatoid arthritis. Significant evidence to support the hypotheses of mutual exclusivity was found only in the case of peptic ulcer in the overall sample. Prevalence was higher in younger, more acute patients than in older, chronic patients, but it is suggested that this finding is difficult to interpret. Parallel inquiry into the prevalence of schizophrenia and the same disorders among family members of the patients yielded inconclusive results. (34 references)—Author abstract.

6573. Rosenberger, P., and Lewine, R.R.J. (Lewine: Illinois State Psychiatric Institute, 1601 West Taylor Street, Chicago, IL 60612) Conceptual issues in the choice of a structured psychiatric interview. Comprehensive Psychiatry, 23(2):116–123, 1982.

Two popularly used interviews, the Present State Examination (PSE) and the Schedule for Affective Disorders and Schizophrenia (SADS), are compared to assist the clinician in making an informed choice of available interview schedules. It is suggested that the interviewer's cognitive task, the conceptualization of psychopathology, and clinical and research objectives must be considered when choosing an interviewing schedule. It is noted that the PSE may be more appropriate for making treatment decisions and more useful in clinical work, while the SADS, oriented toward longer duration illness, might be used to conduct family history studies and to assign subjects to different treatment groups. (23 references)

6574. Rousselot, Y. (No address given) Langfeldt's conception of schizophrenia. Annales Medico-Psychologiques, 139(8):879–892, 1981.

Langfeldt's method of classifying schizophrenia is discussed, and a few current opinions of his system are reviewed briefly. Langfeldt divided schizophrenia into two types: true schizophrenia with dementia, and an illness with symptoms similar to schizophrenia but with a good prognosis. True schizophrenia is recognized by a number of chronic symptoms; in addition, factors such as biotype, character, heredity, and etiology will influence the prognosis. (32 references)

6575. Roux, G. (9, place Royale, F-64000 Pau, France) The finished drawing, sketches, incompleteness. Psychologie Medicale, 13(9):1435–1440, 1981.

Three observations of finished and unfinished drawings by schizophrenics are discussed. The sketch, or unfinished drawing, is usually a preliminary to a more detailed picture, but it can also be a habitual mode of expression as it commonly is for schizophrenics. The drawings discussed are highly individualized but at the same time have common elements. The few pictures that have been completed are cold and inhuman, and they cannot compare with the richness of the sketches.—Journal abstract, modified.

6576. Rozycka, J., and Swornowska, K. (Instytut Psychologii, Uniwersytet Wroclawski, Dawida 1, 50-527

Wroclaw, Poland) Ingratiatory behavior in paranoid schizophrenics. Psychiatria Polska, 15(1):23-27, 1981.

The behavior of 40 paranoid schizophrenic patients was studied to determine whether such patients are capable of ingratiatory behavior. It was found that some paranoid schizophrenics, even those hospitalized for 10 to 20 years, were capable of ingratiatory activities. This confirms the viewpoint that paranoid schizophrenia is characterized by selective rather than total pathology in human interrelations. (4 references)—Journal abstract, modified.

6577. Strian, F.; Heger, R.; and Klicpera, C. (Max Planck Institut für Psychiatrie, Kraepelinstrasse 10, D-8000 Munchen 40, West Germany) The time structure of depressive mood in schizophrenic patients. Acta Psychiatrica Scandinavica, 65(1):66-73, 1982.

The depressive mood of 134 schizophrenic patients was examined. Patients completed a self-rating mood scale every other day. The frequency of depressive mood and the intensity and dynamics of each period of depression were analyzed using defined algorithms. Marked depression was found in more than half of the patients. Depression was seen predominantly at the beginning of hospitalization, but subsequent occurrences were not concentrated in any specific time periods. The results suggest that depression in schizophrenia is predominantly an experience or consequence of the underlying illness and that pharmacological factors are not as important as has been previously assumed in the literature. (18 references)—Author abstract.

6578. Tyszkiewicz, M. (No address given) Artistic works of schizophrenic patients viewed as personal creative expression. Zdrowie Psychiczne, 22(1):43-50, 1981.

An analysis of the creative work of schizophrenic subjects as a means for personal expression is presented which focuses on 94 paintings—22 by professional artists, 52 by amateurs, and 20 by patients in a psychiatric ward. The analysis distinguishes between classical themes such as scenic paintings, portraits, still lifes, and abstract paintings and paintings in which pathological distortions dominate. The type of expression in the paintings is also subdivided into three categories: direct communicative, metaphoric, and hidden expression. Results indicate that the hospitalized patients have the weakest development of verbal expression. It is suggested that art therapy may facilitate contact with the environment and may permit expression of feelings. (8 references)

6579. Watson, C.G.; Kucala, T.; Angulski, G.; and Brunn, C. (Research Service, VA Medical Center, St. Cloud, MN 65301) Season of birth and schizophrenia: A response to the Lewis and Griffin critique. Journal of Abnormal Psychology, 91(2):120-125, 1982.

A response is presented to the Lewis and Griffin (1981) critique of research describing seasonality effects in schizophrenia. They have argued that this effect may be an artifact resulting from the use of inappropriate expected values in χ^2 calculations in previous studies, the age incidence effect, and the age prevalence effect. The birth months of 3,556 schizophrenics at Minnesota Veterans Administration Hospital were examined before and after correcting for year-to-year across-month variations in birth rates in the expected values and the age prevalence bias toward the January/March seasonality effect. Finally, the data were reanalyzed on a subset of patients in whom the age incidence effect should be minimal. Even after these corrections, results support the contention that the winter birth rate of schizophrenics is excessive, at least in severe climates. (18 references)—Author abstract, modified.

6580. Zipper, M.M. (University of Missouri-Columbia, Columbia, MO 64110) Social stress and autism in schizophrenia. Dissertation Abstracts International, 42(8):3451-B, 1982.

The relationship between interpersonal intensity and autistic thinking in schizophrenic subjects was examined as a function of the level of subjective stress. Results indicate that schizophrenic cognitive deficit is exacerbated under conditions of increased threat of interpersonal intimacy. Schizophrenic subjects of both high and low stress conditions failed to demonstrate any noteworthy differences in their self-disclosure regardless of the type of self-disclosure models. In contrast, nonpsychotic psychiatric patients demonstrated little psychopathology across experimental conditions.—Journal abstract, modified.

Diagnosis

6581. Ban, T.A. (Tennessee Neuropsychiatric Institute, 1501 Murfreesboro Rd., Nashville, TN 37217) Chronic schizophrenias: A guide to Leonhard's classification. Comprehensive Psychiatry, 23(2):155-169, 1982.

The development of the concept of schizophrenia is reviewed along with the clinical importance of understanding its various manifestations. It is pointed out that of the three systematic schizophrenias, only systematic catatonias have shown a considerable decrease during the past 25 years. It is concluded that classifying patients on the basis of Leonhard's criteria corresponds with clinical experience and provides seemingly homogeneous populations, a prerequisite to meaningful research to understand and treat the various disorders subsumed under schizophrenia. (39 references)—Author abstract, modified.

6582. Blumenthal, S.L. (California School of Professional Psychology, Berkley, CA 94701) The use of Rorschach thematic content analysis for differential diagnosis. Dissertation Abstracts International, 42(6): 2516-B, 1981.

Rorschach protocols for 65 males who had been inpatients at one of two hospitals were used to identify affective and psychodynamic themes evident in Rorschach content. It was hoped that identification of affective themes could be a diagnostic aid. Major focus was on the hypotheses regarding the more specific subcatagories between the diagnostic groups—paranoid schizophrenic, manic-depressive, and schizoaffective patients. The Affective Inferences Scale was a useful diagnostic instrument when used conservatively in conjunction with structural features of the Rorschach, other tests, and other individual variables. The instrument's main value was its sensitivity to thematic concerns which might be unconscious and conflictory in nature. Evidence for the schizoaffective diagnosis as a blend of two polar conditions was ambiguous. It is recommended that future investigators form diagnostic groups from discrete selection criteria based on symptomatic and case history variables.—Journal abstract, modified.

6583. Bobon, D.P., and Mendlewicz, J. (Clinique psychiatrique universitaire de Liege, Liege, Belgium) Preliminary results of the French adaptation of Montgomery's Schizophrenia Scale. Feuillets Psychiatriques de Liege, 14(4):477-486, 1981.

A preliminary French version of the Montgomery Schizophrenia Scale, a 12-item subscale of the Comprehensive Psychopathological Rating Scale, is discussed. In the proposed French translation, the items and degree of intensity of symptoms were translated rather literally, but the headings were not. Administration of this scale to four delusional subjects before and 3 weeks after treatment with zetidoline showed that the scale is sensitive to neuroleptic use. A copy of the French version of the scale is included. (6 references)

6584. Csemy, L. (Sasankova 2659, 106 00 Praha 10, Czechoslovakia) Indicators of preceptocognitive decline in the Rorschach method. Ceskoslovenska Psychologie, 25(4):330-338, 1981.

A less traditional use of the Rorschach method, a quantitative approach to measuring the perceptocognitive level, was investigated. Five perceptocognitive indexes and two scoring systems of organic psychosis were used. The procedure was verified with three groups (neurotics, schizophrenics, organic psychotics) of 10 persons each. Highly significant differences in the perceptocognitive level were found between neurotics and organic psychotics, and between neurotics and schizophrenics. No significant differences in the perceptocognitive level were found between organic psychotics and schizophrenics. Some difference was found in the symptoms of organic psychosis. This suggests that the decrease in the perceptocognitive level in both groups is different in quality. Attention is directed to the differential diagnostic contribution of the indicators. The procedure is recommended especially for use in clinical practice, because it allows the study of important integrational and regulatory mechanisms of personality. (12 references)—Journal abstract, modified.

6585. Halkias, D.E. (United States International University, San Diego, CA 92106) Exploring differing vantage points on schizophrenia: A survey on the consistency of behavioral cues and clinical usage of psychodiagnostic label among California psychologists. Dissertation Abstracts International, 42(8):3423-B, 1982.

A descriptive survey of California psychologists was conducted to examine the behavioral cues they use to diagnose schizophrenia, and their clinical usage of this psychodiagnostic label. Of 350 psychologists contacted, 108 (31 percent) responded. Results indicate that not one of the behavioral cues reached a consensus rate of 40 percent; nor did one definition of schizophrenia reach a favorable rate of consensus. The probability of obtaining a population consensus was computed from the normal curve error based on the foregoing parameters. While there was higher consensus for behavioral cues and definitions among those having the same specialization and among those having the same theoretical approach, the difference between their level of consensus and that of the sample population was not significant.—Journal abstract, modified.

6586. Mahendra, B. (Dept. of Neurological Sciences, Medical College of St. Bartholomew's Hospital, West Smithfield, London EC1, England) Where have all the catatonics gone? Psychological Medicine, 11(4):669–671, 1981.

The apparent decline in the incidence of catatonic schizophrenia is discussed. The decline is placed as occurring from about the 1930s; i.e., before the introduction of neuroleptics. This decline has been generally attributed to early detection and management, a liberal and humane hospital atmosphere, and increased occupation of the patient. However, it is suggested that the conditions which were studied and described as catatonia included those that ran a basically schizophrenic course with coexisting catatonic features, as well as manic-depressive illnesses with catatonic signs and organic states. It is proposed that the catatonia might have been virally induced. General problems in diagnostics of schizophrenia are considered, such as what are the cardinal features of the disorder and what are epiphenomena which might disappear within a generation. (21 references)

6587. Miller, H.R.; Streiner, D.L.; and Kahgee, S.L. (Dept. of Psychology, Hamilton Psychiatric Hospital, P.O. Box 585, Hamilton, Ontario, Canada L8N 3K7) Use of the Golden-Meehl indicators in the detection of schizoid-taxon membership. Journal of Abnormal Psychology, 91(1):55–60, 1982.

The ability of the seven Minnesota Multiphasic Personality Inventory items identified by Golden and Meehl to indicate schizoid taxon membership was examined. On the basis of a cutting score of four or more items answered in the schizoid direction, 53.1 percent of a group of schizophrenic subjects were identified as taxon members, whereas 70.9 percent of a group of nonschizophrenic, acutely depressed patients were so identified. In contrast, 9.3 percent of a group of nonschizophrenic depressed patients currently in remission were identified as taxon members. Only 33.3 percent of a subgroup of the schizophrenics were consistently identified as taxon members on two testings, whereas 37.8 percent of this group were consistently identified as nontaxon members. The sum of the standard scores for the depression (D), psychasthenia (Pt), schizophrenia (Sc), and social introversion (Si) scales was also unsuccessful in identifying taxon members, regardless of the cutting score used, although it did differentiate the two groups of depressive subjects. The seven items and the sum of the D, Pt, Sc, and Si scales may be reasonable indicators of degree of current psychopathology and emotional distress. (4 references)—Author abstract, modified.

6588. Overall, J.E. (Dept. of Psychiatry and Behavioral Sciences, University of Texas Medical School, Houston, TX 77025) Criteria for the selection of subjects for research in biological psychiatry. In: van Praag, H., ed. Handbook of Biological Psychiatry. Part VI: Practical Application. New York: Marcel Dekker, 1981. pp. 359–391.

Issues in the selection of subjects for biological research in psychiatry are examined in an attempt to develop a conceptual framework within which to view objective diagnostic criteria for mental illness. Current research diagnostic criteria are examined also, and objective procedures for diagnostic classification of schizophrenia and major depressive disorder, based on the Brief Psychiatric History Form and the Brief Psychiatric Rating Scale, are presented. (52 references)

5689. Stephens, J.H.; Astrup, C.; Carpenter, W.T., Jr.; Shaffer, J.W.; and Goldberg, J. (Maryland Psychiatric Research Center, Box 3235, Baltimore, MD 21228) A comparison of nine systems to diagnose schizophrenia. Psychiatry Research, 6(2):127–143, 1982.

The overlap and prognostic value of nine systems for defining schizophrenia were examined using the files of 283 hospitalized patients discharged with a diagnosis of schizophrenia, schizoaffective schizophrenia, or paranoid state, without knowledge of the patient's subsequent history. Kappa values measuring pairwise diagnostic agreement among the nine systems are typically low except among New York Research Diagnostic Criteria (RDC), *DSM-III*, and St. Louis criteria. Long-term followup status is not significantly predicted by Schneider's first rank symptoms, New Haven Schizophrenia Index, or Taylor-Abrams criteria. Diagnosis by means of the other systems, especially the Astrup process/nonprocess distinction, is significantly correlated with followup, but Astrup's process schizophrenia is not operationally defined and could not be expected to be used with the degree of interrater reliability desired by researchers. *DSM-III* schizophrenia has the highest correlation with followup of the operationally defined systems, and is suggested for use by researchers desiring a highly homogeneous, although narrowly defined, population. Investigators wishing to cast a wider net could use a less restrictive system such as the RDC, with or without schizoaffectives included. (38 references)—Author abstract, modified.

6590. van Scheyen, J.D. (Hazelaarslaan 9, Middelburg, The Netherlands) Psychogenic psychosis and religion: Diagnostic observations on psychogenic psychosis and/or schizoaffective psychosis. Tijdschrift voor Psychiatrie, 23(10):590–610, 1981.

It is suggested that in psychogenic (reactive) psychosis vulnerability factors related to the premorbid character may be of more importance than the life events preceding the psychosis. In 12 out of 28 patients with clinical psychogenic psychosis, this vulnerability was related to a typically orthodox Calvinistic attitude. This attitude reflects a discrepancy between a rationalistic dogmatic religious conviction and the subjective religious experience in which anxiety-inducing feelings of existential insecurity can dominate. During the psychotic episode these patients showed a state of excessive emotional disturbance in which anxiety-inducing religious experiences were typical. In a followup study over a period of 4 to 10 years, it appeared that 14 out of 28 patients showed relapses either as psychotic episodes or as depressions which could be diagnosed as unipolar vital depressions. In 9 out of the 28 patients, a family history of affective psychosis could be demonstrated. Further, the reliability of the original diagnosis of psychogenic psychosis seems doubtful, and it is suggested that a number of these patients could have been diagnosed as schizoaffective. (32 references)—Journal abstract, modified.

Epidemiology

6591. Pulver, A.E.; Sawyer, J.W.; and Childs, B. (Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21205) The association between season of birth and the risk for schizophrenia. American Journal of Epidemiology, 114(5):735–749, 1981.

A test was conducted of the hypothesis that season of birth is associated with risk for schizophrenia for subgroups of the schizophrenic populations. Many epidemiologic studies have been conducted to discover factors that might bear on the origins of schizophrenia. In general, the results of these studies have been contradictory; one consistent finding, however, has been an association between season of birth and risk for schizophrenia. The results of regression analyses of data from Monroe County, New York, are reported. All white residents who were reported to have been diagnosed schizophrenic for the first time during the period January 1, 1969 to December 31, 1971, and who were hospitalized at least 1 day in the 5-year period following the first schizophrenic diagnosis formed the group of schizophrenic patients. The population data used for the denominators of the incidence rates consisted of all white residents living in Monroe County as of April 1, 1970. An effect of month of birth on risk for schizophrenia was related to the sex and age of subsets of the whole population.—Author abstract, modified.

The Family

6592. Ammon, G. (Prinz-Friedrich-Leopold-Str., 21, D-1000 Berlin 38, West Germany) Preventive work in psychoanalytical kindergartens—Diagnosing aspects causing schizophrenia in family groups. Dynamische Psychiatrie, 14(4/5):163–178, 1981.

The diagnosis of schizophrenia-causing tendencies in family groups is discussed in terms of preventive work in a psychoanalytical kindergarten with groups of children and parents. Schizophrenogenic families are differentiated from family groups where some aspects or tendencies causing schizophrenia can be observed. Some significant group dynamic and ego structural facets of behavioral patterns which evoke schizophrenic reactions with high probability are presented. (34 references)—Journal abstract, modified.

6593. Schwarz, F. (Forschungsstelle für Psychopathologie und Psychotherapie, Max-Planck-Gesellschaft, Montsalvatstr. 19, D-8000 München 40, West Germany) Family relatives in the psychotherapy of schizophrenic patients. Zeitschrift für Klinische Psychologie und Psychotherapie, 29(3):247–254, 1981.

Dependency and isolation problems that schizophrenics encounter with relatives are considered in relation to individual, group, and family therapy. In individual and group therapy, the therapist becomes familiar with the problems of schizophrenic patients and their relatives by the reports of the patients and by their transference reactions. In family therapy, the patient is confronted directly with his relatives. Contact with the family without a therapeutic frame can occur through letters or phone calls. Independent of the therapeutic setting, the involvement of relatives seems to be a basic factor for a favorable outcome in schizophrenic patients. (40 references)—Journal abstract, modified.

Genetics

6594. Galdi, J.; Rieder, R.O.; Silber, D.; and Bonato, R.R. (Dept. of Psychiatry, University of Cincinnati College of Medicine, Room 7303, MSB, Cincinnati, OH 45267) Genetic factors in the response to neuroleptics in schizophrenia: A psychopharmacogenetic study. Psychological Medicine, 11(4):713–728, 1981.

A psychopharmacogenetic strategy was used to investigate a genetic heterogeneity model of schizophrenia. This model consisted of various genetic subtypes represented by patients classified hypothetically according to the types and genealogical (Mendelian) patterns of illnesses in first-degree relatives. The effect of neuroleptics on these subtypes was tested for evidence of posttreatment responses which discriminated between them. The findings reveal that schizophrenic patients who had depressed relatives tended to exhibit depression and more severe pseudoparkinsonism irrespective of types of neuroleptics and greater remission of paranoid hostility symptoms when treated with neuroleptics of the aliphatic-piperadine type. Schizophrenic patients who had schizophrenic relatives failed to show these responses. The interpretation emphasizes the recognition of these responses as arising from neuroleptic-induced alterations of defective neurological and neurochemical systems underlying this subtype and as pharmacogenetic criteria by which it can be discriminated. (97 references)—Author abstract, modified.

6595. Targum, S.D., and Schulz, S.C. (2141 K. Street, N.W., Suite 704, Washington, DC 20037) Clinical applications of psychiatric genetics. American Journal of Orthopsychiatry, 52(1):45–57, 1982.

The value of psychiatric genetic assessments in clinical practice is demonstrated through discussion of clinical issues pertinent to psychogenetics. Data are presented which suggest that there is a constitutional or temperamental predisposition that is etiologic in the development of the major psychiatric disorders—schizophrenia and affective disorder. It is suggested that psychogenetic evaluations may improve diagnostic and prognostic capabilities, aid in pharmacotherapeutic decisions, and contribute to marital and family planning in instances of familial psychosis. It is concluded that the exclusive use of interpersonal, psychological, or family approaches may be overlooking an important biological element that may facilitate treatment. (60 references)—Author abstract, modified.

High-Risk Studies

6596. Ritzler, B.; Gryll, S.; Kiecolt-Glaser, J.; Jones, F.; and Singer, M. (Psychology Clinic, University of Southern Mississippi, Southern Station, Box 5025, Hattiesburg, MI 39401) Predicting offspring vulnerability to psychopathology from parents' test data. Journal of Personality Assessment, 45(6):600–607, 1981.

Interpretation of parents' psychological test data was used to predict vulnerability to psychopathology of male offspring—4, 7, and 10 years old. Families were chosen for the study according to the previous psychiatric diagnosis of at least one parent. The prediction procedure was styled after methods used in previous studies with adolescent and young adult offspring. Vulnerability was measured by a global rating score obtained from the Rochester Adaptive Behavior Inventory, a scale based on behavioral observations of the behavior of offspring. Evaluation of 11 families at each age level resulted in significantly accurate predictions at the 10-year-old level, but less than significantly accurate predictions at the 7-year-old and 4-year-old levels. These findings have important implications for the family development of schizophrenia and related disorders. (12 references)—Author abstract.

6597. Watt, N.F.; Grubb, T.W.; and Erlenmeyer-Kimling, L. (Dept. of Psychology, University of Denver, 2460 S. Vine St., Denver, CO 80208) Social, emotional, and intellectual behavior at school among children at high risk for schizophrenia. Journal of Consulting and Clinical Psychology, 50(2):171–181, 1982.

The school behavior of 44 children of schizophrenic parents and 70 children of normal parents was rated by classroom teachers unaware of parental disorder. Subjects were 12 to 17 years old. As compared to controls, children of schizophrenic parents showed greater interpersonal disharmony, less scholastic motivation, more emotional instability, and lower intelligence, but no difference in introversion. Two of the four children with two schizophrenic parents had extremely low ratings for scholastic motivation, harmony, emotional stability, and intelligence, but neither was extremely introverted. It is tentatively concluded that disharmony, emotional instability, and low intelligence are the early markers for vulnerability to schizophrenia. Introversion may best be construed, not as a static preschizophrenic characteristic, but as a dynamic phase in a process of coping and retreat that precedes schizophrenic breakdown, sometimes by many years. (19 references)—Author abstract, modified.

Historical

6598. Gershon, E.S. (Section on Psychogenetics, Biological Psychiatry Branch, NIMH, Bldg. 10, Room 3N218, Bethesda, MD 20205) The historical context of Franz Kallmann and pschiatric genetics. Archiv für Psychiatrie und Nervenkrankheiten, 229(4):273-276, 1981.

The pioneering contributions of Franz Kallmann to psychiatric genetics, especially to the genetics of schizophrenia, are discussed Much of Kallmann's work was conducted during the Nazi period in German history, which reflected a racial interpretation of the inherited nature of man. Upon emigrating to the United States in 1936, his major work involved a family study of schizophrenia in 13,581 relatives of 1,087 patients admitted to a Berlin hospital in a 10-year period. He was also the first to identify increased mortality in relatives of schizophrenics, particularly from suicide, and the results of his classic twin studies of schizophrenia and manic-depressive illness in New York State hospitals still provide important support for the genetic hypothesis in these disorders. It is contended that Kallmann's work did not achieve immediate, widespread acceptance in American psychiatry, partly due to the tide of opinion against genetic research and applications in psychiatry in Nazi Germany. (13 references)

Overview

6599. Manschreck, T.C. (Dept. of Psychiatry, Massachusetts General Hospital, Fruit Street, Boston, MA 02114) Current concepts in psychiatry: Schizophrenic disorders. New England Journal of Medicine, 305(27):1628-1632, 1981.

An overview of schizophrenic disorders is presented, with a focus on *DSM-III* advances in definition and diagnosis. The *DSM-III* diagnostic criteria reflect several strategic changes in psychiatric thinking. There is an explicit view of schizophrenia as an idiopathic syndrome with heterogeneous causes; there is a more balanced emphasis on symptoms and course of illness factors; the criteria narrow the previously defined boundaries of schizophrenia; the requirement that affective and organic mental disorders be ruled out makes schizophrenia a diagnosis of exclusion; and traditional phenomenologic subtypes (e.g., paranoid, catatonic, hebephrenic) have been deemphasized because of a lack of validation. Diagnostic criteria for schizophrenic disorder are outlined, and characteristics of the prodromal, active, and residual phases of the disorder are delineated. Course and prognosis of schizophrenia are reviewed; and etiology is discussed. Although the cause of schizophrenia remains obscure, clinical, genetic, epidemiologic, and biochemical investigations provide evidence consistent with the concept of schizophrenia as a disease. Pharmacologic, psychotherapeutic, and behavioral treatments for schizophrenia are summarized. (37 references)

Psychological Theory

6600. Billeter, W. (Analytisches Zentrum, Ehntaler Str. 249, CH-8046 Zurich, Switzerland) The bodily expression of the pathogenic group dynamics in schizophrenia—Diagnosis and therapy. Dynamische Psychiatrie, 14(4/5):277-294, 1981.

The diagnosis and therapy of the bodily expression of pathogenic group dynamics in schizophrenia is discussed in terms of Shamanism and the cathexis of the body scheme. The cathexis of the body scheme can be measured quantitatively as the static electrical charge or as the local resistance of the skin. Thus, it is possible to measure psychic structure beyond the actual symptomatology. The therapeutic program comprises group psychotherapy, individual psychotherapy, and somatic therapy including methods derived from Shamanism, like breathing methods, motion with music, and riding. This enables integration of the disintegrated body. This integration is an aspect of identity development and depends on a reparative group such as a family or community.—Journal abstract, modified.

6601. Jarosz, M. (Instytut Chorob Ukladu Nerwowego AM, ul. Narutowicza 96, K1. B, 90-141 Lodz,

Poland) The pros and cons of the "Pracoxgefuhl": Not intuition but diagnosis. Psychiatria Polska, 15(2):167-172, 1981.

An overview of arguments for and against the Pracoxgefuhl theory, or early premonition of schizophrenia, is presented, focusing on the nature of the feeling as a means of establishing an early diagnosis of schizophrenia. It is suggested that instead of focusing on the intuitive aspects, it is more valuable to establish and observe symptoms that may indicate the early onset of schizophrenia. At the same time, it is important to remember that many of the early symptoms of schizophrenia, such as memory loss, divergent thought patterns, anxiety, an inclination to magical and exaggerated thought processes, and other forms of eccentrism, may surface at times in healthy individuals. For this reason, all initial symptoms should be analyzed carefully, and the Pracoxgefuhl should be considered as a hypothesis that needs verification. (16 references)

6602. Jarosz, M. (Instytut Chorob Ukladu Nerwowego Am, ul. Narutowicza 96, K1. B, 90-141 Lodz, Poland) Early premonition of schizophrenia. Psychiatria Polska, 15(2):173-178, 1981.

The early premonition of schizophrenia, or *Pracoxgefuhl*, is discussed in light of the literature on the characteristics of true schizophrenia. Psychiatry has been unable to designate specific schizophrenic symptoms clearly since many of schizophrenia's characteristic symptoms may also appear in other syndromes, and often appear in healthy individuals. In particular, Rumke's work on the intuitive diagnosis of schizophrenia is described. Although Rumke felt that the Pracoxgefuhl was a valuable instrument in diagnosing schizophrenia in a patient, it is suggested that an inexperienced psychiatrist may not be able to verify the symptoms under consideration. (12 references)

6603. Kodandaram, P.; Vinutha, M.A.; and Sathyavathi, K. (Dept. of Clinical Psychology, National Institute of Mental Health and Neurosciences, Bangalore 560029, India) Social identity among criminals, mental patients and normals. Indian Journal of Clinical Psychology, 8(2):137-140, 1981.

The social/psychological role theory model of Sarbin (1970) for understanding behavioral dysfunctions was experimentally examined. Sarbin's three-dimensional model of conduct asserts that social identity can be assessed by evaluating both a person's status and the degree of involvement in the role enacted. Consequently, a person with dysfunctional conduct is a person who has lost his or her social identity. Sarbin's Conduct Impairment Scale was administered to 30 subjects in each of four groups: criminals, schizophrenics, neurotics, and normal subjects. Results show that criminals and mental patients differed significantly from normal subjects on test scores. (7 references)—Author abstract, modified.

6604. Lang, H. (Psychosomatische Klinik, Universitat Heidelberg, Landfriedstr. 12, D-6900 Heidelberg 1, West Germany) The relationship between obsession and schizophrenia. Nervenarzt, 52(11):643-648, 1981.

The relationship between obsession and schizophrenia is discussed based on three cases. The need to establish research limits is explored, particularly when the symptoms themselves serve as reminders of cognitive disturbances in schizophrenia. This controversial differentiation is discussed against the background of a brief overview of the literature in the field. Based on this overview, a causal relationship for a possible reciprocal effect between obsession and schizophrenia is developed. It appears that the manifestation of obsessive phenomena in schizophrenia is less a defense of overly strong motivations and more an existential attempt to counteract psychotic personality disintegration. (37 references)—Journal abstract, modified.

6605. Smith, D.B. (University of Massachusetts, Amherst, MA 01002) An interactional model of psychosis and schizophrenia. Dissertation Abstracts International, 42(8):3443-B, 1982.

A model of psychosis and schizophrenia, based primarily on symbolic interaction theory, is developed. The development of dialogic consciousness, defined as an internalized communicative process, is discussed, and problematic processes in the developmental sequence are delineated. Prelinguistic and linguistic experience are considered. Psychosis is defined as the disintegration of dialogic consciousness, and the emergence of nondialogically organized interactive phenomena into consciousness. Schizophrenia is defined as a particular way to construct reality that both increases vulnerability to psychosis and determines the content of what nondialogically organized phenomena emerge in psychosis. The schizophrenic drama is viewed primarily as a struggle to remain related to social processes in some way that protects the person from psychological destruction within that social process.—Journal abstract, modified.

Treatment

6606. Abuzzahab, F.S., Sr., and Zimmerman, R.L. (Clinical Psychopharmacology Consultants, PA, Suite 818 Fairview, 606 24th Avenue South, Minneapolis, MN 55454) Psychopharmacological correlates of post-psychotic depression: A double-blind investigation of haloperidol vs. thiothixene in outpatient schizophrenia. Journal of Clinical Psychiatry, 43(3):105–110, 1982.

A 24-week double-blind study was conducted to compare haloperidol and thiothixene for efficacy and safety in 46 schizophrenic outpatients. Results of global evaluations suggest that haloperidol produced slightly more rapid relief of symptoms than did thiothixene. The inclusion of depression scales was useful in following patients who exhibited depressive symptoms; clinically significant depression was seen in five patients receiving haloperidol and three receiving thiothixene. A high incidence of akathisia in the thiothixene group was responsible for a statistically significant difference between groups in the number of central nervous system symptoms. Mean doses of test drugs were 17.5 mg/day for haloperidol and 31.8 mg/day for thiothixene. The study shows that haloperidol was equal to and in some parameters superior to thiothixene in improving the symptoms of psychosis. (17 references)—Author abstract, modified.

6607. Bomba, J.; Badura, W.; Mamrot, E.; and Orwid, M. (Dept. of Child and Adolescent Psychiatry, Academy of Medicine, ul. Kopernika 21, 31-501 Krakow, Poland) Results of psychotherapy in adolescent schizophrenia. Dynamische Psychiatrie, 14(6):345–354, 1981.

Sixty-six adolescents suffering from schizophrenia were asked to complete a questionnaire when released from the hospital and again 5 years after their release from the Adolescent Unit, Department of Psychiatry in the Academy of Medicine, Krakow, Poland. The workings of the therapeutic unit and the treatment goals are explained. Patients' progress was assessed using the Turku Schizophrenia Assessment Form and the Modified Followup Form. It was found that most patients showed either no symptoms or some symptomatic improvement over the 5-year period. It is concluded that the emotional and social development of adolescent schizophrenics was not arrested by the patients' illness. (5 references)

6608. Carleton, J.L. (World Association for Social Psychiatry, 2323 Oak Park Lane, Santa Barbara, CA 93105) Therapeutic dependency in the treatment of schizophrenia. Dynamische Psychiatrie, 14(4/5):202–232, 1981.

The concept of therapeutic dependency as a form of treatment is presented. It is suggested that successful dynamic therapy with schizophrenics is dependent upon the nature of the interpersonal relationships which can be created between the patient and his therapeutic groups and with his therapist. The schizophrenic patient's needs for physical, emotional, and intellectual contact and involvement with other people are the same as those of any other person. The psychopathology of the schizophrenic patient creates special obstacles to the development of mutuality with others. Therapy must undo the damage from early childhood experience and motivate the reconstruction of adequate ego structures and processes. This can only be done in a social therapeutic environment where normal physical, emotional, and intellectual experiences are possible. (20 references)

6609. Costa, J.-G.; Silva, E.; and Dreyfus, J.-F. (Silva: Faculté de Sciences Medicales, Université d'Etat de Rio de Janeiro, Rio de Janeiro, Brazil) Clinical evaluation of pipotiazine and its palmitic ester in the treatment of schizophrenia. Annales Medico-Psychologiques, 139(4):439–445, 1981.

The effectiveness of pipotiazine and of pipotiazine palmitate in the treatment of schizophrenia was evaluated clinically. Twenty chronic schizophrenic inpatients hospitalized for an acute attack were given oral pipotiazine in doses of 10 to 20 mg/day. After their release, they received a monthly injection of 80 mg pipotiazine palmitate. Subjects were evaluated on the basis of a physical evaluation and the Brief Psychiatric Rating Scale. Results show that the oral pipotiazine controlled symptoms beginning with the second week and had the greatest effect on conceptual disorganization, thought disorders, anxiety, depression, and tension. The injections not only prevented relapses but also produced further improvement. Side effects were minimal and easily controlled. It is concluded that pipotiazine would be especially useful in countries where the psychiatric infrastructure is still weak. (11 references)—Journal abstract, modified.

6610. Erdreich, M. (Government Mental Health Center, P.O. Box 9, Tirat Carmel, Haifa, Israel) Psychotherapeutic journeys with schizophrenic patients living in the community. Dynamische Psychiatrie, 14(4/5):233-249, 1981.

Psychotherapy for schizophrenics living in the community, after having passed the acute stage in the hospital, is discussed in terms of providing various support systems for the schizophrenic which help him to cope with his problems. A case study of a patient treated in the community is discussed, as is a pilot study of the effects of psychotherapy on schizophrenic patients. (3 references)

6611. Freedman, R.; Kirch, D.; Bell, J.; Adler, L.E.; Pecevich, M.; Pachtman, E.; and Denver, P. (Depts. of Psychiatry and Pharmacology, University of Colorado Health Sciences Center, 4200 E. Ninth Avenue, Denver, CO 80262) Clonidine treatment of schizophrenia: Double-blind comparison to placebo and neuroleptic drugs. Acta Psychiatrica Scandinavica, 65(1):35-45, 1982.

Antipsychotic effects of clonidine were evaluated in one schizoaffective and seven schizophrenic patients, using a double-blind, crossover design to compare placebo, clonidine, and standard neuroleptic drugs. Mean improvement on clonidine and neuroleptics was equal, and improvement scores on the two treatments were closely correlated for individual patients. Clonidine was selected because it blocks noradrenergic but not dopaminergic neurotransmission. Patients were selected because of coexisting psychosis and tardive dyskinesia. For all patients, tardive dyskinesia improved when neuroleptics were discontinued during clonidine and placebo periods of the study. The data provide preliminary evidence that clonidine may be an effective alternative to neuroleptics, particularly for patients for whom the dopaminergic blocking action of the neuroleptics is undesirable. (27 references)—Author abstract.

6612. Gershon, S.; Sakalis, G.; and Bowers, P.A. (Dept. of Psychiatry, Wayne State University, 951 East Lafayette, Detroit, MI 48207) Mesoridazine—A pharmacodynamic and pharmacokinetic profile. Journal of Clinical Psychiatry, 42(12): 463-469, 1981.

Data on the clinical effectiveness and side effects of mesoridazine are reviewed, and an attempt is made to relate clinical efficacy to phenothiazine pharmacokinetics. The published results of 21 controlled clinical studies comparing mesoridazine with a reference drug or placebo are summarized. Both controlled and open-labeled clinical studies have attested to the efficacy of mesoridazine in schizophrenia, as well as certain other psychiatric disorders. Clinical observations of the effectiveness of mesoridazine in patients refractory to treatment with thioridazine and other neuroleptics may be related to its slow rate of inactivation and to the relatively large proportion of free mesoridazine that is available for penetration to the target sites in the brain. (84 references)—Author abstract, modified.

6613. Gift, T.E., and Gift, J.M. (Dept. of Psychiatry, Strong Memorial Hospital, 300 Crittenden Boulevard, Rochester, NY 14642) Depot fluphenazine and depression: Possible mechanism. Journal of Psychiatric Treatment and Evaluation, 3(6):557-558, 1981.

A modest number of cases have been reported linking fluphenazine with depression. Two cases are presented which suggest that depot administration of fluphenazine prevents the use of psychotic regression, otherwise facilitated by discontinuing oral neuroleptics, as a means of escaping from painful depressive affect. Implications of this for psychotherapy and problems of noncompliance are discussed with respect to the treatment of certain schizophrenic and schizoaffective patients. (12 references)—Author abstract, modified.

6614. Joshi, V.G.; Eswaran, S.; Nagesh, R.; Pai, M.; Mathews, G.; and Mallick, P.N. (7-Jutica Makarand Society, Sawakar, Marg, Bombay 400 016, India) Vitamins B_1, B_6, and B_{12} in the adjunctive treatment of schizophrenia—Further studies to examine the effect of reduction of chlorpromazine dosage. Journal of Orthomolecular Psychiatry, 11(1):45-49, 1982.

A double-blind, placebo-controlled randomized study explored the possibility of reducing the drug doses of acute schizophrenic patients. Patients were given placebo or chlorpromazine with an injection of Macraberin Glaxo containing 100 mg vitamin B_1, 1,000 μg vitamin B_{12}, and 50 mg vitamin B_6. It was found that as the daily dose of chlorpromazine was reduced, recovery in the vitamin therapy group was retarded to a greater extent than in the placebo group. The difference between recovery rates in both groups became statistically significant when the daily chlorpromazine dose was at a subtherapeutic level of less than 150 mg per day. Possible explanations of the experimental results are proposed. It is hypothesized that the vitamins help in the recovery process by decreasing the supersensitivity of postsynaptic neurons to a more normal level. (5 references)—Author abstract, modified.

6615. Kon, A., and Gaoni, B. (Shalvata Hospital, Hod-Hasharon, P.O.B. 94, Tel-Aviv, Israel) Dynamic aspects of insulin therapy. Evolution Psychiatrique, 46(2):473–487, 1981.

A new look at insulin therapy for schizophrenics is presented. Sakel's insulin therapy is now considered outdated; however, when insulin therapy is used in conjunction with psychotherapy, there have been surprising results in some cases of schizophrenia. In a group of 18 young schizophrenics who were unresponsive to standard neuroleptic treatment and electroshock therapy, a combination of insulin therapy and psychotherapy resulted in 12 remissions and 3 cases of improvement 3 years after treatment. It is noted, however, that results were less impressive immediately upon completion of treatment; the effects were often delayed. (21 references)—Journal abstract, modified.

6616. Lange, E.; Konig, L., and Eymann, B. (Medizinische Akademie "Carl Gustav Carus," Fetscherstr. 74, DDR-8019 Dresden, German Democratic Republic) Peculiarities and difficulties in the systematic use of drugs in the treatment of chronic schizophrenics. Psychiatrie, Neurologie und Medizinische Psychologie, 33(1):34–38, 1981.

Difficulties encountered in the treatment of chronic schizophrenic psychoses with various syndromes were investigated. There is a crisis in psychopharmacology involving the many new products constantly being introduced into the market. This creates specific problems for those with schizophrenic psychoses. Ten defect schizophrenics, average age 42 and hospitalized for 13 years, were treated with carpipramin for 3 months. Six of the subjects showed increased drive and spontaneous behavior, but those treated with a higher dosage experienced an outbreak of latent psychotic symptoms. More specific knowledge with regard to the pathological and psychodynamic course of schizophrenic defects is needed. It is concluded that hospital personnel needs to be increased and reeducated.—Journal abstract, modified.

6617. Mandel, M.R.; Severe, J.B.; Schooler, N.R.; Gelenberg, A.J.; and Mieske, M. (Schooler: National Institute of Mental Health, 5600 Fishers Ln., Room 10C06, Rockville, MD 20857) Development and prediction of postpsychotic depression in neuroleptic-treated schizophrenics. Archives of General Psychiatry, 39(2):197–203, 1982.

Data were collected during a 1-year multicenter collaborative trial comparing short-acting fluphenazine hydrochloride with long-acting fluphenazine decanoate in a group of schizophrenic patients being maintained in the community. The emergence and course of postpsychotic depressive symtomatology, operationally defined by the Hamilton Depression Scale total score, were studied. About 25 percent of the 211 patients had developed depression within 5 months after discharge. Depressed subjects had a more chronic psychiatric history and, contrary to the hypothesis that depression is a favorable prognostic indicator, they were more likely to relapse. There were no differences in incidence of emergent depression between the two neuroleptic-treated groups. (40 references)—Author abstract, modified.

6618. Morales, E.; Levy, V.; and Schilkrut, R. (Servicio de Psicofarmacologia Clinica, Hospital "El Peral," Facultad de Medicina Sur, Universidad de Chile, Santiago, Chile) Evaluation of a prolonged action neuroleptic: Penfluridol—Clinical and psychosocial evolution in a 1-year followup. Acta Psiquiatrica y Psicologica de America Latina, 27(3):186–193, 1981.

Twenty schizophrenic patients treated for 1 year with penfluridol were evaluated clinically and psychologically to determine the drug's antipsychotic and resocializing effect. Interviews and home visits allowed investigators to rank patients on a series of specially designed scales. A reduction in the treatment program dropout rate was observed. Patients showed gradual but steady improvement on the clinical social questionnaire. Significant improvement was seen in social readjustment. The majority of patients resumed a satisfactory level of activity as a result of treatment. Although the family dynamic was judged pathological in 80 percent of the cases studied, patients in over half of these families were seen to enjoy improved family status as a result of treatment. The importance of treating the family as well as the patient is emphasized. (16 references)—Journal abstract, modified.

6619. Nahunek, K.; Svestka, J.; and Ceskova, E. (Psychiatricka klinika LF UJEP, Jihlavska 102, 615 07 Brno-Bohunice, Czechoslovakia) On the

question of differences between therapeutic responses after particular neuroleptics in comparison to perphenazine in schizophrenics. Ceskoslovenska Psychiatrie, 77(1):25-30, 1981.

The frequency of atypical therapeutic response by schizophrenics was studied using 15 neuroleptics, with perphenazine as a referent substance. In the majority of patients, the effects of most of the neuroleptics overlapped with perphenazine with only small quantitative differences. The differences ranged between 0 percent to 12.5 percent. Within this range, a certain specificity of effect in a small number of patients might be conceivable, especially in the following neuroleptics: flupenthixol, clozapine, clorotepine, oxyprothepine, methophenazine, thiothixine, and triperidol. No difference was found between perphenazine and levomepromazine, or between chlorpromazine and clothiapine.—Journal abstract, modified.

6620. Petrie, W.M.; Ban, T.A.; and Ananth, J.V. (Tennessee Neuropsychiatric Institute, 1501 Murfreesboro Rd., Nashville, TN 37217) The use of nicotinic acid and pyridoxine in the treatment of schizophrenia. International Pharmacopsychiatry, 16(4):245-250, 1981.

The hypothesis that combined administration of nicotinic acid and pyridoxine has greater therapeutic effects than the component drugs in chronic schizophrenic patients was tested. This could not be substantiated in a 48-week study in which supplementation of neuroleptic treatment with a single vitamin, i.e., nicotinic acid or pyridoxine, produced significant therapeutic changes, while supplementation with both vitamins did not. (9 references)—Author abstract, modified.

6621. Pietzcker, A.; Poppenberg, A.; Schley, J.; and Muller-Oerlinghausen, B. (Dept. of Psychiatry, Free University of Berlin, Eschenallee 3, D-1000 Berlin 19, West Germany) Outcome and risks of ultra-long-term treatment with an oral neuroleptic drug: Relationship between perazine serum levels and clinical variables in schizophrenic outpatients. Archiv für Psychiatrie und Nervenkrankheiten, 229(4):315-329, 1981.

Evidence is presented from 33 schizophrenic outpatients treated continuously with perazine over 20 years. The intensity of psychopathologic symptoms and side effects was very low. The high intraindividual constancy of perazine plasma levels and the tight correlation between dose and plasma levels indicated satisfactory patient compliance. Plasma levels amounted to only 25 percent of those under acute treatment and correlated positively with the severity of the disease. Higher plasma levels coincided with more frequent side effects, such as slightly pathologic liver function and moderate impairment of oral glucose tolerance. Results suggest that low dose maintenance treatment of schizophrenic patients with oral neuroleptics is effective and relatively safe. (50 references)—Journal abstract.

6622. Populin, M.; De Vanna, M.; and Poldrugo, F. (Clinica Psichiatria, Università di Trieste, Trieste, Italy) Fluphenazine decanoate in the outpatient treatment of schizophrenic patients. Minerva Psichiatrica, 22(2):85-92, 1981.

The clinical effects of long-term treatment with fluphenazine decanoate on 75 chronic schizophrenic outpatients were assessed. Good therapeutic results were achieved in 78.6 percent of the cases. A significant reduction in relapses was observed, compared to the period preceding treatment. The most frequently observed side effects were asthenia and somnolence (60 percent), parkinsonism (56 percent), weight gain (44 percent), and akathisia (36 percent). Extrapyramidal side effects were relieved quickly by administration of antiparkinson drugs. The necessity for tailoring the dose and administration of long-term drugs to each patient is emphasized, with the aim of avoiding interfering side effects. (74 references)—Journal abstract, modified.

6623. Rhoades, L.J. (NIMH, 5600 Fishers Lane, Rockville, MD 20857) Treating and assessing the chronically mentally ill: The pioneering research of Gordon L. Paul. NIMH Science Reports Series, Washington, DHHS Pub. No. (ADM) 81-1100, 1981. 65 pp.

The work of Paul and his colleagues in developing a comprehensive social learning program for treating chronic mental patients and a comprehensive treatment assessment technology is described. During a 6-year study of the differential effectiveness and efficacy of three treatment approaches (social learning therapy, milieu therapy, and traditional hospital treatment), the researchers found social learning therapy to be the treatment of choice for severely disabled patients. The research subjects, on the average, had been hospitalized for 17 years before the study and had been rejected for community placement. Subjects had diagnoses of schizophrenia and bleak prospects; yet, they responded particularly well to psychosocial interventions developed by Paul, especially the social learning program. Social learning and milieu therapy were noteworthy for additional reasons: the use of drugs was substantially reduced or eliminated; almost all of the treatment in the psychosocial programs was administered by high school equivalent aides trained in the programs; a further reduction in costs may be possible; the measures of patient functioning were able to predict both release success and subsequent level of functioning maintained in the community; and two of the observational instruments developed for the project were subsequently tested on 36 programs. (81 references)

6624. Rosen, A.J.; Tureff, S.E.; Lyons, J.S.; and Davis, J.M. (Dept. of Psychology, University of Illinois, Chicago Circle, Box 4348, Chicago, IL 60608) Pharmacotherapy of schizophrenia and affective disorders: Behavioral assessment of psychiatric medications. Journal of Behavioral Assessment, 3(2):133-148, 1981.

Ward behaviors (body activity, extremity activity, scanning, social interaction, proximity, participation, laughing/smiling, and idiosyncratic behavior) emitted by psychiatric inpatients with either schizophrenic or affective disorders were time sampled both before and during the administration of psychiatric medications. The data indicate that the primary effects of the pharmacological interventions are confined to activity measures and symptoms rather than social behaviors. The implications of these results for treatment protocols are discussed in terms of interactions between pharmacological and psychosocial interventions. In addition, rates of behavior during treatment were related to baseline rates via log/log function of negative slope, a result that is consistent with data derived from the infrahuman laboratories. These results provide support for attempts to relate preclinical and clinical psychopharmacology, and suggest that behavioral assessment can be applied profitably to drug effects in clinical situations. (34 references)—Author abstract.

6625. Seeman, M.V., and McGee, H. (Clarke Institute of Psychiatry, 250 College Street, Toronto, Ontario, M5T 1R8, Canada) Treating depression in schizophrenic patients. American Journal of Psychotherapy, 36(1):14-22, 1982.

Various ways in which depressive symptoms can be understood in the context of schizophrenic illness are described. Despite careful clinical observation, the distinction between schizoaffective illness, schizophrenic prodrome, schizophrenic resolution phase, depression of insight, reactive depression, and depression secondary to neuroleptics is not easy to make. Successful intervention in the depressive reactions of schizophrenic patients depends on accurate diagnosis, careful observation, the use of many modalities of treatment, and the maintenance of therapist optimism. Therapy needs to be pragmatic and focused on the special needs of the individual patient. (23 references)—Author abstract, modified.

6626. Seidel, M.; Ernst, K.; and Lindenau, K. (Bereich Medizin (Charite), Humboldt-Universitat, Schumannstr. 20/21, DDR-1040 Berlin, German Democratic Republic) The state of extracorporeal detoxication procedures as therapy methods in schizophrenia. Psychiatrie, Neurologie und Medizinische Psychologie, 33(12):705-717, 1981.

The literature on the problem of extracorporeal detoxication in chronic schizophrenia is reviewed. In the 132 cases of chronic schizophrenia without uremia treated with hemodialysis, hemoperfusion, and hemofiltration published in the literature, it was found that 38 percent were considered improved. Because of the insufficient comparability of examination methods and the lack of clarity of the specific therapy influence (placebo effect, etc.), the effectiveness of these treatment methods in chronic schizophrenia is still questionable. (55 references)—Journal abstract, modified.

6627. Shopsin, B.A., and Kline, N. (Unit for the Study and Treatment of Affective Disturbances, New York University, Bellevue Medical Center, New York, NY 10003) Treatment of outpatients with high doses of haloperidol. Acta Psiquiatrica y Psicologica de America Latina, 17(3):236-242, 1981.

Thirty-seven patients, the majority of whom were diagnosed as schizophrenic, were treated with haloperidol in doses ranging from 15 mg to 80 mg. The average treatment period was 37 weeks. Six of the patients manifested extrapyramidal symptoms during the course of treatment. The long-term effectiveness of treatment with high doses of haloperidol was established. Caution is urged in the prescription of antiparkinsonian agents. Choice of neuroleptic and dose determination are discussed. The value of treatment with drugs such as haloperidol is found in maintaining the patient in an episode-free state, while the disadvantage lies in the possible harmful side effects of long-term use. Elevated doses of haloperidol are judged effective in maintenance programs for outpatients. (12 references)

6628. Spotnitz, H. (41 Central Park West, New York, NY 10023) Aggression in the therapy of schizophrenia. Modern Psychoanalysis, 6(2):131-140, 1981.

Clinical experience with aggressive schizophrenic patients is described to support the view that schizophrenic psychosis is psychologically reversible on a sustained basis. It is contended that the therapist who works with aggressive schizophrenic patients must employ emotional communication to resolve the patient's resistance to expressing aggression verbally rather than physically. The schizophrenic patient is seen as suffering from both unchanneled aggression and emotional deprivation, and needs to learn slowly how to express aggressive impulses in language. Successful treatment is shown to depend in large measure on the personality of the therapist and his self-analytic ability. (6 references)

6629. Svestka, J.; Bartova, D.; Ceskova, E.; and Rysanek, R. (Psychiatricka klinika LF, CS-657 15 Brno-Bohunice, Czechoslovakia) Depot neuroleptic preparations in psychiatric practice. Ceskoslovenska Psychiatrie, 77(3):187-197, 1981.

Evidence is presented that depot neuroleptics resulted in remission of endogenous, mainly schizophrenic, psychosis in 78 percent of 469 patients. Among these patients, 11.5 percent discontinued treatment and 28 percent were employed during treatment. During administration of depot neuroleptics, 68 percent of 183 patients in the acute stage of psychosis achieved complete or partial remission. In 46 patients, the same depot neuroleptic was used subsequently for maintenance therapy, and in 78 percent of the cases a positive correlation of therapeutic and prophylactic action was recorded. Schematic classification of the neuroleptics according to the affinity to extrapyramidal and actonomous system revealed that extreme positions are held by oleoclorotepine (with the highest) and thioridazine retard (with the lowest) incidence of side effects. Pharmacogenic depressions were observed in 9.5 percent of the treated patients. Administration of depot neuroleptics, particularly oxyprothepine decanoate, proved promising in the treatment of abnormal sexual behavior and of substance addiction. (32 references)—Journal abstract, modified.

6630. Tung-Yep, T. (No address given) The use of orthomolecular therapy in the control of schizophrenia—A study preview. Australian Journal of Clinical Hypnotherapy, 2(2):111-116, 1981.

The effectiveness of megavitamin therapy to treat schizophrenia was investigated. A sample of 485 individuals who had taken the Hoffer Osmond Kelm Diagnostic (HOD) Test were administered Hivita 400 for 4 weeks, complemented by weekly retesting on the HOD test. The results were as follows: 392 people improved their total HOD score by more than 75 percent; 47 persons improved their total score by more than 75 percent when psychological counseling, psychometric analysis, or hypnotherapy was used as an adjunct to megavitamin therapy; 38 people did not improve appreciably; 8 people relapsed. For the 439 people who showed improvement, a 67-month time period on maintenance doses showed that those who discontinued Hivita 400 therapy relapsed completely within a period of 6 weeks, but improved again on starting doses over approximately 2 weeks. (11 references)—Author abstract, modified.

6631. Ungvari, G., and Pethö, B. (Psychiatric Clinic, Semmelweis University, Balassa 6, H-1083 Budapest, Hungary) Treating schizophrenia with large doses of neuroleptics. Psychiatrie, Neurologie und Medizinische Psychologie, 33(4):230-236, 1981.

Results of megadosage therapy with the neuroleptic drug, haloperidol, in the treatment of 30 schizophrenics with acute psychoses are presented. In several years of investigations, patients given an average daily dose of 65 mg of haloperidol for 6 consecutive days showed rapid improvement without dangerous side effects. Haloperidol is recommended in the initial phase of a combined psychotherapeutic program. (21 references)—Journal abstract, modified.

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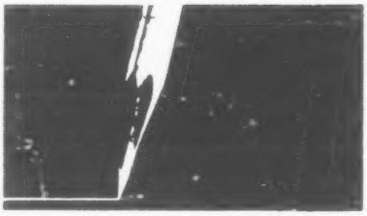

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About the Cover

The *Schizophrenia Bulletin* features art done by current and former mental patients. The drawing that appears on the cover of this issue was sent to us by Pamela Lowenthal-Bowers, art therapist at Crossing Place, Washington, DC. The artist, David Harris, drew this picture outside of his art therapy sessions, yet it is consistent in figure representation, style, and meaning with work done in art therapy. Mr. Harris has provided the following description of the drawing:

When I was sketching this picture, my aim was to produce a beautiful stylized painting. I did not reflect on the composition of the painting until much later. I was feeling peaceful when I painted it and hopeful about the future. The woman is offering a flower to the viewer. The flower represents peace, perhaps peace of mind. The woman is rosy cheeked and full in face, i.e. abundance and youth. There is tranquility in her large clear eyes, and she is not threatening or morose in any way. She faces the viewer full of hopeful innocence but also of resolve.

The Israeli High-Risk Study: Some Critical Remarks

by Mordecai Kaffman

At Issue

Abstract

This article addresses a number of methodological weaknesses in the group comparison design of the Israeli High-Risk Study. It would seem that 25 years ago constraints of experimental design may have led the investigators to select as schizophrenic probands patients who today would no longer qualify for schizophrenic diagnosis according to *DSM-III*. In addition, a series of relevant dimensions (e.g., the course of the assumed schizophrenic process, family variables, life circumstances, and psychosocial factors) were not taken into account. For these reasons, many of the study's hypotheses and conclusions can be considered basically speculative, and it is suggested that many of the findings may be artifacts of the research design. Nor are there any evident grounds for the supposition that the differences between kibbutz and town children, in terms of the nature and severity of particular psychopathological problems, should be decisively ascribed to the differences in social framework and childrearing practices in the kibbutz and in town. The weight of many other variables, in particular intrafamily influences, is no less marked in the kibbutz setting than in town.

A 1985 issue of *Schizophrenia Bulletin* (Vol. 11, No. 1) reports at length on the findings and conclusions of an investigation known as the Israeli High-Risk Study, which was conducted for more than 20 years by a large number of researchers. As Nagler and Mirsky (1985) put it, "The project is an attempt to add some knowledge to the nature-nurture dilemma in the etiology of schizophrenia" (p. 25). In an effort to examine the respective influences of genetic and environmental factors in the development of schizophrenia, the investigators studied a group of 50 Israeli preadolescent, high-risk children who were born and grew up in two different environments: 25 children born in a kibbutz to a schizophrenic parent and 25 children born to a schizophrenic parent in town and raised in the customary family setting. Each index subject was matched with a control kibbutz or town child. The children, 100 in all, were interviewed and thoroughly examined (by psychological, neurological, and electrophysiological examinations) when the mean age of the study population was 11 and then again at age 16. The index group was uniformly rated as more impaired than the control group, with statistically significant differences in severity of psychopathology, quality of social and school adjustment, and presence of neurological "soft" signs. In the first two stages of the followup, which covered a 15-year span, the investigators failed to find any consistent differences between the kibbutz and nonkibbutz rearing environments, in either the subjective or objective measures that were used on the index and control children. On the basis of these findings, the investigators concluded that the higher rate of neuropsychological disturbance in the index children was mainly related to the schizophrenic diathesis and not to environmental influences.

Some years later, however, when the index subjects reached an average age of 25, the investigators—using a test battery that included the Schedule for Affective Disorders and Schizophrenia, Lifetime Version

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Reprint requests should be sent to Dr. M. Kaffman, Medical Director and Chief Psychiatrist, Kibbutz Child and Family Clinic, 147 Derech Haifa, Tel-Aviv, Israel.

(SADS-L) (Spitzer and Endicott 1977)—discovered to their surprise an increased incidence of major psychiatric pathology in the kibbutz index subjects, particularly schizophrenic spectrum cases and affective disorders. In light of this later finding, they hypothesized that "the interaction of the kibbutz environment with a schizophrenic diathesis may have a psychopathogenic effect" (Mirsky et al. 1985, p. 152).

Although the preceding brief summary of some of the findings of this extensive research project—with its great mass of data and information—does not do it justice, for present purposes, I believe the summary gives an adequate picture of the main points of this multidisciplinary study.

Evaluating "Nature": Were the "Schizophrenic Parents" Really Schizophrenic?

For the past 25 years I have served as medical director and chief psychiatrist of the Kibbutz Child and Family Clinic, which provides psychological and psychiatric services for the majority of kibbutzim in Israel. During this time I have had occasion, together with the staff of the clinic, to examine, treat, and follow up a large number of schizophrenic parents and their children in the kibbutz setting. The experience has left me perplexed in the face of the obvious differences between our clinical experience and the findings of the Israeli High-Risk Study. I have read carefully and reread the various papers that make up the study to try to identify and understand properly the source of these differences. The present article summarizes my queries and critical comments on the material that has been published so far on this most impressive study.

The study's authors deserve praise for their outspokenness in describing in detail the methodological difficulties, pitfalls, and limitations they faced more than once during their investigation. Silberman and Mirsky (1985) themselves point out the serious problems in their research design—among others, the geographic separation between the team members, lack of mutual agreement about statistical methodology, small size of the study sample due to the objective difficulties to find schizophrenic subjects, extended age range of the index kibbutz children (over 7 years), etc. In addition to these obstacles, however, the study contains a considerable number of methodological flaws that might place the reliability of the conclusions in question.

One of the study's main limitations lies in the regrettable fact that the psychiatric diagnosis of schizophrenia among the index parents was not made by the researchers themselves, thus leaving some doubt as to its reliability. The investigators depended on the diagnostic labels put on the parents during the 1950's and 1960's by a heterogeneous group of Israeli psychiatrists who volunteered to cooperate with the researchers (after the hospitals and various government bodies, on the grounds of confidentiality, refused to release the registered information). Thus, the index subjects were selected through extramural individual or group meetings with the therapists (doctors, psychologists, social workers and nurses). The researchers could console themselves with the fact that the registered information was faulty and incomplete, anyway, so that "Often memory proved more reliable than stacks of illegible reports in files, written in various languages, according to the psychiatrists' origins" (Nagler 1985, p. 33).

The psychiatrists who made the diagnosis of schizophrenia were working in various hospitals all over Israel. They had no unified diagnostic criteria, and sometimes their ideas on the definition and nature of schizophrenia were divergent. Having worked at that time in several Israeli psychiatric hospitals and having experienced the problem of the lack of a common language for diagnostic assessment, I can only verify the picturesque description of Nagler (1985): "Diagnostic labels such as process schizophrenia, schizophrenic reaction, pseudoneurotic schizophrenia, and borderline schizophrenia reflected the psychiatrist's 'Weltanschauung' rather than the psychic condition of the patients" (p. 33). Along with the psychiatrists working with me in the Kibbutz Clinic, I can testify that in at least one-third of the patients who were labeled schizophrenics by the psychiatric hospitals, and were turned over to us for ambulatory treatment and followup, we found no signs of schizophrenia according to the diagnostic criteria established in the *DSM-III* (American Psychiatric Association 1980) for Schizophrenic Disorders. The researchers of the Israeli High-Risk Study apparently had a good many reservations about the validity and reliability of the past psychiatric diagnoses as well. Therefore, they added two more criteria to strengthen the validity of the original findings: (1) there had to be a record of several rehospitalizations in the anamnesis of the patient; and (2) the patient's medical file had to contain at least 3 symptoms from a checklist of 22 that "by consensus of the research team, were felt to be closely related to the diagnosis of schizophrenia" (p. 33).

Even the addition of these two parameters does not seem sufficient to establish the validity of the schizophrenia diagnosis. First, 25 years ago, the phenomenon of the "revolving door," with a high rate of rehospitalization, was very common not only in schizophrenia but also in a wide range of other diagnostic categories. It was also accepted practice, when a patient was readmitted, for the psychiatrist in charge to stick to the original diagnosis in almost every case. This was especially true if the label of schizophrenia had been attached to the patient, because of the prevailing fatalistic premise "Once a schizophrenic—always a schizophrenic." The additional criterion of finding at least three symptoms common to the patient's medical file and the checklist the researchers devised seems of doubtful value because of the objective difficulties already mentioned in getting at the medical records, and also because of the difficulty in deciphering the multilingual records when they were available. Our chief reservation involves the questionable reliability of the suggested symptom checklist. A careful study of the list shows that at least half the items are not specifically symptoms of schizophrenic disorders. They may indicate other psychiatric conditions, such as affective, paranoid, and nonschizophrenic psychotic disorders. To exemplify the checklist's lack of specificity, 10 of the 22 symptoms that are said to be indicative of schizophrenia are listed here: marked apathy, marked negativism, marked hypobulia, lack of interest in own appearance, lack of interest in other patients, overaggressiveness, concretization, continuous ideas of persecution, megalomania, and no participation in occupational therapy. If the researchers believe that three of the preceding symptoms are sufficient to verify schizophrenia, the validity of the assertion that the group of index parents consists solely of schizophrenics seems open to serious doubt.

Indeed, in so ambitious a research project, one aiming to examine the relative weight of hereditary and environmental factors in the children of schizophrenic parents, the researchers should present more convincing evidence of the validity of the diagnosis of schizophrenia in the index parents' group. Of course, criticism of this nature seems reasonable and self-evident in view of the diagnostic tools at our disposal today. When the study was initiated 25 years ago, however, it was no simple matter to locate a group of pure schizophrenics whose psychiatric diagnosis would be acceptable according to present-day criteria. In addition to the absence of standardized diagnostic criteria for schizophrenia, there was an exaggerated tendency in Israel at that time to arrive at a diagnosis of schizophrenia. Indeed, many different types of paranoid disorders, major affective disorders with delusional features, and cases of organic and atypical psychoses were included under that label. If such was the state of psychiatric diagnosis 25 years ago, all the more reason today for reservations and caution in drawing conclusions from such research. Fifteen years after the initial examination, Mirsky et al. (1985) found that 10 of 46 index cases showed evidence of affective disorders; this seems to be a red light on the validity of the original diagnosis of schizophrenia, at least for some index parents.

Evaluating "Nurture": Kibbutz vs. Nonkibbutz Children

It is difficult to understand why authors of various articles in the study hypothesize that significant differences between kibbutz and nonkibbutz index children in the nature and severity of their psychopathology should be ascribed to the differences in childrearing practices in the two settings. The researchers frequently use the somewhat misleading phrase, "the differential effects of town and kibbutz rearing." There is, of course, a conceptual error at work here; many other factors may affect the development of psychological problems and cause differences in outcome. Kibbutz children, to no lesser a degree than town children, respond to various traumatic events, particularly to stressful family situations (Kaffman 1972; Elizur and Kaffman 1983). It is regrettable that the researchers failed to identify and estimate the specific weight of the multiple variables (other than differences between kibbutz and nonkibbutz methods of upbringing) that may have played a decisive role as pathogenic or corrective influences on children at risk. Before hypothesizing about the particular role of differential childrearing practices, the researchers should have examined with great care whether the index groups (of both parents and children) were well matched in all relevant dimensions and, thus, whether they could really be compared fairly on the presence or absence of major stressful occurrences. While reporting on the deep grief that befell members of the research team at the unexpected death of three of their teammates during the years of research, they should also have been aware of the possibility that the high-risk families under study—both adults and children—may have found themselves in no less stressful situations. Since small groups are involved (25 children each), it is clear that variables relating to different life circumstances and particular traumatic stressors may have played a decisive role in the study's outcome—with a weight much greater than that attached to being brought up in a kibbutz or in town.

Following is a partial list of factors on which the researchers did not report but that may have had a considerable influence on the differences in type and severity of the index children's psychopathology, without regard to their respective social setting:

1. Factors Related to the Index-Parent Pathology. There are no details on the severity of the pathology of the index parent, no appraisal of the premorbid personality or the degree of subsequent deterioration in important areas of psychosocial functioning. Especially obvious is the lack of consideration and assessment of the quality of impairment in the parent-child relationship. There are no details on the course of the mental illness—chronic, subchronic, or in remission—or on the number or length of psychiatric hospitalizations.

2. Factors Related to the Family. There are no comparative data between the index groups with regard to the integrity of the family unit and the respective rates of divorce and marital separation. The researchers do state that, at the beginning of the study, the parents were separated or divorced in 20 percent of the index families, but there is no information on the differences between the kibbutz families and the town families on this important variable or on the changes that took place over time. Consequently, we know nothing about the proportions of one-parent families, remarriages, and blended families. Nor was any attempt made to determine the possible presence of other sources of family stress or dysfunction. Furthermore, there is no information whatever on the presence or absence of family and social support systems and networks.

3. Factors Relating to the Index Children. There is no information on the stressful situations and traumatic events that may have occurred in the lives of individuals or families. Nor are we told which children received psychological or psychiatric treatment, what the nature of the treatment was, or what its effect, if any, was on their psychopathology. If the overall design and methodology of this study are to be judged by the material presented, doubt remains about whether the two groups of index children were indeed comparable in the nature and severity of their illness and their parents' pathology, and also about family integrity, family functioning, and other environmental influences that may have played a role during the long period of the research.

The selection criteria for the control children also leave room for doubt about whether the differences in extent of psychological disturbance may properly be ascribed solely to the presence or absence of "schizophrenic diathesis." The researchers who set up the group comparison design seem not to have reckoned sufficiently with the basic tenet that the selection of control cases should minimize all biases and differences that may favor one side or another. Against the control children, almost all of whom came from intact families, there was placed a group of index children, a high proportion of whom came from broken families and had been through repeated traumatic experiences because of the recurrent hospitalization of one of their parents. Nagler (1985) notes that in 36 percent of the index cases, one of the parents was away from home, because of hospitalization or for some other reason, at the time of initial testing, and that at that time, 20 percent of the index group parents were separated or divorced. It is reasonable to suppose that in the 15-year followup period, the differences in the frequency of family breakups in the two groups of parents grew. Wallerstein and Kelly (1980) found serious psychological difficulties in more than a third of the cases in a sample of children and adolescents of ordinary divorced families they studied over a 5-year period. An important result of that study, which the researchers of the Israeli High-Risk Study might well have taken into account before reaching their conclusions, was that 37 percent of the children and adolescents of divorced couples exhibited moderate to severe symptoms of depression (Wallerstein and Kelly, p. 211).

The bias in favor of the control group becomes even more obvious as it is learned that the researchers found it necessary—for reasons not stated—to replace almost a quarter (12 children) of the original sample of control subjects after they discovered some type of psychopathology in the control parents (Nagler 1985; Shotten 1985). Under these circumstances, the comparison between the experimental and control groups seems almost devoid of any meaning or significance.

Characteristics of the Psychopathology of the High-Risk Children

One of the study's most interesting findings relates to the essence of the neuropsychological symptoms displayed by the index children in the first two phases of the investigation. These children showed an overall pattern of impaired psychosocial functioning, poor school adjustment, attention difficulties, deficits in perceptual-motor skills, learning disabilities with a lower proficiency level in arithmetic, and "a basic distortion in cognitive integration" (Sohlberg and Yaniv 1985, p. 48).

Forty-four percent of the children of index parents showed signs of a neurointegrative defect in at least one of the testing periods, compared with only 6 percent of the control group. The most common neurological signs were "soft" perceptual-sensory signs, poor motor coordination, poor right-left orientation, poor balance, and motor overflow (Marcus et al. 1985). These very special findings point clearly to the presence among the index children of a typical clinical syndrome of attention deficit disorder (or minimal brain dysfunction [MBD], as some clinicians persist in calling it). If that syndrome was present, then it is reasonable to hypothesize that there was a group among the index parents in whom the presumed schizophrenic disorder would appear related to MBD on a family basis. In the Kibbutz Clinic we found that of 200 children diagnosed as MBD, 16 percent of them appeared to have a genetically determined syndrome (Kaffman, Sivan-Sher, and Carel 1981). Those with genetically determined MBD are a high-risk group for psychiatric disease, and several authors have suggested a link between particular cases of familial MBD and schizophrenia (Bellak and Charles 1979).

It would seem that the researchers did not take this hypothesis into account, since in their third and final followup examination, they did not test for possible remnants of neuropsychological dysfunction. Further neurological assessment of the sample seems required, mainly to determine the role of MBD-linked, familial-genetic influences in the development of pathology in some of the index children.

Comparison of Outcomes: Kibbutz and Town Children 15 Years Later

At the end of the two main stages of the investigation (1967 and 1973), Silberman and Tassone (1985) concluded that: "This study failed to find any consistent differences between rearing environments on the subjective or objective measures that were used either for the whole group or in the index children. It is reasonable to hypothesize, therefore, that the deficits found in our index children reflect the schizophrenic diathesis and are not highly subject to modification by the environment" (p. 144). Eight years later (1981), however, the research teams decided on a third examination, this time restricted to a single interview by a clinical psychologist (Arje Latz) brought in especially from the United States. A test battery consisting of the Schedule of Affective Disorders and Schizophrenia—Lifetime Version, the Social Adjustment Scale, and six subtests of the Wechsler Adult Intelligence Scale was administered to 23 kibbutz index subjects and 23 town index subjects whose average age by this time was 25. On the basis of the data from the SADS-L interviews, Mirsky et al. (1985) reported a higher incidence of psychiatric disorders among the kibbutz index cases: ". . . nine schizophrenia spectrum disorder cases occurred in the index group, and six of these were in the kibbutz-index cell. Even more surprising is the finding of 11 instances of affective disorder (five major), all but two of whom were in the kibbutz index cell" (p. 151). Hence

> our followup data clearly suggest adolescence as the period in which the differential effects of environment began to appear, even though such effects were not apparent in the measures previously gathered; [and] our results indicate an increased incidence of psychiatric disorder in the kibbutz index-subjects, suggesting that the interaction of the kibbutz environment with a schizophrenic diathesis may have a psychopathogenic effect. (Mirsky et al. 1985, p. 152)

In view of the numerous methodological weaknesses in this work, it would seem that the researchers' hypothesis—that the particular quality of kibbutz life may interact negatively with a schizophrenia diathesis and may potentiate psychopathology in early adulthood—is too far-fetched and lacks a sufficiently strong base. Even if we accept the validity of the *DSM-III* psychiatric diagnoses determined by a single clinical psychologist after only one interview; even if we disregard the fact that the examination was not blind, since "the environment from which they [the subjects] came (kibbutz or town) was too easily recognizable to be kept a secret" (Nagler 1985, p. 37); even then it seems that hypothesizing a causal connection between the mode of life in the kibbutz and the psychopathology observed in the 25 kibbutz children of presumed schizophrenic parents is quite speculative. As already noted, before automatically relating the differences in psychopathology between kibbutz children and nonkibbutz children to the differences in way of life or childrearing practices, the researchers ought first to note and evaluate other important factors (e.g., severity of parental pathology, degree of recurrence of mental illness, level of deterioration, family functioning, marital breakups, other stressful events). Our 25 years of clinical experience in the Kibbutz Clinic, involving more than 100 families having children with a schizophrenic parent diagnosed according to *DSM-III* criteria, points to the decisive importance of the influences just mentioned—and not necessarily of specific kibbutz factors—on the development of psychological problems among the children. In addition, our experience with families in town does not support the study's findings. On the whole, in our everyday practice with offspring of schizophrenics, we have found a lower incidence of psychopathology in the kibbutz than in town. This may be due to the planned use of the support of social networks in the kibbutz and the active implementation of deinstitutionalization policies (Kaffman 1981).

To those who are not familiar with daily kibbutz life, it should be pointed out that every year the kibbutzim absorb from town hundreds of boys and girls in early adolescence, children who for one reason or another are sent to the kibbutz to continue their education during the formative high school years. Among these are city children born to schizophrenic parents, youngsters who were separated from their parents (except for an occasional visit) and sent to the kibbutz to remove them from a stifling, pathogenic home environment. We have had occasion to follow the development of more than 40 such boys and girls, each of whom lived in a kibbutz for between 10 and 20 years. The vast majority showed no signs of any significant illness; on the average, they exhibited good psychosocial functioning. The history of these children offers no support for the researchers' contention that the interaction of the kibbutz environment with a schizophrenic diathesis may have pathogenic consequences. Were this hypothesis true, we would be hard put to explain why this allegedly harmful environment was not operative before adolescence. According to the researchers, the kibbutz-reared index children exhibited, up to age 16, similar and sometimes less psychopathological symptoms than their counterparts from town. Neither we, nor the researchers themselves, possess the information on many relevant dimensions to form sufficiently well grounded hypotheses about the differences between the two index groups at the end of adolescence. One possible explanation may be that the two index groups were *ab initio* quite different in their parents' psychopathologies. The fact that there were nine subjects (39 percent) in the kibbutz index group with depressive spectrum disturbances when they reached the average age of 25 indicates (with a high degree of probability) that the kibbutz index-parent group included a number of individuals with affective disorders accompanied by psychotic features. Of the nine, five had major affective disorders, compared with only one case of major affective disorder in the corresponding town index group. As is well known, major affective disorders are more common among family members than in the population at large, particularly if the first major depressive episode occurs at about age 20 (Weissman et al. 1984).

Under the conditions of this study it would be difficult to negate the possibility that in some cases of psychiatric disorder in the index group in late adolescence, the pathology was related to endogenous factors. It seems almost certain that in other cases, a complex of stressors related to intrafamily conflict and/or other unknown environmental pressures played some role in creating the psychopathology.

With all that, if the researchers still maintain that, in adolescence, the combination of schizophrenic diathesis and the conditions of kibbutz life is liable to increase the risk of serious psychopathology, they should provide more substantial proof of how the supposed pathogenic factors operated in the cases they studied. Being well versed in the details of life in the kibbutz for both children and adults, I do not deny that this form of life—not unlike any other social setup—contains sources of tension, conflict, frustration, and stress. Based on my experience and that of the staff of the Kibbutz Clinic, however, it must be emphasized that no verification has been found for any of the following contentions:

1. That kibbutz children of schizophrenic parents display a higher incidence of psychiatric disorders on reaching maturity than do their counterparts in the city.

2. That within the wide variety of nosological categories that may show up among children of schizophrenics, the diagnosis of major affective disorder occupies a central place. On the contrary, the incidence of major endogenous depression or bipolar disorder in the offspring of confirmed schizophrenics is minimal and highly exceptional.

3. That it is possible to identify specific factors in kibbutz life that for some reason begin to have a detrimental effect on the young when they have reached the age of 20.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: American Psychiatric Association, 1980.

Bellak, L., and Charles, E. Schizophrenic syndrome related to minimal brain dysfunction: A possible neurologic subgroup. *Schizophrenia Bulletin*, 5:480–481, 1979.

Elizur, E., and Kaffman, M. Factors influencing childhood bereavement. *American Journal of Orthopsychiatry*, 53:668–676, 1983.

Kaffman, M. Family conflict in the psychopathology of the kibbutz child. *Family Process*, 11:171–188, 1972.

Kaffman, M. Community care for the kibbutz chronic psychiatric patient. *International Journal of Family Therapy*, 3:193–204, 1981.

Kaffman, M.; Sivan-Sher, A.; and Carel, C. Obstetric history of kibbutz children with minimal brain dysfunction. *Israel Journal of Psychiatry*, 18:69–84, 1981.

Marcus, J.; Sohlberg, S.C.; Lifshitz, M.; and Silberman, E.K.; Neurological findings in high-risk children. *Schizophrenia Bulletin*, 11:85–100, 1985.

Mirsky, A.F.; Silberman, E.K.; Latz, A.; and Nagler, S. Adult outcomes of high-risk children: Differential effects of town and kibbutz rearing. *Schizophrenia Bulletin*, 11:150–154, 1985.

Nagler, S. Overall design and methodology of the Israeli High-Risk Study. *Schizophrenia Bulletin*, 11:31–37, 1985.

Nagler, S., and Mirsky, A.F. Introduction: The Israeli High-Risk Study. *Schizophrenia Bulletin*, 11:19–29, 1985.

Shotten, J H. The family interview. *Schizophrenia Bulletin*, 11:112–116, 1985.

Silberman, E.K., and Mirsky, A.F. The NIMH-Israeli schizophrenia project: The history of a high-risk study. *Schizophrenia Bulletin*, 11:146–149, 1985.

Silberman, E.K., and Tassone, E.P. The Israeli High-Risk Study: Statistical overview and discussion. *Schizophrenia Bulletin*, 11:138–145, 1985.

Sohlberg, S.C., and Yaniv, S. Social adjustment and cognitive performance of high-risk children. *Schizophrenia Bulletin*, 11:61–65, 1985.

Spitzer, R., and Endicott, J. *Schedule for Affective Disorders and Schizophrenia—Lifetime Version*. 3rd ed. New York: New York State Psychiatric Institute, 1977.

Wallerstein, J., and Kelly, J. *Surviving the Breakup: How Children and Parents Cope With Divorce*. New York: Basic Books, 1980.

Weissman, M.; Wickramaratne, P.; Merikangas, K.; Leckman, J.; Prusoff, B.; Caruso, K.; Kidd, K.; and Gammon, D. Onset of major depression in early adulthood. *Archives of General Psychiatry*, 41:1136–1143, 1984.

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An Invitation to Readers

Providing a forum for a lively exchange of ideas ranks high among the *Schizophrenia Bulletin's* objectives. In the section **At Issue**, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. **At Issue** is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the *Bulletin* or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—*The Editors.*

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The Israeli High-Risk Study: Reply to Kaffman

by Allan F. Mirsky

Abstract

Kaffman's critique (1986) of the Israeli High-Risk Study centers on a number of issues: research design, information reported, appropriateness of sample, and Kaffman's own direct experience with psychiatric populations in kibbutzim and elsewhere in Israel. I contend that Kaffman has not fully understood the goals, methods, design, or results of the investigation. The information reported was as complete as possible if it is borne in mind that many of the issues and problems he has raised were not considered in research done on psychiatric disorders 25 years ago. Nevertheless, I contend that the Israeli study meets currently accepted standards of psychiatric research, that the results are valid, and that they raise questions about possible pathogenic influences on vulnerable persons raised on kibbutzim.

Dr. Kaffman (1986) has offered a series of criticisms of the Israeli High-Risk Study, most of which stem from his 25 years of experience as Medical Director and Chief Psychiatrist of the Kibbutz Child and Family Clinic, "which provides psychological and psychiatric services for the majority of kibbutzim in Israel" (p. 152). Criticisms of our study from such a source would appear to carry special importance and to merit a reply. I appreciate that our findings may be difficult for him to accept in view of his experience and professional position. Nevertheless, I believe the results are valid, however unexpected they may be.

Dr. Kaffman's criticisms can be classified into four categories: (1) the design of the study was flawed; (2) insuffiсent information was either gathered or reported; (3) the sample was inadequate, biased, or both; (4) our findings must be in error because they are contrary to his own experience. I shall attempt to deal with the majority of these criticisms.

I wish to state at the outset that I am grateful for Dr. Kaffman's comments. Although I argue against the saliency of most of them, I appreciate the time he spent preparing his analysis of our study. I must add that, in the interests of expediency, I did not communicate with the other major participants in this work. These comments, for better or for worse, are mine.

It is my view that Dr. Kaffman is essentially misinformed with respect to the first and third categories of criticism, and this may be due in part to a misunderstanding of the principles of research design employed in clinical research and, specifically, of the design of this study. With respect to the second class of criticisms, although we have attempted to report the data as fully as possible, there are some gaps. We are attempting to provide additional information on the subjects as time permits (e.g., Marcus et al., in press; Mirsky et al., in press). Concerning the fourth criticism, I must say that assertions are only assertions unless they can be supported by facts, preferably in the form of published data.

Let us deal with some of the specific questions Dr. Kaffman has raised.

1. Study Design. The quadron analysis, whereby subjects were matched in squads of four (an at-risk and control subject within each rearing condition), was implemented

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after considerable study of the problem by Rosenthal and colleagues (1971). This design provided a powerful tool with which to evaluate (in experimental design terms) main effects as well as interaction effects in the study. The children were, in fact, matched on as many variables as were considered relevant and possible to achieve at the time; the original criteria were violated (e.g., with respect to age range) only when it proved infeasible to find suitable subjects in the age group intended. Dr. Kaffman argues that the study is biased because about one-fourth of the original sample of control subjects was replaced after some type of psychopathology was discovered in their parents. He obviously misunderstands the nature of the study and of control subjects. Replacement of control parents with psychopathology by those without psychopathology would yield more sharply defined differences between the at-risk and control groups and an increased likelihood of an unambiguous outcome.

The objection was raised that in the index cases, there would more likely be recurrent family difficulties and traumatic experiences because of frequent hospitalizations of one of the parents. It is obvious that this would occur in families of schizophrenics, and it is for this reason that the kibbutz setting, with its potential to moderate these traumatic effects, was selected.

It is alleged that there was an exaggerated tendency to diagnose schizophrenia in the early 1960s. It is certainly unfair to criticize a study designed in 1964 on the basis of standards used in 1986. Nevertheless, European-trained psychiatrists (this encompasses virtually all our psychiatrists) have tended to use the diagnostic label of schizophrenia more sparingly than American-trained psychiatrists, so I am not impressed by this argument. The researchers who reviewed the diagnoses, including Drs. David Rosenthal, Joseph Marcus, and Shmuel Nagler, were extremely sophisticated; collectively, they represented the state of the art in psychiatric diagnosis in the 1960s, if not the 1980s. While it is true that severity of illness data were not provided in the *Schizophrenia Bulletin*, some data that bear on that question do exist and were presented in conjunction with the outcome data analysis in the paper by Mirsky et al. (1985). This variable (i.e., severity) is not often reported in this research, since it can be defined in many ways and is therefore extremely difficult to quantify.

Dr. Kaffman has pointed to the outcome of 10 children with affective disorders as proof that the original diagnoses of the parents were incorrect. This is a weak argument. As already noted, strenuous efforts were made to exclude persons who did not fit the diagnosis of schizophrenia as generally agreed upon in the early 1960s; symptoms that were included have been listed and described by Nagler (1985). As pointed out by Mirsky et al. (1985), we were not able to show that parents with affective-type symptoms (which are, after all, common in schizophrenia) had children with affective diagnoses. The symptoms we found that differentiated the parents of subjects with affective disorders from those of subjects with schizophrenia spectrum disorders included inappropriate affect, catatonia, and total number of symptoms. In each case, the schizophrenia spectrum parents had more of the symptoms in question.

The criticism, further, that the diagnostic interviews were not conducted by the researchers themselves misses the point about the double-blind condition in behavioral research. Since the researchers were excluded from this contact, they could be relatively unbiased in their evaluations and testing when they saw the children. It should be recalled that in the landmark studies of adopted-away Danish offspring of schizophrenic parents (e.g., Rosenthal et al. 1971), only Joseph Welner actually interviewed the patients; the other investigators (Kety, Rosenthal, Wender, Schulsinger, and others), as in the present instance, reviewed the records and scored the interview data.

2. **Insufficient Information.** I do not agree that we have provided insufficient information about the subjects and related issues in this investigation. In our *Schizophrenia Bulletin* report, we provided more than 130 pages of detailed study findings. This does not include prior publications by Marcus et al. (1981) on neurological (soft sign) factors in these subjects. Additional details concerning the early history of these subjects are forthcoming in subsequent issues of the *Schizophrenia Bulletin*, as well as in other publications. In passing, I can add that we did look into the location of the kibbutzim vis-à-vis terrorist attacks. We found no relation between proximity of the kibbutz to hostile borders and/or a history of terrorism, and the diagnoses of the children living therein. We did provide considerable detail about the nature of family factors in the risk families with respect to the parents' attitudes toward their offspring; this is summarized in some detail in the chapter by Shotten (1985). It would, of course, have been most illuminating to have had family interaction-derived measures of

Affective Style (Goldstein 1985), Expressed Emotion (Browne et al. 1962, 1972), or Communication Deviance (Wynne et al. 1976); but those measures either did not exist or were not in wide use in 1964.

It is true that we did not provide sufficient information about the pathology of the spouses of the index parents, since we know that assortative mating does occur in patients with schizophrenic disorders. The possible influence of psychopathology in the spouses of index parents was not fully appreciated in genetic studies in the 1960s, nor is it always considered or measured even now. We intend to publish what information we have in the near future. Some preliminary analyses indicate that there is no evidence of a relation between the pathology of the spouses of the index parents and disorders in the children.

3. Inadequate or Biased Sample. Dr. Kaffman has proposed that, because of the early findings of cognitive and "soft" neurological sign differences between the index and control cases, we were really following a group of children with the diagnosis of ADD (attention deficit disorder) who happened also to be at risk for schizophrenia. Dr. Kaffman does not acknowledge the extensive data on neurological factors in children at genetic risk for schizophrenia (e.g., Fish 1977; Marcus et al. 1981, 1985). Nor does he make it clear why more of these ADD risk cases were found in the kibbutz setting. I firmly believe that children with ADD become adults with residual ADD and not schizophrenia or other related spectrum diagnoses. The two disorders are distinct entities with distinct clinical courses and outcomes.

Dr. Kaffman has also raised the question of whether one interview by a psychologist who was "brought in especially from the United States" (p. 155) was sufficient to characterize the subjects. The majority of current research in schizophrenia is also based on one patient contact. In the present case, however, this was a detailed interview involving administration of the Schedule of Affective Disorders and Schizophrenia—Lifetime Version, the Social Adjustment Scale, and various subtests of the Wechsler Adult Intelligence Scale. Although Dr. Latz was brought in from the United States, he consulted extensively with Dr. Nagler, who usually made the initial contact with the subject. Dr. Latz is an experienced, senior clinical psychologist who is also an Israeli. He was blind to the identity of the subjects during conduct of the study. Furthermore, the actual diagnoses were made after thorough discussion and consultation on each case with Dr. Silberman, an experienced psychiatrist.

4. Findings Counter to Dr. Kaffman's Experience. It seems to me that the underlying difficulty Dr. Kaffman has with the study is that the results came out the wrong way; that is, the high-risk children raised on kibbutzim appear now to be faring worse than those who grew up in their nuclear families. He asserts that there is less psychopathology in kibbutzim than in towns in Israel. Further, in some subjects he is following (although whether or not this is a study is not clear), he has obtained a result contrary to our own. That is, the vast majority of a group of 40 offspring of schizophrenic parents who were brought to kibbutzim to be raised showed, on average, "good psychosocial functioning" and no signs of any "significant illness." This would be a unique outcome in the literature on the offspring of schizophrenics. One must ask the questions of his study that he has asked of ours: How, and by whom, were the parents diagnosed? What instruments or interview techniques were used to evaluate the parents and the children? Who performed the research, and were the examiners aware of the genetic history of the children? Were there control subjects? Until and unless such data are provided and can be evaluated, Dr. Kaffman's assertion must be viewed as unsupported.

A surprising, if not incomprehensible, finding for Dr. Kaffman is that somewhere between ages 17 and 25 (not 20, as he asserts), some factors may come into play that have a detrimental effect on kibbutz children. Although we do not yet have the data, we suspect that the military experience could provide such an influence. Many discussions with knowledgeable Israelis have emphasized the fact that kibbutz children are under special pressure to achieve when serving in the Israeli Defense Forces. Certainly, kibbutzniks contribute vastly more in numbers to the officer corps than would be expected on the basis of the modest 3 to 4 percent of the Israeli population they constitute. The stress of having to cope and even to excel under the dangerous conditions of military life could interact with genetic vulnerability and lead to the excess of psychopathology we found. This may not be a completely satisfactory explanation for the excess of affective disorders in the kibbutz-index group, since the majority of such cases (seven of nine in the kibbutz index group) were female and women are known to be assigned to less personally dangerous roles in the Israeli Defense Forces. Nevertheless, the effects of the stress of military life on vulnerable women must be considered. The additional stress of having to decide whether or not to remain on the kibbutz after military service may also provide special difficulties for vulnerable children, both male and female.

We are attempting to learn more about this period of the lives of our subjects; we *do* know that more persons manifest schizophrenia for the first time between the ages of 17 and 25 than during any other time of life (Gottesman and Shields 1982). To the extent that this period represents some stage of biopsychological vulnerability, it is conceivable that there are stresses in kibbutz life that are especially salient at this time. In his critique of our study, Breznitz (1985) has provided some useful ideas about what some of those pressures might be—including the special role of the peer group in kibbutzim.

In conclusion, I am concerned when a senior clinician reports that the outcome of a research study and his experience based on day-to-day clinical practice are discrepant. I do not believe, however, that Dr. Kaffman has established that a discrepancy actually exists or that his comments have shed much light on the supposed discrepancy. His arguments, in my view, have not ruled out the possibility that there are features of kibbutz life that may have pathogenic effects in vulnerable children (e.g., Mirsky and Duncan-Johnson 1984) and that may provide useful clues to the etiology of schizophrenia.

References

Breznitz, S. Chores as a buffer against risky interaction. *Schizophrenia Bulletin*, 11(3):357–360, 1985.

Browne, G.W.; Birley, J.L.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, 121:241–258, 1972.

Browne, G.W.; Monck, E.M.; Carstairs, G.M.; and Wing, J.K. The influence of family life on the course of schizophrenic illness. *British Journal of Preventive and Social Medicine*, 16:55–68, 1962.

Fish, B. Neurobiologic antecedents of schizophrenia in children: Evidence for an inherited, congenital neurointegrative defect. *Archives of General Psychiatry*, 34:1297–1313, 1977.

Goldstein, M.J. "The UCLA Family Project." Presented at NIMH High-Risk Consortium, San Francisco, April 1985.

Gottesman, I.I., and Shields, J. *Schizophrenia: The Epigenetic Puzzle.* Cambridge, England: Cambridge University Press, 1982.

Kaffman, M. The Israeli High-Risk Study: Some critical remarks. *Schizophrenia Bulletin*, 12:151–157, 1986.

Marcus, J.; Auerbach, J.; Wilkinson, L.; and Burack, C.M. Infants at risk for schizophrenia. *Archives of General Psychiatry*, 38:703–713, 1981.

Marcus, J.; Hans, S.L.; Mednick, S.A.; Schulsinger, F.; and Michelsen, N. Neurological dysfunctioning in offspring of schizophrenics in Israel and Denmark. *Archives of General Psychiatry*, 42:753–761, 1985.

Marcus, J.; Hans, S.L.; Nagler, S.; Auerbach, J.G.; Mirsky, A.F.; and Aubrey, A. A review of the NIMH Israeli kibbutz-city study and the Jerusalem infant development study. *Schizophrenia Bulletin*, in press.

Mirsky, A.F.; Duncan, C.C.; Silberman, E.K.; Nagler, S.; Kugelmass, S.; Sohlberg, S.; and Shotten, J.H. Comparison of the early history of persons with schizophrenia spectrum and affective diagnoses in the Israeli High-Risk Study. *Schizophrenia Bulletin*, in press.

Mirsky, A.F., and Duncan-Johnson, C.C. Nature versus nurture in schizophrenia—The struggle continues. *Integrative Psychiatry*, 2:137–141, 1984.

Mirsky, A.F.; Silberman, E.K.; Latz, A.; and Nagler, S. Adult outcomes of high-risk children: Differential effects of town and kibbutz rearing. *Schizophrenia Bulletin*, 11(1): 150–154, 1985.

Nagler, S. Overall design and methodology of the Israeli High-Risk Study. *Schizophrenia Bulletin*, 11(1):31–37, 1985.

Rosenthal, D.; Wender, P.H.; Kety, S.S.; Welner, J.; and Schulsinger, F. The adopted away offspring of schizophrenics. *American Journal of Psychiatry*, 128(3):307–311, 1971.

Shotten, J.H. The Family Interview. *Schizophrenia Bulletin*, 11(1): 112–116, 1985.

Wynne, L.C.; Singer, M.; Bartko, J.; and Toohey, M. Schizophrenics and their families: Recent research on parental communication. In: Tanner, J.M., ed. *Psychiatric Research: The Widening Perspective.* New York: International Universities Press, 1976. pp. 254–286.

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The Need for Asylum for the Chronically Mentally Ill

by Mona Wasow

Abstract

Our society highly values autonomy, independence, and freedom. In our model programs for the chronically mentally ill (CMI), we aim for these values and, in the process, may be doing a disservice to those people who cannot attain them. This article proposes that even the best in community care is not enough protection for many CMI. We need to do a better job of discriminating between who can and who cannot benefit from model community programs. We also need to develop a whole continuum of care that accepts the dependency of CMI people, and their various levels of functioning, as well as the progress, deterioration, and inevitable fluctuations that occur. Emphasis is placed on the need for sanctuary and asylum for many that only permanent housing of some kind can provide.

> Give sorrow words: the grief that does not speak, whispers to the over-wrought heart and bids it break.
>
> *Macbeth*, Act IV, Scene 3

My 28-year-old son is a success statistic in a model community treatment program for the chronically mentally ill (CMI). He is a success because he has been rehospitalized only once in the 12 years of his mental illness. He is very quiet and unobtrusive and does not bother anyone.

He had been living on his own in a one-room apartment, but I brought him into my home a few days ago to get him ready for a National Institute of Mental Health (NIMH) research project, where I know he will get adequate food and shelter and humane care. He is filthy and very skinny; he has boils on his body and is essentially mute. There was no toilet paper in his room, no food, no sheets on the mattress; the chaos in the room was congruent with the chaos in his poor sick mind.

He has lived in 15 different places in 12 years: three times with relatives, four times in a room by himself, twice at the YMCA, in two different group homes, once in jail for 6 weeks while waiting for a court hearing to determine placement (that was his only crime: truancy—no place to live), once in a room with a roommate, once in an unlocked institution for 130 retarded and mentally ill people, and now off to the NIMH project. There were also two hospitalizations, one for 4 months and one for 1 month, but I do not count those. His case is quite typical for a CMI person. Moves are stressful even for people in good health. Imagine what all that moving does to a frightened, fragile, mentally ill person.

None of our model community treatment programs would consider my son or others like him a "success." They understand that rehospitalization should not be the only outcome criterion—that quality of life, patient satisfaction, and social functioning all need to be considered. But the *reality* of the situation is that despite massive deterioration, the mental health system as a whole does not provide him asylum.

So unless he commits an outwardly violent act, my sons gets listed as a "success" in the deinstitutionalization movement. That is not how he looks to me. I think he looks like a giant, broken plastic throwaway toy. And he breaks my heart.

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We do not need yet another article describing the horrors of deinstitutionalization when there is no good community care. The literature is replete with such, as are our daily newspapers. This article presents the idea that perhaps even the best in community care is not enough protection for many CMI. It is *not* my intention to question what is good but rather (1) to suggest better discrimination in selecting who can and cannot benefit from these model programs and (2) to make a plea for permanency planning for those in need of care and protection. Emphasis is placed on the need for sanctuary and asylum[1] by many, protection that only an institution of some kind can provide.

Ideologies vs. Reality

All cultures have a value-laden approach to almost everything. In the United States we have strong beliefs about the virtues of independence, autonomy, and self-reliance. Civil rights and personal freedoms are also very important in our country. A review of cross-cultural perspectives of mental illness (Lefley 1985*a*) shows a sharp contrast between Western ideologies and those found in developing countries. It also suggests that our Western values and ideologies may be harmful to CMI people. Cultural differences were found in the World Health Organization study (1979) showing that the course of chronic mental illness was significantly better in developing countries than in the United States. Countries that stressed mutual interdependence, rather than independence and autonomy, appear to provide a better environment for the CMI.

Most CMI people cannot function well independently; they are not self-reliant and cannot take care of themselves or others. Our value that says we must do all that we can to promote these virtues may be doing more harm than good.

Lefley (1985*b*) wrote:

> There is a single overarching American core value, according to anthropologist Francis Hsu (1972); and that is *fear of dependency*. The emphasis on self-reliance and personal autonomy that so characterize our value system have been generalized to our most basic diagnostic and treatment philosophies. Indeed, throughout deinstitutionalization and rehabilitation literature, the phrase "return the patient to independent living" appears over and over again, regardless of whether the goal is realistic or even advisable for all classes of patients. [p. 61]

The proponents of our model programs for the CMI are proud of the vigorous outreach being done to maintain people in the community and prevent hospitalization. For those people who function relatively well under such circumstances, these are indeed model programs. But where these programs exist—and they surely are better than nothing at all, which is the sad situation in too many areas of the world—they are being applied indiscriminately to all or most CMI.

The ideologies of independence and functioning in the least restrictive environment are good ones when they work. The reality, however, is that for many they do not work well. It is not possible at this time to know what "many" means. Commonsense observation of what one sees in the streets, bus stations, parks, and shelters for the homeless in the United States leads one to believe it means far too many people. Pepper (1985) writes: "For many of the CMI—and we do not yet have the experience to predict either percentages or numbers—it appears likely that a degree of supervision or special support in their living arrangements will always be needed to maintain and enhance community adjustment" (p. 3).

If an outreach worker is bringing medication to a mentally ill person's room, and that person is just lying in bed staring into space, is that so wonderful? Is being filthy, alone, totally without structure and without human contact better than being in humane, unlocked institutional housing of some kind?

In Lamb's book *Treating the Long Term Mentally Ill* (Lamb 1982), Bachrach (1982) writes:

> It was inevitable that chaos would result, and it did. Ego-investment in specific approaches to patient care ran high and often resulted in deeply rooted territorial feelings that exacerbated service delivery difficulties. In short, despite noble intentions, the efforts of service planners often resulted in patient neglect as more and more individuals endured severe barriers to treatment. And the CMI, who were to have been the beneficiaries of a "bold new approach" to service delivery, continued to suffer from inadequate attention to their most basic needs [p. xi].
>
> [We charge them] with responsibilities their illnesses make them incapable of handling. [Lamb points out that] Commitment to an ideology . . . must never reign. What is best for the individual patient is what counts [p. xiii].

Even Lamb, a proponent of community care, feels that long-term

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[1] "An inviolable place of refuge and protection giving shelter . . . a place of retreat and security . . . an institution for the relief or care of the destitute or afflicted and especially the insane" (*Webster's*).

CMI people who function well in community programs are in the minority of the total CMI population. Another authority on the CMI, John Wing (1978), writes: "By letting mentally ill people retain their ordinary freedom, society gives them the freedom also to remain ill . . . and indeed the freedom to deteriorate mentally" (p. 239).

Another finding of the cross-cultural studies is that cultures that do not believe in mastery over fate and nature seem to do better by their CMI people. When CMI people and their dependency needs are better accepted, they are not forced to try to achieve where they cannot. We have created artificial work situations in many of our model programs that offer make-believe "job training" (Estroff 1981) to CMI who recognize the futility of it all and their inability to function independently in our culture. True acceptance and "normalization" would be accepting their real limitations.

As kind and caring as our monumental attempts may be for sheltered workshops, mainstreaming, and encouraging the CMI to function as normally (meaning independently, self-supporting, autonomously) as possible, the result is that they know they have failed. Our efforts might be better spent in broadening the continuum of care to include more structure, supervision, and human contact in their living situations.

Housing

Human beings' feelings about being properly oriented in space, and having a place they can call their own, run deep. "Such knowledge is ultimately linked to survival and sanity. To be disoriented to space is to be psychotic" (Estroff 1981, p. 53). Eviction from housing is a very common experience for CMI people. "Some clients felt they had no place to go and that they do not belong anywhere" (Estroff 1981, p. 60); and they were right.

As presently set up in our model programs, as soon as mentally ill persons show any strengths at all, they are encouraged to live more independently (e.g., to leave a group home and live in their own room or apartment in town). This is frequently followed by a vicious circle of deterioration. A CMI person must become *very* ill before he or she can get help, perhaps hospitalization, then a protected living environment, some improvement, and then on to more independent living again—until the next time. Many of us have watched our children and our clients spiral through this vicious vortex circle over and over again. How devastating it must be for the CMI as they endure unending stigma, shame, failure, upheaval, filth, poor nutrition, neglect, and rejection.

In the words of Segal and Aviram (1978): "Once we move into the realm of community care, it is not clear whether such rights guarantees are really in the best interests of the resident or whether they create a situation open to more abuse than the benefits produced by the guarantee" (p. 269).

The position that the CMI will become too dependent if too many of their needs are met may be turned around and an argument made for the opposite view: that they will become too dependent if most of their needs are not met (indeed, this is how human beings usually function). Other researchers talk about "patients' need for periods of minimal change or demand in order for recompensation and consolidation of gains to take place" (Stein and Test 1985). Stability of housing, food, and care is a basic requirement for this minimal demand; and that is hard to obtain in the community, even with all the good outreach provided by our model programs.

Writing about their model program in Chicago, Witheridge and Dincin (1985) point out that a large number of hospitalizations occur for nonpsychiatric reasons: "Problems of this sort include having no income, having no place to live, and having no food on the table" (p. 72).

Pepper (1985), feels that the key issue to the problems and treatment of the CMI is the lack of appropriate places for them to live: "To paraphrase Susan Sheehan [1982], 'Is there no place on earth for them?' The hazards and stresses of community life place these vulnerable young persons seriously at risk, and the cheerless room in a welfare hotel . . . is hardly a solution" (p. 1). He goes on to say: "By now a major lesson learned in that experiment (deinstitutionalization and model programs) is that the community has not been able, by any of the means tried so far, to offer an adequate substitute for what might be called the homemaking function—*the kitchen, bedroom, and living room*—of the institution" (p. 5, emphasis added).

Pepper's suggestions for a comprehensive continuum of housing (in decreasing order of supervision) for the CMI are as follows:

1. Crisis housing with total care and support.
2. Supervised residences with around-the-clock staffing. This would usually be temporary housing, lasting anywhere from several weeks to 9 months.
3. "Developmental" housing with actively supportive and rehabilitative programming. This could last from months to years, depending on

whether or not the person was able to move on; it would include heavy staffing and structured use of time.

4. Supportive residences: permanent for some, a further step toward independence for others.
5. Supervised apartments, providing daily staff visits but also as much independence as the individual can handle.
6. Supportive apartments, providing only semiweekly staff visits. These apartments could be on campus or in the community itself.

Pepper's suggestion, with which I agree, is that this housing continuum be in the same geographic area so that it would be efficient to administer. That does conjure up an image of the old hospital grounds but with a very different attitude and atmosphere. No one is talking about locked housing, except for the few who may sometimes need it.

This kind of housing would be planned to address from the outset the total spectrum of clients' various levels of functioning, and to allow clients to move freely to a more or a less structured setting as their functional capacity progresses, fluctuates, or deteriorates.

We very much need some *permanency planning* for the CMI. Health and social services are finally recognizing the need for this with dependent foster children. For the dependent foster child or the dependent CMI person, having a permanent home to call his or her own is very important. Fifteen moves in 12 years cannot be good for anybody.

Hospitalization

When mentally ill people require hospitalization, our present aim is to get them out of the hospital as quickly as possible, for fear that too long a stay may cause dependency. "The subject of dependency is rarely noted, except as a pathological sign, in the research literature of the West" (Lefley 1985*a*, p. 61).

I have heard veterans of the mental health system, CMI people over the age of 65, talk about "the good old days" in our (admittedly above average) quality state mental hospital. They reminisced about a time when "we had our own gardens, and a chorus, and doctors and nurses would really get to know you. . . . Now it's just in the slammer, get on meds, and out you go again. Nobody cares."[2]

Mechanic (1980) writes:

> Many patients find their hospitalization experience a relief. The community situation from which they come is often characterized by extreme difficulty and extraordinary personal distress. . . . Many patients in mental hospitals report that hospital restrictions do not bother them, that they appreciate the physical care they are receiving, and that the hospital—despite restrictions—enhances their freedom rather than restricts it. [p. 129]

We are all aware of the horrors of bad hospitals. That does not mean, however, that good unlocked hospitals cannot exist. It is my belief that we need many small, humane, unlocked institutions of some kind, places in which dependent people can feel at home.

The Issues of "Rights"

Community mental health programs tend not to include families unless family members rather assertively ask to be included.[3] Many such members are worn down by the stresses of seeing their children so ill and the frustrations of trying to deal with unworkable mental health and legal systems. So after not too many years have gone by, both sides tend to leave each other alone.

Lefley (1985*a*) points out that deinstitutionalization addresses only the rights of patients. What of the rights of their families and the rest of society? "Aging parents have a right to live their lives without taking care of dependent adult children. . . . Thus patients' rights to reject treatment are seen as a direct threat to the happiness and integrity of other people" (p. 9).

Lefley (1985*a*) did a survey of 84 health care professionals with adult CMI children. These families "provide an educated judgement of the aversive impact of . . . deinstitutionalization policies on families as well as patients" (p. 5). As members of the social science professions, most of these families were quite good at getting the best in model community services for their CMI member. "There are plenty of data, however, among the National Alliance for the Mentally Ill families to support the notion . . . that commitment is the only possibility for treating a loved one who is literally killing himself with neglect" (p. 10).

Mittleman's (1985) personal account captures the burden on families well. She describes decades of many different living situations and treatments, saying she could never find a living situation where they "would not give him back to me after a short time" (p. 301). She finally found permanent housing in a Community Mental Health Center residence program. "I can't tell you the relief I feel in knowing that I can have some kind of life for myself. . . . I don't have to worry constantly about where or when I will have to move him . . . or which residence . . . he will be asked to leave next" (p. 301).

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[2] Three older CMI's who spoke with social work students, 1983.

[3] I hasten to add that this has *not* been my personal experience. I have been well treated. But this is not the experience of most parents in the United States, according to the National Alliance for the Mentally Ill (Hatfield 1978; Wasow 1981; Lefley 1985*a*, 1985*b*).

The issue of the rights of our larger society is complex. Unfortunately, we live in a very prejudiced world; we cannot ignore the impact of that prejudice on the CMI. No part of me is saying that, since there is prejudice toward the CMI, we should reinstitutionalize them. I am saying that we should not underestimate how virulent and destructive that prejudice is. Anyone who has been involved in trying to set up a group home for the CMI knows how hostile and fearful people in the community can be toward this vulnerable population. When our model programs talk about placing CMI people in the "least restrictive environment," we have to be honest with ourselves and realize that that environment is often hazardous and ugly.

There are many issues and problems within the subject of people's "rights":

1. The CMI person's right to freedom, which currently can mean the right to be neglected.
2. Family members' rights to lives of their own. In its most honest form, this can mean institutionalizing the patient so the family can survive. That may be a terrible concept, but not as terrible as watching an entire family fall apart because of the never-ending stresses caused by one sick member.
3. The rights of society—and the responsibilities, too.

In briefest summary, ideal model programs have proven helpful to reasonably well functioning CMI; more power to them! For many, perhaps the majority, however, it is not enough. With all the best intentions—and I believe there are many dedicated, wonderful professional people[4] working in this field—many CMI are still leading terrible, unprotected lives and are being shunted about from pillar to post.

Suggestions

1. Keep on developing model programs in the community for those who can benefit from them but be more discriminating in deciding who can best use them. Predicting just who can and cannot benefit from model programs is, at this point in our knowledge about CMI and its development, very difficult to do. When it becomes obvious that people are not benefiting, however, they should be able to get asylum. At present the CMI have become pawns in a legal/philosophical push that seems more concerned with procedural safeguards than with what happens to sick people on their own.
2. Develop an extensive continuum of care—from locked hospitals to independent living—that reflects the diverse needs of this population. Emphasis needs to be placed on more asylum and protection for those in need.
3. Plan permanency for CMI that recognizes their dependency needs. These people must not continue to be moved around all the time.
4. Create a more structured environment—accepting the real inability of most CMI people to put structure into their lives.

There is a great tendency to swing between extremes in our culture. I see even our model programs as part of the deinstitutionalization extreme, except as part of a larger continuum of care. I am not calling for massive reinstitutionalization. I am calling for *permanent*, structured, supervised housing for the CMI, so that they may have the protection and humane treatment that everyone deserves. To say that we should maximize their independence and autonomy when it is shown over and over again that many cannot benefit from this practice is doing no one any favor, and does many great harm.

Afterthoughts

Musing back to earlier days, when my son was still healthy, I remember being enamored of Thomas Szasz and R.D. Laing. If my son had not become ill, if I had not spent 10 years learning as much as I could about mental illnesses, I might very well be in the other camp today, campaigning for community care and the civil rights of CMI. Some will read this and say: She cannot possibly be objective—she's a parent. True, but rather than accede to the accusation that being a parent blurs my ability to see clearly, I will tenaciously hold to the opposite view that being a parent has made me especially sensitive to this issue. The mental health system sees only that model programs have kept my son out of the hospital. I see his boils and malnutrition; I see the terror and pain as he faces the hostile stares of those in our community who do not really want him there. He is not a statistic, he is my son.

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[4] The people with whom I have worked closely, who have done the painstaking, careful research on these model programs—the administrators, nurses, doctors, social workers, and the frontline workers who do the daily work with the CMI—are the finest people I know. They are conscientious, caring, intelligent, and very kind. I am fighting systems and unrealistic ideologies, not people.

References

Bachrach, L.L. Foreword. In: Lamb, H.R., ed. *Treating the Long Term Mentally Ill.* San Francisco: Jossey-Bass, 1982.

Estroff, S.E. *Making It Crazy.* Berkeley, CA: University of California Press, 1981.

Hatfield, A. "Providing Social Supports for the Families of the Mentally Ill." Vol. III. Prepared for the President's Commission on Mental Health, 1978.

Lamb, H.R. *Treating the Long Term Mentally Ill.* San Francisco: Jossey-Bass, Publishers, 1982.

Lefley, H.P. Families of the mentally ill in cross-cultural perspective. *Psychosocial Rehabilitation Journal,* 8(4):57–73, 1985*a*.

Lefley, H.P. "The Role of Family Groups in Community Support Systems." Issue paper for the 7th NIMH-CSP Learning Community Conference, Rockville, MD, April 28-May 1, 1985*b*.

Mechanic, D. *Mental Health and Social Policy.* 2nd ed. Englewood Cliffs, NJ: Prentice-Hall, 1980.

Mittleman, G. The pain of parenthood of the mentally ill. *Schizophrenia Bulletin,* 11:301–303, 1985.

Pepper, B. Where (and how) should young adult chronic patients live? The concept of a residential spectrum. *Tie Lines,* 2(2):1–4, 6, 1985.

Segal, S.P., and Aviram, U. *The Mentally Ill in Community Based Sheltered Care.* New York: John Wiley and Sons, 1978.

Sheehan, S. *Is There No Place on Earth for Me?* Boston: Houghton Mifflin Company, 1982.

Stein, L.I., and Test, M.A. The evolution of the training in community living model: A decade of experience. *New Directions for Mental Health Services,* 26:7–16, 1985.

Wasow, M. *Coping With Schizophrenia.* Palo Alto, CA: Science and Behavior Books, Inc., 1982.

Wing, J.K. *Reasoning About Madness.* Oxford, London, New York: Oxford University Press, 1978.

Witheridge, T.F., and Dincin, J. The Bridge: An assertive outreach program in an urban setting. *New Directions for Mental Health Services,* 26:65–76, 1985.

World Health Organization, *Schizophrenia. An International Follow-up Study.* New York: John Wiley & Sons, Inc., 1979.

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Minimization and Overreaction to Tardive Dyskinesia

by Mark R. Munetz and S. Charles Schulz

Abstract

Psychiatry's response to the risk of tardive dyskinesia (TD) from long-term neuroleptic drug use continues to swing from one extreme (overreaction) to the other (minimization or denial). The potential negative effects on psychiatric practice of these extreme responses are reviewed. We suggest that while concerns about litigation may partially account for the continued overreaction and minimization, a more fundamental explanation is psychiatry's continuing inability to integrate itself fully as a medical discipline. We suggest that four basic concepts about schizophrenia and its long-term treatment need to be accepted before TD can be responded to objectively: (1) schizophrenia is a serious brain disease; (2) neuroleptics are helpful when properly used; (3) there are limits to the value of neuroleptic drugs in the treatment of schizophrenia; and (4) neuroleptic drugs can cause serious side effects.

> There is a need for a rational acceptance of two facts: Neuroleptics are very helpful when used properly, and tardive dyskinesia is a genuine problem with long-term neuroleptic treatment.
>
> Jeste and Wyatt (1982, p. 9)

Soon after phenothiazines were recognized as effective antipsychotic agents, tardive dyskinesia (TD) was described as a potentially irreversible neurologic syndrome resulting from their long-term use. These two observations set the stage for a polarization of the field of psychiatry into two camps: one that minimized the risks of persistent, drug-induced dyskinesia compared with the benefits of antipsychotic drugs and one that minimized the value of neuroleptics in treating schizophrenia compared with the risks of TD. This dichotomy in part paralleled the ideologic differences between the biologic and dynamic schools of psychiatric thought.

For example, in 1968 Nathan Kline (1968) wrote that the "rarity of irreversible oral dyskinesias must be weighed against the mass of benefits" (p. 53), while Silvano Arieti (1974) maintained that neuroleptic drug therapy produces "only a symptomatic improvement" (p. 627) so "we have to decide in each instance whether we gain or lose more with adjuvant drug therapy" (p. 628). Gardos and Cole (1976) suggested that for many patients, neuroleptic drugs might offer a "cure" that, accompanied by TD and other adverse effects, might be "worse than the disease" (p. 32).

Jeste and Wyatt (1982) have discussed neuroleptics as following the "law of the new drug" in which a pendulum swings first in the direction of overvaluing the drugs as risk-free panaceas, later to the extreme of overemphasizing the drugs' toxicity and thus limiting their clinical usefulness, and still later reaching a midpoint at which drugs are appraised realistically as beneficial when properly used but with genuine, long-term side effects.

There are some indications that the psychiatric profession is approaching the stage of realistic appraisal. The American Psychiatric Association's (APA) Task Force Report on TD (Baldessarini et al. 1979) provided an objective review of the benefits of antipsychotic drugs and the risks of late neurologic side effects. The Task Force Report provided rational guidelines for the prevention and management of TD.

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The recommendations of the APA Task Force, however, have not been universally adopted in the 5 years since their publication. The APA's own chief counsel, in fact, has recently contradicted the Task Force guidelines by recommending that psychiatrists use written consent forms to obtain informed consent for neuroleptic drug treatment (O'Connor 1984). Gualtieri and Sprague (1984) have suggested that neither the APA report nor any other publication or presentation has modified "actual physician behavior with respect to neuroleptic drugs" (p. 347).

It is the thesis of this article, therefore, that we have not reached a stage of realistic appraisal of neuroleptic drugs. Our professional behavior indicates that the pendulum of psychiatric opinion about TD continues to swing from overreaction to denial or minimization.

We have been confronted with these contrasting responses to TD during our own attempts to conduct and publish research regarding the medical-legal and clinical management of TD (Munetz, Roth, and Cornes 1982; Munetz and Roth 1985; Munetz and Schulz 1986). These defensive reactions to TD have had varied presentations.

In 1979 we proposed to study contrasting methods of obtaining informed consent for neuroleptic drug treatment from patients with TD (Munetz, Roth, and Cornes 1982; Munetz and Roth 1985). Our colleagues responded strongly to the proposed study, suggesting, on one hand, that TD was too small a problem to warrant such a study and warning, on the other, that providing patients with information about TD would be so frightening that large numbers would discontinue their medication and decompensate. The study was completed (Munetz and Roth 1985), and the fear that informing patients about tardive dyskinesia would adversely affect their clinical course proved unfounded. Only 2 of 25 study patients chose to discontinue medication after the informed consent procedures. Relapse rates in the year following information disclosure were no greater for the study population than for a comparison group, and all study patients remained in active treatment.

More recently, we recommended that clinicians routinely use the Abnormal Involuntary Movement Scale examinations to screen outpatients for TD. This recommendation was made after demonstrating that such a policy was feasible and effective (Munetz and Schulz 1986). In submitting our study of TD screening for publication, we were cautioned by initial reviewers about making recommendations with implications for standards of care. This warning came despite our perception that the APA Task Force had established reasonable standards of care upon publication of their report in 1979.

In the remainder of this article we describe the effects on psychiatric practice of this defensiveness about TD and suggest some ways it can be understood and overcome.

Effects on Psychiatric Practice

Minimization and overreaction can both negatively affect psychiatric practice. Minimization or denial of TD can result in the overuse of neuroleptic drugs. A lack of appreciation of their potential adverse effects can result in the use of neuroleptic drugs to treat anxiety, depression, or certain character pathology or as a "chemical restraint" to treat behavioral disorders in children or the mentally retarded. Neuroleptic drugs may also continue to be prescribed for that portion of schizophrenic patients who do not clearly benefit from their use.

The psychiatrist's denial of TD also has an impact on care of the patient for whom neuroleptic drugs are appropriate and effective. In such cases early signs of TD may go undetected or ignored, decreasing the chance for rapid intervention and reversibility of movements. Furthermore, patients who are not informed about TD by their treating psychiatrist are increasingly likely to hear about it from friends or the media. Such secondhand reports are likely to be inaccurate and more frightening than information disclosed by the treating physician. Withholding such important information may well be viewed as a reason for distrusting the psychiatrist.

Overreaction to TD can be equally detrimental to clinical care. Neuroleptics are extremely effective drugs when used properly. Consistently discontinuing neuroleptics in schizophrenic patients at the first signs of involuntary movements may well be performing a great disservice. Psychotic relapse rates for schizophrenic patients who stop taking neuroleptics are two to three times greater than for those who continue taking such drugs (Davis 1975).

The psychiatric profession's conflicting response to the TD problem has had another profound effect on clinical practice. We have made ourselves vulnerable to external regulation. The courts and, in turn, our own lawyers have responded to the minimization of TD by some of our colleagues by imposing standards suggested by other colleagues who have overreacted to TD (Stone 1984). Thus, psychiatry is slowly being forced to follow externally imposed standards of care based on inadequate empirical data that make little clinical sense.

Comment

Most psychiatric clinicians and researchers can readily agree with Jeste and Wyatt (1982) that neuroleptics are very helpful when properly used and that tardive dyskinesia is a real problem with maintenance neuroleptic treatment. Our profession has reacted to TD, however, much as the public reacts to cancer: we either pretend it does not exist or act as though it is an unspeakable horror. This article was written so that this conflicting, inconsistent behavior can be recognized, understood, and eventually changed.

How have we gotten ourselves into this predicament? Superficially, the defensive extremes of denial and overreaction can be viewed as an understandable response to our litigious society. Many psychiatrists who prescribe neuroleptic drugs worry that someday they will be defendants in TD lawsuits. Stone (1984) recently described three ways in which the field of psychiatry has contributed to legal skepticism about antipsychotic drugs:

> First of all convincing evidence has been presented of the widespread prescription of neuroleptic drugs often without medical supervision, as a method of chemical restraint in many public facilities. . . . Secondly, there is evidence that some physicians practicing in public mental hospitals lack minimally adequate training in psychopharmacology. Thirdly, the psychiatric profession's belated recognition of the serious implications and extent of tardive dyskinesia has been vividly portrayed to the courts by lawyers opposed to the "orgy" of involuntary drug treatment. [p. 1385]

Concerns about litigation may perpetuate irrational behavior. For example, a clinician who has minimized TD for years and has not talked to patients treated with neuroleptic drugs about tardive dyskinesia rationalizes that it is best not to bring up the subject now and risk being asked, "Why didn't you tell me sooner?" On the other hand, it seems logical to the clinician who overreacts to TD to follow the advice of legal counsel and routinely require written informed consent for maintenance neuroleptic drugs. This can occur even though the clinician recognizes that such consent is not required elsewhere in medicine for the appropriate use of approved drugs and is aware of the inherent clinical and legal limitations of written informed consent for ongoing pharmacotherapy.

We need to look beyond the medical-legal concerns, however, to understand fully the basis for the continued overreaction to and minimization of TD. We believe the problem lies in psychiatry's continuing inability to integrate itself fully as a *medical* discipline. Psychiatrists have not routinely had to face the possibility that severe adverse effects may result from powerful but effective treatments for serious medical illness. Internists and surgeons, on the other hand, routinely weigh the risks of proposed treatments against their benefits and frequently do not hesitate to recommend a potentially dangerous treatment for a severe disorder if the likelihood of its efficacy is high. Patients are generally willing to accept the risks entailed in being treated for life-threatening diseases. Psychiatrists are less familiar with the concept of considering a cost-benefit ratio for their treatments. The public has not been educated to view psychiatric illnesses as serious diseases requiring medical treatment. If our ambivalence continues, then, compounded by public perception, it will be impossible for us to deal rationally with schizophrenia and its pharmacotherapy.

There are four basic concepts about schizophrenia and its long-term treatment that psychiatry and, in turn, the public have not fully accepted, despite our increasing knowledge. Until these concepts are fully integrated into daily practice, the extremes of overreaction and minimization seem inevitable.

1. Schizophrenia Is a Serious Brain Disease. While this is a concept dating back at least to Kraepelin, and for which there is now overwhelming evidence, there is a strong tendency to continue to view schizophrenia as a disease of the mind (i.e., a psychological reaction to certain key developmental stresses) or as a disorder resulting from a dysfunctional family or society. We do not intend to minimize the role of environmental or psychosocial stressors on the course and outcome of schizophrenia. The view of schizophrenia as a mind disease, a nebulous concept often associated with a lack of will, however, tends to minimize its severity and make it difficult to justify the use of neurotoxic drugs in its treatment. Our patients and their families know from experience, however, that the impact of schizophrenia is as dreaded as that of cancer and other severe medical illness (Torrey 1983). The psychiatric literature confirms the tremendous morbidity and mortality of this disease.

2. **"Neuroleptics Are Very Helpful When Used Properly"** (Jeste and Wyatt 1982, p. 9). On one hand, Jeste and Wyatt have tremendously understated the effectiveness of neuroleptic drugs. Neuroleptic drugs have revolutionized the practice of psychiatry and have had a profound effect on the lives of schizophrenic patients worldwide. On the other hand, neuroleptics provide only symptomatic control of the predominantly positive symptoms of schizophrenia; they do not offer a cure. While palliation rather than cure is the outcome of most medical treatments, we suggest that psychiatric training in general, and psychoanalytic thinking in particular, make cure—by way of personality restructuring—the goal. Thus, our professional discipline may still minimize the tremendous value of neuroleptic drugs.

3. **Despite Their Demonstrated Efficacy, There Are Limits to the Value of Neuroleptic Drugs in the Treatment of Schizophrenia.** A significant proportion of patients who improve on neuroleptic drugs continue to have persistent negative symptoms of schizophrenia such as poverty of speech, anergia, and amotivation (Csernansky, Kaplan, and Hollister 1985). While these negative symptoms are generally not improved by neuroleptic drugs, it is not uncommon for psychiatrists to increase neuroleptic dose in the hope of alleviating these symptoms. Similarly, psychiatrists frequently increase drug dosage in patients with persistent positive symptoms (i.e., hallucinations and delusions) in the hope of achieving increased antipsychotic effects. While a trial of high-dose neuroleptic treatment may be reasonable for patients with persistent psychotic or negative symptoms, high doses should not be maintained over time in the absence of a clearly increased benefit. Striving for the lowest effective maintenance dose is likely to decrease the prevalence and morbidity of long-term side effects.

A minority of schizophrenic patients show no improvement when treated with neuroleptic drugs. Using neuroleptic drugs in the absence of any beneficial response is inappropriate.

4. **Neuroleptic Drugs Can Cause Serious Side Effects.** Tardive dyskinesia is a significant problem and is relatively common. Between 20 and 30 percent of patients treated with neuroleptic drugs over time are likely to develop tardive dyskinesia (Jeste and Wyatt 1982). The majority of cases are mild, and reversibility of TD may be much more likely than was previously believed. Nonetheless, psychiatrists who commonly prescribe neuroleptics are likely to see irreversible movement disorders develop in some of their patients.

Conclusions

If the field of psychiatry can accept that schizophrenia is a brain disease that is devastating to its victims in terms of morbidity, mortality, and family burden, and if the profession can accept the enormous benefits as well as the limitations and potential side effects of neuroleptic drugs, then perhaps we can prescribe these drugs appropriately and deal with tardive dyskinesia dispassionately instead of defensively. Then we can follow rational guidelines, such as those suggested by the APA Task Force, on the use of neuroleptic drugs and on the prevention and management of TD. As we learn more about TD, management can change appropriately to suit that growing knowledge.

By acknowledging and overcoming our persistent defensiveness, we will be able to ensure that the psychiatric profession, and not the courts, will continue to establish rational standards of care.

References

Arieti, S. Individual psychotherapy of schizophrenia. In: Arieti, S., ed. *American Handbook of Psychiatry.* Vol. 3. New York: Basic Books, 1974. pp. 627–651.

Baldessarini, R.J.; Cole, J.O.; Davis, J.M.; Gardos, G.; Preskorn, S.H.; Simpson, G.M.; and Tarsy, D. *Report of the American Psychiatric Association Task Force on Late Neurological Effects of Antipsychotic Drugs.* Washington, DC: APA Task Force Report, 1979.

Csernansky, J.G.; Kaplan, J.; and Hollister, L.E. Problems in classification of schizophrenics as neuroleptic responders and nonresponders. *Journal of Nervous and Mental Disease,* 173:325–331, 1985.

Davis, J.M. Overview: Maintenance therapy in psychiatry: I. Schizophrenia. *American Journal of Psychiatry,* 132:1237–1245, 1975.

Gardos, G., and Cole, J.O. Maintenance antipsychotic therapy: Is the cure worse than the disease? *American Journal of Psychiatry,* 133:32–36, 1976.

Gualtieri, C.T., and Sprague, R.L. Preventing tardive dyskinesia and preventing tardive dyskinesia litigation. *Psychopharmacology Bulletin,* 20:346–348, 1984.

Jeste, D.V., and Wyatt, R.J. *Understanding and Treating Tardive*

Dyskinesia. New York: Guilford Press, 1982.

Kline, N.S. On the rarity of "irreversible" oral dyskinesia following phenothiazines. *American Journal of Psychiatry,* 124:48–54, 1968.

Munetz, M.R., and Roth, L.H. Informing patients about tardive dyskinesia. *Archives of General Psychiatry,* 42:866–871, 1985.

Munetz, M.R.; Roth, L.H.; and Cornes, C.L. Tardive dyskinesia and informed consent: Myths and realities. *Bulletin of the American Academy of Psychiatry and the Law,* 10:77–88, 1982.

Munetz, M.R., and Schulz, S.C. Screening for tardive dyskinesia. *Journal of Clinical Psychiatry,* 47:75–77, 1986.

O'Connor, J. Issues in the treatment of chronic mental patients. *Hospital & Community Psychiatry,* 35:989–994, 1984.

Stone, A.A. The new paradox of psychiatric malpractice. *New England Journal of Medicine,* 21:1384–1387, 1984.

Torrey, E.F. *Surviving Schizophrenia: A Family Manual.* New York: Harper and Row, 1983.

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Expressed Emotion in Families

Expressed Emotion in Families: Its Significance for Mental Illness, authored by Julian Leff and Christine Vaughn, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). The discovery that discharged schizophrenic patients who returned home to parents or spouses often fared worse than those living alone led researchers to look for conditions within the family that might influence the schizophrenic patient's condition. At the vanguard of this endeavor was George Brown, whose recognition of the debilitating effect of high levels of expressed emotion—such as hostility, criticism, and overinvolvement—has stimulated important insights as well as considerable controversy. In this volume, Leff and Vaughn, together with two other prominent investigators, address some of the confusions and misconceptions that have arisen regarding the measures of expressed emotion and the techniques for obtaining data, and present important new findings which significantly expand Brown's original insights. The book will be of great interest to psychiatric researchers as well as to all mental health professionals who work with schizophrenic patients and their families.

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Season of Birth: Schizophrenia and Bipolar Disorder

by Jeffrey H. Boyd, Ann E. Pulver, and Walter Stewart

Abstract

Studies investigating the association between the risk of schizophrenia and season of birth are reviewed and the association clearly established. This association cannot be explained on the basis of age-incidence or age-prevalence artifacts. Other studies suggest there may be an association between bipolar disorder and season of birth. The leading theory in explaining the season of birth phenomenon is that a seasonal factor (such as viral infection, malnutrition, vitamin deficiency, prenatal or obstetrical complications, or ambient temperature) can damage an infant's brain and thereby predispose the child to later development of psychosis. Evidence suggests that the seasonal effect is associated with a subgroup of schizophrenics who have early onset of psychosis, less genetic loading than other schizophrenics, and better prognosis. Case-control studies are needed comparing winter-born to nonwinter-born schizophrenics.

Since 1929, it has been reported that more schizophrenics are born during the winter and early spring than during other seasons (Tramer 1929). Although the basis for this finding is unknown, it has been suggested that it may be a clue to the identification of a subtype of schizophrenia (Dalen 1975). Some studies also indicate that this is true for patients with bipolar affective disorder (mania) (Hare, Price, and Slater 1974; Dalen 1975; Hare 1975*a*).

The purpose of this paper is fivefold: (1) to review the literature on the relationship between season of birth and schizophrenia; (2) to discuss whether this association is valid or due to artifact; (3) to review the relationship between season of birth and bipolar affective disorder; (4) to review the evidence that season of birth distinguishes a subtype of schizophrenia; and (5) to review the theories offered to explain the basis for the association and to suggest ways to test them.

Investigations of the Relationship Between the Risk for Schizophrenia and Season of Birth

Table 1 provides a summary of reports in English of investigations of the seasonal distribution of births of schizophrenic patients. Season of birth studies have been done in 14 different countries. Despite the variety of study methods and possible differences in diagnostic criteria, strikingly consistent results have been found.

The studies with a large *n* (over 1,000) are based on case registries of a State or nation, with the exception of Koehler and Jacoby's study (1976). Case registry data may be considered a representative sample of hospitalized schizophrenic patients.

In all but two of the studies (Jones and Frei 1979; Pulver, Sawyer, and Childs 1981), the seasonal distribution of births of schizophrenics has been compared with that of births in the general population; a goodness of fit test is then applied. Pulver, Sawyer, and Childs (1981) calculated the probability for being diagnosed as schizophrenic using the census population for the denominators of the rates. Jones and Frei (1979) used matched controls.

Several sources of error should be considered in evaluating these

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Table 1. Schizophrenia season of birth

Place, time of study	Sample size	Adequate controls[1]	Adjustment for age-incidence effect?	Months of excess	Percent excess	Statistical significance	Comment	Reference
USA 1917–1980	536 twins	no	no	Jan–Mar	4	N.S.		Kendler 1982
Minnesota, USA 1963–1978	3,556	yes	yes	Dec–Mar	7	< .001		Watson et al. 1982
Philippines 1975	3,508	no	no	Dec–Feb	15	< .01		Parker & Balza 1977
Ireland 1972–1975	4,855	yes	no	Apr–June	11	< .001		O'Hare, Walsh, & Torrey 1980
Missouri, USA 1966–1975	10,363	yes	yes	Dec, Feb	9	< .01		Lewis & Griffin 1981
Australia 1974	467	no	no	Winter[2]	—	< .01	Males	Jones & Frei 1979
Australia 1974	448	no	no	Winter[2]	—	N.S.	Females	Jones & Frei 1979
Australia 1970–1974	1,061	no	no	Winter[2]	—	N.S.	Males	Parker & Nielson 1976
Australia 1970–1974	1,195	no	no	Winter[2]	12	< .01	Females	Parker & Nielson 1976
USA 1973–1974	53,584	yes	no	Jan, Mar, Apr, May	—	< .05		Torrey, Torrey, & Peterson 1977
Denmark 1972–1974	207	no	no	Jan–Mar	15	< .03	Urban, high genetic risk	Machon, Mednick, & Schulsinger 1983
Japan 1897–1973	5,431	yes	no	Apr	12	< .05	Born ≥ 1900	Shimura & Miura 1980
Japan 1897–1973	2,529	yes	no	May	20	< .01	Born < 1900	Shimura & Miura 1980
England 1970–1973	9,760	yes	yes	Jan–Mar	7	< .001		Hare 1975*a*; Hare 1975*b*
Denmark 1969–1972	7,427	yes	no	Jan–Apr	—	< .001		Videbech, Weeke, & Dupont 1974
West Germany 1962–1972	1,576	yes	no	—	—	N.S.		Koehler & Jacoby 1976

England 1970-1971	5,139	yes	no	Jan-Mar	7	< .01		Hare, Price, & Slater 1974
South Africa 1971	1,713	yes	no	Winter²	6	< .05	Not chronic	Dalen 1975
South Africa 1971	1,234	yes	no	Winter²	—	N.S.	Chronic patients	Dalen 1975
Monroe County, NY 1969-1971	378,404	yes	yes	Winter	—	< .001	Females	Pulver, Sawyer, & Childs 1981
Australia 1961-1971	6,291	no	no	—	—	N.S.		Krupinski, Stoller, & King 1976
Norway 1970	19,740	yes	no	Jan-Mar	20	< .001		Ødegård 1974
London, England 1967-1969	40	no	no	Apr-June	100	< .001	High genetic risk	Shur 1980
London, England 1967-1969	106	no	no	—	—	N.S.	Low genetic risk	Shur 1980
Maryland, USA 1963-1968	1,455	yes	yes	Jan-Apr	8	< .01		Pulver, et al. 1983
Sweden 1962-1968	19,820	yes	no	Jan-Apr	0-10	< .01		Dalen 1975
Missouri, USA 1967	22	no	no	—	—	N.S.		Woodruff, Guze, & Clayton 1974
London, England 1951-1963	3,596	no	no	Jan-Apr	13	< .01		Hare & Price 1968
Maryland, USA 1944-1961	94	no	no	Jan-Mar	35	< .05	Blacks only	Gallagher, McFalls, & Jones 1983
Canada 1959	3,617	yes	no	Jan, Apr, May	—	< .05		Norris & Chowning 1962
Sweden 1949	2,232	no	no	—	—	N.S.		Dalen 1975 (Laestadius)
Virginia, USA 1949	1,469	no	no	—	—	N.S.		Pile 1951
Massachusetts USA, 1933-1945	1,453	yes	no	Jan-Apr	—	< .01		Barry & Barry 1961
USA 1938	10,000	no	no	Jan-Mar	—	—	Graph only	Dalen 1975 (Huntington)

Table 1. Schizophrenia season of birth—Continued

Place, time of study	Sample size	Adequate controls[1]	Adjustment for age-incidence effect?	Months of excess	Percent excess	Statistical significance	Comment	Reference
Holland 1934	2,589	no	no	Nov–Mar	—	—	Graph only	Dalen 1975 (Nolting)
Illinois, USA 1934	3,467	?	no	Dec–Jan	—	—	Graph only	Dalen 1975 (Petersen)
Germany 1931	3,976	no	no	—	—	N.S.		Dalen 1975 (Lang)
Connecticut, USA 1907–1931	6,751	no	no	—	—	N.S.	Upper class patients	Barry & Barry 1964
Switzerland 1876–1927	3,100	no	no	Dec–Mar	15	< .001	Psychosis	Dalen 1975 (Tramer)

[1] Controls matched to patients on years of birth and geographical region of birth.
[2] Winter in the southern hemisphere

studies. Since the seasonal distribution of births in the general population may vary from year to year and from region to region, it is necessary to choose controls whose place and time of birth match those of the cases. Furthermore, since infants who do not survive cannot have schizophrenia, the seasonal effects of infant mortality on the distribution of live births must be considered.

To increase the power of the analysis, many investigators grouped the dates of birth into quarters of the year (e.g., January through March, April through June). This could mask a true seasonal pattern; more schizophrenics than expected are likely to be born in December, but if October and November lack an excess, the fourth quarter may fail to show any excess.

The season of excess risk is shown in the fifth column of table 1. Studies in both the northern and southern hemispheres show an excess risk for the winter months. This is especially significant in terms of statistical artifacts, such as the age-incidence and age-prevalence effects, which result from the use of calendar time and not age of risk. In studies with a sample size over 1,500, the excess ranges from 0 to 20 percent, with a mean of about 10 percent.

Of the 30 studies, 19 showed differences that were significant at a level of .05. Six of these studies are significant at a level of .001. The only two studies that had a control group matched to patients on year of birth and geographical region of birth, and that also controlled for the age-incidence effect, found significant results at the .01 level (Hare 1975*a*, 1975*b*; Lewis and Griffin 1981).

In summary, table 1 shows that a majority of studies have found an association between the risk for schizophrenia and birth in the winter months. This is true in populations in both the northern and southern hemispheres. Although this finding has not been universal (Pile 1951; Barry and Barry 1964; Dalen 1975; Koehler and Jacoby 1976; Krupinski, Stoller, and King 1976; Parker and Nielson 1976; Jones and Frei 1979; Kendler 1982), there is a remarkable degree of consensus given the wide variety of study settings and methods.

This difference in the cumulative risk for relatively small differences in the age period of risk is the source of the age-incidence and age-prevalence effects.

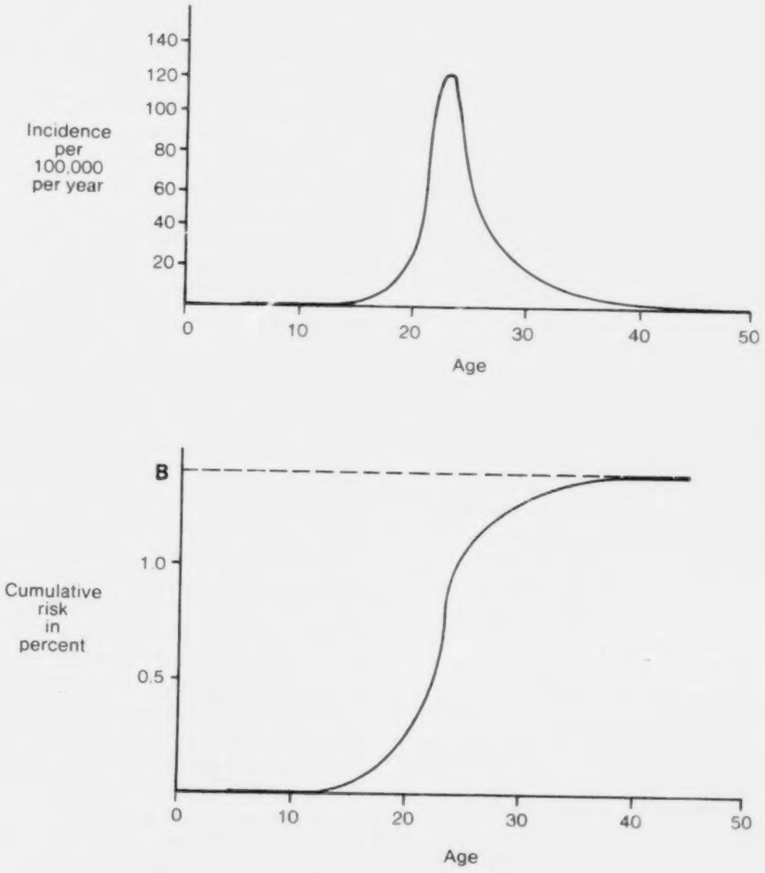
The problem arises because calendar time, not age, is the basis for deriving cumulative measures of risk. For those people born in a given year (Y1) and first hospitalized in another year (Y2), those born in January of year Y1 will be 11 months older by the end of year Y2 than those born in December of year Y1. This age difference, depending on month of birth, is the basis of both the age-incidence effect (Dalen 1975; Hare 1978; Pulver, Sawyer, and Childs 1981) and the age-prevalence effect (Lewis and Griffin 1981). Hypothetical age-incidence and age-prevalence curves are shown in figure 1.

Validity of the Association

Several investigators have suggested that the apparent association may be due to artifact (Hare, Price, and Slater 1974; Dalen 1975; Lewis and Griffin 1981). In more recent studies, however, artifacts have been disposed of by appropriate statistical techniques; the resulting pattern is often more significant (Hare, Price, and Slater 1974; Hare 1978; Pulver, Sawyer, and Childs 1981; Watson et al. 1982; Pulver et al. 1983; Shur and Hare 1983).

The most recent challenge is that of Lewis and Griffin (1981). They propose that the increased risk is due to a statistical artifact that they call the "age-prevalence effect," a measure that is the sum (or integral) of risk a defined population will experience over a given period of time. The older a person is, the greater is the cumulative risk of disease. The cumulative risk of schizophrenia is relatively high for the age period 17 to 24—so high, in fact, that there are differences in the cumulative risk for small differences in age. For example, a cohort of 20-year-olds born in January will have a significantly higher cumulative risk of schizophrenia than a cohort of 20-year-olds born in June when the cumulative risk is integrated to the same calendar time for both cohorts.

Figure 1. Age-incidence curve and cumulative risk curve



Note.—Hypothetical data.

Age-Incidence Effect. On the ascending side of the age-incidence curve (top of figure 1), those who are 11 months older will have a higher incidence rate. On the descending side of the age-incidence curve, those who are 11 months older will have a lower incidence rate. Therefore, the ascending and descending sides of the age-incidence curve produce opposite (but not equal) effects.

Age-Prevalence Effect. The cumulative risk curve (lower part of figure 1) ascends but never descends. Therefore, those who are 11 months older will have a higher cumulative risk for the onset of schizophrenia.

The age-prevalence effect (or cumulative-risk effect) has a simple mathematical relationship to the age-incidence effect: at a given age it represents the area under the age-incidence curve. This relationship can be expressed as follows: If age is *a*, incidence rate for that age is *I(a)*, and the cumulative risk for that age is *R(a)* then

$$R(a) = \int_{0}^{a} I(a)\, da$$ [1]

At any given age the cumulative-risk that a person will have developed schizophrenia by that age is the lifetime prevalence of schizophrenia for that age group. This is why Lewis and Griffin referred to the cumulative-risk effect as an age-prevalence effect. The cumulative-risk curve plateaus at a line that we have marked "B" on the bottom of figure 1. This point, "B", represents the lifetime risk that a person will develop schizophrenia.

Correction factors have been suggested to adjust for both the age-incidence effect (Hare 1978; Pulver, Sawyer, and Childs 1981) and the cumulative-risk effect (Lewis and Griffin 1981; Pulver, Sawyer, and Childs 1981, 1983; Watson et al. 1982; Shur and Hare 1983). Lewis and Griffin (1981) introduced a crude and not clearly defined method to correct for the difference in exposure to the period of risk, depending on month of birth; they found that the season of birth phenomenon vanished from their data. They note, however, that the age-prevalence effect cannot account for the presence of the season of birth phenomenon in the southern hemisphere. Since the calendar year in the southern hemisphere is also divided at the December 31–January 1 point, according to their argument the excess risk of schizophrenia should occur for cohorts born between January through March, and not July through September. In fact, the excess births of schizophrenics in the southern hemisphere occur in the winter months, which in the southern hemisphere are July through September.

Similarly, the cumulative-risk effect could not explain an increased risk for schizophrenia for individuals in the northern hemisphere born in December (Pulver, Sawyer, and Childs 1981), the month of birth cohort that—by Lewis and Griffin's 1981 definition—should have the lowest expected risk (Shensky and Shur 1982; Watson et al. 1982). A recent study has employed Lewis and Griffin's statistical adjustment for the age-prevalence effect, and the results show that a significant season of birth effect remains (Watson et al. 1982). Another study shows that the season of birth effect persists even when a calendar year is defined as July to June (Shur and Hare 1983).

One could completely eliminate both the age-incidence effect and the age-prevalence effect by defining the study population in a different way. This could be accomplished by defining the study population on the basis of month of age at admission to a hospital, rather than as all patients admitted to a psychiatric facility during a specific calendar time. Pulver et al. (1983) have reported results of an investigation in which the study population was defined by month of age at admission. Using standard life table techniques, the probability of being diagnosed as schizophrenic over the same specified age period for individuals born in each month was calculated. There was an increased risk for winter-born individuals to be diagnosed as schizophrenic.

Investigations of the Relationship Between the Risk for Bipolar Disorder and Season of Birth

The question of a season of birth effect for affective disorders has been examined by a number of investigators (Barry and Barry 1961; Hare and Price 1968; Hare, Price, and Slater 1974; Ødegård 1974; Videbech, Weeke, and Dupont 1974; Woodruff, Guze, and Clayton 1974; Dalen 1975; Hare 1975*a*; Parker and Nielson 1976). The data do not suggest a seasonal effect in affective disorders.

There is reason to reexamine this question vis-à-vis bipolar disorder (Hare, Price, and Slater 1974; Dalen 1975; Hare 1975*a*). Most of the studies have focused on manic depressive illness rather than bipolar disorder. These diagnoses have quite different meanings; it has been reported that between 15 and 32 percent of manic depressives have had a manic episode (Krauthammer and Klerman 1979) while the remainder have had depression without mania. Since the winter excess of births for depressives is either nonexistent (Barry and Barry 1961; Ødegård 1974; Videbech, Weeke, and Dupont 1974; Parker and Nielson 1976) or muted relative to that of manics (Hare, Price, and Slater 1974; Dalen 1975; Parker and Nielson 1976), the effect of studying manic depressive illness has been to obscure the question of whether manics (bipolars) have a distintive seasonal birth pattern.

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[1] For those not familiar with mathematical notation, the symbols "∫" and "d" stand for the "integral" function. In other words, the cumulative risk curve is the "integral" of the incidence curve.

Table 2 shows the results of studies of the season of birth in bipolars. In the Australian study, no significant excess of manic patients was born in the winter—June, July, August—(Parker and Nielson 1976). A significant excess of manic patients born in the first quarter of the year was found in two studies with overlapping populations reported by Hare and others (Hare, Price, and Slater 1974; Hare 1975*a*). The other reports in table 2 are different aspects of one study conducted by Dalen in Sweden (1975). He found that there was a seasonal pattern to manic patients born in some years but not in others. The years 1921 to 1930 and 1942 to 1950 did show this seasonal phenomenon at the .01 level of statistical significance. The other years between 1901 and 1940 showed an excess, but it did not reach statistical significance.

In summary, there may be a seasonal excess of risk for bipolar disorder for those born between January and March and possibly in April. The evidence is not as strong as that for schizophrenics, but this may be due in part to the far smaller sample sizes for mania. If there is a winter excess of bipolar births, the magnitude of the excess could be as great as that for schizophrenia: table 2 shows over 10 percent excess winter births.

Table 2. Bipolar disorder season of birth

Place, time of study	Sample size	Adequate controls[1]	Adjustment for age-incidence effect?	Months of excess	Percent excess	Statistical significance	Comment	Reference
Australia 1970–1974	220	no	no	—	—	N.S.		Parker & Nielson 1976
England, Wales 1970–1973	1,183	yes	yes	Jan–Mar	13	< .05		Hare 1975a
England, Wales 1970–1971	533	yes	no	Jan–Mar	21	< .01		Hare, Price, & Slater 1974
Sweden 1962–1968	318	yes	no	Jan–Apr	4	< .01	Born 1941–1950	Dalen 1975
Sweden 1962–1968	453	yes	no	Jan–Apr	19	< .01	Born 1921–1930	Dalen 1975
Sweden 1962–1968	1,949	yes	no	—	—	N.S.	Born 1901–1940	Dalen 1975

[1] Controls matched to patients on years of birth and geographical region of birth.

One plausible explanation of the seasonal pattern in both psychoses is that those born in the winter months may represent a subtype of patients who may be diagnosed either as manic or schizophrenic. Perhaps winter birth predisposes to an atypical psychosis that sometimes mimics schizophrenia and sometimes mimics bipolar disorder. A second possibility is that these seasonal patients are given both diagnoses at different times. In a followup of the Iowa 500 patients, 3 percent of those originally diagnosed as schizophrenic were rediagnosed as bipolar 30 to 40 years later, and 13 percent of those originally diagnosed bipolar were later rediagnosed as schizophrenic (Tsuang et al. 1981). A third possibility is that the season of birth phenomenon represents a necessary but not sufficient cause for both these disorders.

Evidence Suggesting That Season of Birth May Be a Clue to a Distinctive Group of Schizophrenic Patients

It has been proposed that schizophrenia is a heterogeneous disorder analogous to mental retardation. Accordingly, even without knowing the biological meaning of the season of birth effect, the idea that it is a clue to the identification of a distinct group of schizophrenics should be tested. Some evidence favoring this hypothesis has already been reported.

Sex Differences. Seven investigators subdivided their samples of patients by sex (Roche and Dalen 1974; Dalen 1975; Parker and Nielson 1976; Parker and Balza 1977; Jones and Frei 1979; Pulver, Sawyer, and Childs 1981; Pulver et al. 1983). Five reported a sex difference in the season of birth effects; four of the five found the seasonal effects to be more marked among the females.

Chronicity. There is some evidence that the winter-born form of schizophrenia is a less chronic form of the disease. Dalen noted a more marked seasonal effect for patients hospitalized less than 3 years in his Swedish sample but not in a sample of South African patients (Dalen 1975). Pulver et al. (1983) divided their population according to duration of first psychiatric hospitalization (more than 180 days and 180 days or less). Patients hospitalized 180 days or less were more likely to be born in the winter; patients hospitalized more than 180 days were not. Unfortunately, the sample size for those hospitalized more than 180 days was small. Watson et al. (1984), however, have shown that the season of birth effect is more pronounced among unmarried, presumably severe schizophrenics.

Genetic Risk. There is some evidence that season of birth is important to the etiology of a subgroup of schizophrenic patients who do not have a family history of schizophrenia. Kinney and Jacobsen (1978) divided their sample of 34 schizophrenic patients into (1) patients with a family history of schizophrenia or a history of postnatal brain damage and (2) patients with neither of these factors. The patients with neither risk factor were more likely to be born during the winter months. Shur (1982) divided his sample of 973 schizophrenic patients according to family history of psychiatric illness. The patients with a family history of either severe psychiatric illness or schizophrenia were born less often in the first quarter of the year. Other studies also provide supporting evidence that the season of birth effect plays a larger role in persons who have lesser genetic predisposition (Shensky and Shur 1982; Templer and Veleber 1982).

Machon, Mednick, and Schulsinger (1983) *assume* that there is a positive interaction between genetic predisposition and the season of birth effect. According to their hypothesis, perinatal viral infections specifically attack the nervous systems of genetically vulnerable fetuses. They do not, however, present data to show that the season of birth effect is higher in children at high genetic risk than in children at low genetic risk. In other words, they have not yet presented data to support this assumption. Their assumption of a gene-viral interaction contradicts the weight of published data from studies that have examined this issue (Kinney and Jacobsen 1978; Shensky and Shur 1982; Shur 1982; Templer and Veleber 1982).

Age of Onset. There is some evidence that early onset is associated with winter birth. Pulver, Sawyer, and Childs (1981) provide evidence suggesting that age of onset of schizophrenia (age at first hospitalization) may be used to identify a group of schizophrenic patients who are more likely to have an excess of winter births. Hare (1978) had previously concluded that either age of onset or year of birth may affect the seasonal distribution of birthdates of schizophrenic patients.

The Meaning of the Season of Birth Phenomena

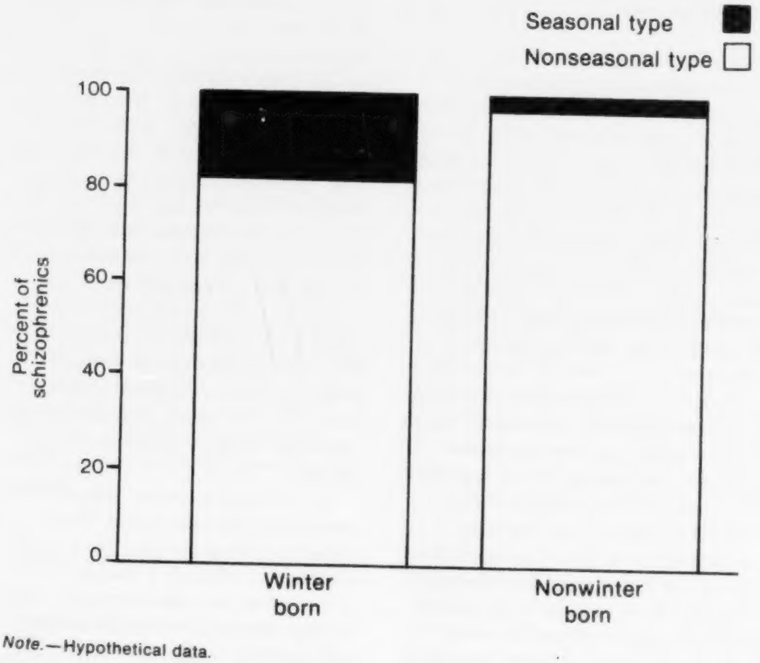
Two theories have been offered to explain the season of birth phenomenon: (1) Some seasonably varying factor occurring during intrauterine life or during the first postnatal months alters the central nervous system, in some way increasing the risk for schizophrenia; or (2) parents of schizophrenics are more likely to conceive in the spring or early summer.

Theory 1: Seasonally Varying Factor Alters the Central Nervous System

The season of birth phenomenon has been of interest primarily because it has been assumed to be evidence that the etiology of some as yet unidentified subtype of schizophrenia is due in part to a seasonal factor, such as viral infection, obstetrical or perinatal complications, malnutrition, vitamin deficiency, or temperature (Dalen 1975; Hare 1979).

If this theory is true, then there should be at least two types of schizophrenia: one type that is related to this seasonal etiologic factor ("seasonal type") and all others ("nonseasonal types"). The same may be true of bipolar affective disorder. The proportion of schizophrenics who are the seasonal type is unknown. The relationship of the seasonal to nonseasonal types may be as illustrated in figure 2. In the figure the seasonal type schizophrenics are shown to be about 5 to 10 percent of all schizophrenics; however, they entirely account for the season of birth phenomenon. The nonseasonal types of schizophrenia, by definition, have the same season of birth distribution as the general population.

Figure 2. Winter-born schizophrenics compared with nonwinter-born schizophrenics



Note.—Hypothetical data.

One way to discover which schizophrenics are the seasonal type is to compare those born in the winter with those born in other times of the year, looking for qualities that distinguish a segment of the winter-born schizophrenics from schizophrenics born at other times. Characteristics that might be studied this way are discussed next.

Brain Damage. All the agents that have been suggested to cause the seasonal type of schizophrenia are thought to do so through some sort of damage to the brain. There is likely to be a spectrum of degrees of brain damage. Some of this damage may be too subtle for detection by ordinary means, but some of it may be discovered by neurological exam, electroencephalogram, or computed axial tomography. Although these procedures have been done on many schizophrenics, we know of no studies in which an attempt has been made to correlate abnormal neurological results with season of birth.

Socioeconomic Status of the Parents at the Time of the Patient's Birth. It is important to stratify schizophrenics on the basis of parental socioeconomic status at the time of the patient's birth, because pregnant women and newborn infants of lower socioeconomic status are disproportionately exposed to almost all the etiologic factors of interest. They have higher risk pregnancies, more obstetrical and perinatal complications, more exposure to viral infection, poorer nutrition, more vitamin deficiency, and more exposure to seasonal temperatures (Bierman et al. 1965; Pasamanick and Knobloch 1971; Frederick and Adelstein 1978; Eisner et al. 1979; Gaziano, Freeman, and Allen 1981). If any of these factors are associated with etiology of the seasonal subtype of schizophrenia, then one might find schizophrenics born to parents of the lower socioeconomic groups to be overrepresented among the winter born. In the general population, socioeconomic status is not associated with an excess of winter births (Dalen 1975), and the socioeconomic status of parents of schizophrenics does not differ from that of the general population. In summary, we are suggesting that although parental socioeconomic status is not clearly associated with the risk for schizophrenia, except perhaps in large cities (Kohn 1973), nevertheless it may be associated with the risk for the seasonal subtype of schizophrenia.

Gallagher, McFalls, and Jones (1983) present data to show that the season of birth effect is found among black rather than white schizophrenics. This is taken as evidence of a socioeconomic status effect (Gallagher, McFalls, and Jones 1983).

Good Prognosis vs. Poor Prognosis Schizophrenia. As noted above, there is some evidence that winter-born schizophrenics have shorter first hospitalizations (Dalen 1968; Dalen 1975; Pulver et al. 1983). This relationship should be verified in other samples. It will be possible to examine the issue in the United States when data from the third edition of the *Diagnostic and Statistical Manual (DSM-III)* (American Psychiatric Association 1980) are available in State and national registries. In *DSM-III* "schizophrenia" is a diagnosis that should be used only for patients with at least a 6-month duration of that disorder. Thus, many of those previously given this diagnosis in the United States will no longer qualify. Those with a disorder of shorter duration will receive a *DSM-III* diagnosis of "schizophreniform" or "atypical psychosis." Thus, it will be possible to investigate whether the season of birth phenomenon is associated, not with *DSM-III* schizophrenia, but with these other diagnoses. No one has examined this question.

Furthermore, *DSM-III* provides data on premorbid functioning in Axis V. Strauss and Carpenter (1974, 1977) have shown that the level of premorbid functioning is the best indicator of prognosis. The question is whether schizophrenics with a higher level of premorbid functioning are more likely to have been born in the winter than those with a lower level of premorbid functioning. It is a question that could be investigated in a large data set that includes date of birth, *DSM-III* diagnosis, and Axis V information.

Theory 2: Parents of Schizophrenics Are More Likely to Conceive in the Spring or Early Summer

This theory suggests that the parents of schizophrenics and patients with bipolar affective disorder are more likely to conceive in the spring or early summer (Barry and Barry 1961; Hare and Price 1968; Dawson 1978). If that is true, the siblings of schizophrenics and patients with bipolar affective disorders should show the same surplus of winter births (Hare 1976; McNeil, Kaij, and Dzierzykray-Rogalska 1976; Buck and Simpson 1978).

Table 3 shows the results of studies that have tested this hypothesis. Hare found a significant excess of first-quarter births among the siblings of manic-depressive probands (Hare 1976). Hare also found a nonsignificant excess of first-quarter births among the siblings of schizophrenics. This may be due to the small sample size. In a larger sample, however, Buck found a nonsignificant *deficit* in first-quarter births for the siblings of schizophrenics (Buck and Simpson 1978). Not shown on table 3 is a study by Lang that compared season of birth of manic depressive patients with their siblings and found no difference (Hare, Price, and Slater 1974).

The results of these studies are ambiguous but certainly do not prove a seasonal birth pattern in the siblings of schizophrenics. The largest study found negative results (Buck and Simpson 1978). Even if siblings were found to have a winter excess of births, two conceptual problems must be considered:

1. The relationship of season of birth between siblings in the general population must be examined. Let us suppose, for example, that there were always a significant tendency for one sibling to be born in the same season as the other sibling, unrelated to psychiatric diagnosis. This would indicate a seasonal mating pattern for all couples, but it would not provide any evidence for why schizophrenics should have a different season of birth distribution than other people. If schizophrenics have an excess of winter births, based on, for example, viral infections during pregnancy, then one would expect that their siblings would likewise have an excess of winter births by virtue of the seasonal mating pattern of all couples. This line of hypothetical reasoning would lead to the possibility that the siblings of schizophrenics have an excess of winter births, yet the season phenomenon is still due to viral infection in the womb.

2. It would be unclear whether the seasonal births were due to their status as siblings, or whether a small subgroup of siblings is destined later in life to become schizophrenics. The siblings who will eventually develop schizophrenia would be expected to

Table 3. Season of birth in siblings

Place, time of study	Number of probands	Number of siblings	Control group	Findings	Reference
Maudsley Hospital, London, 1967–1971	Not stated	451	Nonpsychotic patients	Siblings of manic depressives have a significant ($p < .01$) excess of births in the first quarter of the year, 20 percent more than expected.[1]	Hare 1976
Maudsley Hospital, London, 1967–1971	Not stated	219	Nonpsychotic patients	Siblings of schizophrenics have 13 percent more births in the first quarter than expected, but the sample is too small to reach statistical significance.[1]	Hare 1976
Sweden 1973	91	332	General population	Siblings of schizophrenics have a nonsignificant excess of births in the first quarter.	McNeil, Kaij, & Dzierzykray-Rogalska 1976
Ontario, Canada 1975	210	777	General population	Siblings of schizophrenics have a nonsignificant deficit (– 7 percent) of births in the first quarter.	Buck & Simpson 1978

[1] The present authors recalculated the data, using a chi square with 1 degree of freedom.

have a winter excess of births, because when they later become schizophrenics, they will have a winter excess of births. In this case a winter excess of births among siblings could indicate that some of them had been exposed to a seasonal factor that damaged their brains and predisposed them to schizophrenia.

Thus, even if a winter excess of births were found among the siblings of schizophrenics, it would not necessarily imply that the season of birth phenomenon is an artifact attributable to a tendency by the parents of schizophrenics to conceive in the spring.

Conclusion

There is substantial evidence that schizophrenics are more likely to be born in the winter than other seasons, even in the southern hemisphere, where the winter months are June, July, and August. Attempts to explain this apparent phenomenon as an artifact have been refuted. When various artifacts (such as the age-incidence or age-prevalence effects) are eliminated or statistically controlled, the excess risk for winter borns remains. There is evidence to suggest that the same winter excess of births may be found in bipolar affective disorder, although more studies are needed to verify the season of birth phenomenon for the bipolar disorder.

If a seasonal factor damages the central nervous system during fetal or perinatal development and thereby predisposes the fetus to later developments of schizophrenia, one might expect to find that the subgroup of schizophrenics exposed to that seasonal factor differs from other schizophrenics. There have been few studies investigating the characteristics that distinguish winter-born schizophrenics from nonwinter-born schizophrenics. There is nevertheless some evidence to suggest that winter-born schizophrenics are more likely to have a better prognosis, less genetic risk for schizophrenia, and an earlier age of onset of the disorder. Although early age of onset is associated with poor prognosis when all schizophrenics are considered together, this relationship does not necessarily hold for all subtypes of schizophrenics. In fact, the seasonal subtype appears paradoxically to have both early onset and a good prognosis, in contrast to the majority of schizophrenics. We know of no studies that have investigated neurologic or radiographic evidence of central nervous system damage or that have investigated parental socioeconomic status in relationship to season of birth; however, these areas need investigation.

The only other theory that has been proposed to explain this phenomenon is that the parents of schizophrenics are more likely to conceive in the spring or early summer. This theory has been investigated by studying the season of birth of siblings of schizophrenics, with ambiguous results. The largest study of siblings found a deficit of births in the winter months. At the present time, there is little evidence to support the theory of increased spring conception by parents of schizophrenics.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Barry, H. III, and Barry, H., Jr. Season of birth: An epidemiological study. *Archives of General Psychiatry,* 5:292–300, 1961.

Barry, H. III, and Barry, H., Jr. Season of birth in schizophrenics: Its relation to social class. *Archives of General Psychiatry,* 11:385–391, 1964.

Bierman, J.M.; Siegel, E.; French, F.E.; and Simonian, K. Analysis of the outcome of all pregnancies in a community. *American Journal of Obstetrics and Gynecology,* 91:37–45, 1965.

Buck, C., and Simpson, H. Season of birth among the sibs of schizophrenics. *British Journal of Psychiatry,* 132:358–360, 1968.

Dalen, P. Month of birth and schizophrenia. *Acta Psychiatrica Scandinavica* (Supplement), 203:55–60, 1968.

Dalen, P. *Season of Birth: A Study of Schizophrenia and Other Mental Disorders.* Amsterdam: Elsevier/North Holland, 1975.

Dawson, D.F. An explanation for the seasonality of births in schizophrenia. *American Journal of Psychiatry,* 135:1434–1435, 1978.

Eisner, V.; Brazie, J.V.; Pratt, M.W.; and Hexter, A.C. The risk of low birthweight. *American Journal of Public Health,* 69:887–893, 1979.

Frederick, J., and Adelstein, P. Factors associated with low birth weight of infants delivered at term. *British Journal of Obstetrics and Gynecology,* 85:1–7, 1978.

Gallagher, B.J. III; McFalls, J.A., Jr.; and Jones, B.J. Racial factors in birth seasonality among schizophrenics: A preliminary analysis. *Journal of Abnormal Psychology,* 92:524–527, 1983.

Gaziano, E.P.; Freeman, D.W.; and Allen, T.E. Antenatal prediction of women at increased risk for infants with low birth weight. *American Journal of Obstetrics and Gynecology,* 140:99–107, 1981.

Hare, E.H. Manic depressive psychosis and season of birth. *Acta Psychiatrica Scandinavica,* 52:69–79, 1975*a*.

Hare, E.H. Season of birth in schizophrenia and neurosis. *American Journal of Psychiatry,* 132:1168–1171, 1975*b*.

Hare, E.H. The season of birth of siblings of psychiatric patients. *British Journal of Psychiatry,* 129:49–54, 1976.

Hare, E.H. Variations in the seasonal distribution of births of psychotic patients in England and Wales. *British Journal of Psychiatry,* 132:155–158, 1978.

Hare, E.H. Schizophrenia as an infectious disease. *British Journal of Psychiatry,* 135:468–473, 1979.

Hare, E.H., and Price, J.S. Mental disorder and season of birth: Comparison of psychoses with neurosis. *British Journal of Psychiatry,* 115:533–540, 1968.

Hare, E.H.; Price, J.S.; and Slater, E. Mental disorder and season of birth: A national sample compared with the general population. *British Journal of Psychiatry,* 124:81–86, 1974.

Jones, I.H., and Frei, D. Seasonal births in schizophrenia: A southern hemisphere study using matched pairs. *Acta Psychiatrica Scandinavica,* 59:164–172, 1979.

Kendler, K.S. The season of birth of schizophrenic, neurotic, and psychiatrically normal twins. *British Journal of Psychiatry,* 141:186–190, 1982.

Kinney, D.K., and Jacobsen, B. Environmental factors in schizophrenia: New adoption study evidence. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment.* New York: John Wiley & Sons, 1978.

Koehler, K., and Jacoby, C. Season of birth and Schneider-oriented diagnosis of schizophrenia, "neurosis" and psychopathy. *Archiv fur Psychiatrie und Nervenkrankheiten,* 223:69–75, 1976.

Kohn, M.L. Social class and schizophrenia: A critical review and a reformulation. *Schizophrenia Bulletin,* 1 (Experimental Issue No. 7):60–79, 1973.

Krauthammer, C., and Klerman, G.L. The epidemiology of mania. In: Shopsin, B., ed. *Manic Illness.*

New York: Raven Press, 1979. pp. 11–28.

Krupinski, J.; Stoller, A.; and King, D. Season of birth in schizophrenia: An Australian study. *Australian and New Zealand Journal of Psychiatry*, 10:311–314, 1976.

Lewis, M.S., and Griffin, P.A. An explanation for the season of birth effect in schizophrenia and certain other diseases. *Psychological Bulletin*, 89:589–596, 1981.

Machon, R.A.; Mednick, S.A.; and Schulsinger, F. The interaction of seasonality, place of birth, genetic risk, and subsequent schizophrenia in a high risk sample. *British Journal of Psychiatry*, 143:383–388, 1983.

McNeil, T.; Kaij, L.; and Dzierzykray-Rogalska, M. Season of birth among siblings of schizophrenics: A test of the parental conception habits interpretation. *Acta Psychiatrica Scandinavica*, 54:267–274, 1976.

Norris, A.S., and Chowning, J.R. Season of birth and mental illness: A critical examination. *Archives of General Psychiatry*, 7:206–212, 1962.

Ødegård, Ø. Season of birth in the general population and in patients with mental disorders in Norway. *British Journal of Psychiatry*, 125:397–405, 1974.

O'Hare, A.; Walsh, D.; and Torrey, E.F. Seasonality of schizophrenic births in Ireland. *British Journal of Psychiatry*, 137:74–77, 1980.

Parker, G., and Balza, B. Season of birth and schizophrenia—An equatorial study. *Acta Psychiatrica Scandinavica*, 56:143–146, 1977.

Parker, G., and Nielson, M. Mental disorder and season of birth—A southern hemisphere study. *British Journal of Psychiatry*, 129:355–361, 1976.

Pasamanick, B., and Knobloch, H. Epidemiologic studies on the complications of pregnancy and the birth process. In: Caplan, G., ed. *Prevention of Mental Disorders in Children*. New York: Basic Books, 1971. pp. 74–79.

Pile, W.J. A study on the correlation between dementia praecox and the month of birth. *Virginia Medical Monthly*, 78:438–440, 1951.

Pulver, A.E.; Sawyer, J.W.; and Childs, B. The association between season of birth and the risk for schizophrenia. *American Journal of Epidemiology*, 114:735–749, 1981.

Pulver, A.E.; Stewart, W.; Carpenter, W.T., Jr.; and Childs, B. Risk factors in schizophrenia: Season of birth in Maryland, U.S.A. *British Journal of Psychiatry*, 143:389–396, 1983.

Roche, S.D., and Dalen, P. The South African sample. In: Dalen, P., ed. *Season of Birth in Schizophrenia and Other Mental Disorders*. Gotesborg: Gotesborg Press, 1974. pp. 95–98.

Shensky, T., and Shur, E. Season of birth of schizophrenics: Cyclic trends and their analysis. *British Journal of Psychiatry*, 141:316–317, 1982.

Shimura, M., and Miura, T. Season of birth in mental disorders in Tokyo, Japan, by year of birth, year of admission and age at admission. *Acta Psychiatrica Scandinavica*, 61:21–28, 1980.

Shur, E. Season of birth in high and low risk schizophrenics. *British and Journal of Psychiatry*, 140:410–415, 1982.

Shur, E., and Hare, E. Age-prevalence and the season of birth effect in schizophrenia: A response to Lewis and Griffin. *Psychological Bulletin*, 93:373–377, 1983.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia. *Archives of General Psychiatry*, 31:37–42, 1974.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia. *Archives of General Psychiatry*, 34:159–163, 1977.

Templer, D.I., and Veleber, D.M. Seasonality of schizophrenic births: Harmful effects or genetic morphism. *British Journal of Psychiatry*, 140:323–324, 1982.

Torrey, E.F.; Torrey, B.B.; and Peterson, M.R. Seasonality of schizophrenic births in the United States. *Archives of General Psychiatry*, 34:1065–1070, 1977.

Tramer, M. Uber die biologische Bedeutung des Geburtsmonates, insbesondere fur die Psychoseerkrankung. *Schweizer Archiv fur Neurologie und Psychiatrie*, 24:17–24, 1929.

Tsuang, M.T.; Woolson, R.F.; Winokur, G.; and Crowe, R.R. Stability of psychiatric diagnosis. *Archives of General Psychiatry*, 38:535–539, 1981.

Videbech, T.; Weeke, A.; and Dupont, A. Endogenous psychoses and season of birth. *Acta Psychiatrica Scandinavica*, 50:202–218, 1974.

Watson, C.G.; Kucala, T.; Ansulski, G.; and Brunn, C. Season of birth and schizophrenia: A response to the Lewis and Griffin critique. *Journal of Abnormal Psychology*, 91:120–125, 1982.

Watson, C.G.; Kucala, T.; Tilleskjor, C.; and Jacobs, L. Schizophrenic birth seasonality in relation to the incidence of infectious diseases and temperature extremes. *Archives of General Psychiatry*, 41:85–90, 1984.

Woodruff, R.A., Jr.; Guze, S.B.; and Clayton, P.J. Psychiatric illness and season of birth. *American Journal of Psychiatry*, 131:925–926, 1974.

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Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work, in cooperation with several community and mental health organizations, has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside."

For more information about the rental or purchase of these videotapes, please contact: **Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.**

The NIMH-DIS in the Assessment of *DSM-III* Schizophrenic Disorder

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by *Javier I. Escobar, Eugenia T. Randolph, Joy Asamen, and Marvin Karno*

Abstract

The National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS) was administered by trained lay interviewers to a sample of 82 outpatients with clinical diagnoses of *DSM-III* schizophrenic disorder. Of these subjects, 77 percent were also diagnosed as suffering from *DSM-III* schizophrenic disorder according to the structured interview (NIMH-DIS) administered by a lay interviewer. The DIS interviews were scrutinized to find the reasons for their discrepancy with the clinical diagnoses. A majority of the DIS-negative schizophrenic subjects acknowledged significant psychopathology in the DIS and missed only one of the six *DSM-III* criteria items for schizophrenia. Test-retest results showed consistency in the subjects' reporting of lifetime schizophrenic symptoms.

The National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS) is a highly structured diagnostic interview developed by Robins and collaborators at Washington University, St. Louis (Robins et al. 1981) that is currently being used in large-scale epidemiological surveys in the United States (Eaton et al. 1981). The DIS offers advantages over other structured interviews: it can be administered by lay interviewers, and it uses a system of probes to explore whether a symptom is related to substance or medication use, physical illness, or injury before scoring it as a positive psychiatric symptom. The DIS yields (through scoring by computer algorithm) *DSM-III* criteria (American Psychiatric Association 1980), Feighner criteria (Feighner et al. 1972), and Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978) diagnoses that are comparable to those made clinically by a psychiatrist (Robins et al. 1981).

Robins et al. (1981) evaluated the DIS on a sample that included 157 psychiatric patients, 24 enrollees in a medical care group, and 10 members of Gamblers Anonymous. Test-retest reliability of the instrument was found acceptable for use in clinical populations, and comparisons between clinicians' and lay interviewers' diagnoses yielded adequate concordance for a number of *DSM-III* diagnoses as measured by the kappa statistic (K) (Robins et al. 1981). A more recent study by Burnam et al. (1983) on 151 outpatients confirmed the comparability of the English and Spanish versions of the DIS.

DIS Assessment of Schizophrenic Disorders

In the Robins et al. (1981) and Burnam et al. (1983) studies, DIS diagnoses of *DSM-III* schizophrenic and schizophreniform disorder made by lay interviewers and by clinicians were found to have adequate, though far from perfect, concordance (K values of about 0.60).

The use of a structured format, such as the DIS, which relies so heavily on the respondent's awareness and cooperativeness, involves particular problems when subjects who have cognitive disturbance or psychotic symptoms are being assessed. With schizophrenic subjects, most of whom tend to remain chronically symptomatic, such assessment may be limited by the subject's lack of insight, poor attention span, suspiciousness, and

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negativism. Moreover, regardless of the population under study, assessment of "lifetime" symptoms is limited by faulty recall (Jenkins, Hurst, and Rose 1979).

A report by Pulver and Carpenter (1983) on the DIS assessment of lifetime psychotic symptoms and a recent study using the DIS on a sample of minority patients (Hendricks et al. 1983) have raised questions about the schedule's sensitivity in detecting schizophrenic symptoms. This is an important concern, since the true prevalence rates of schizophrenic disorders may be underestimated by DIS interviews, which—according to the reports just cited—at least appear to yield a high number of false-negative results. The two studies (Hendricks et al. 1983; Pulver and Carpenter 1983) appear limited by methodological problems. Karno et al. (1984) and Helzer and Robins (1984) have recently commented on these limitations in Pulver and Carpenter's (1983) study. In the report by Hendricks et al. (1983), the few cases of schizophrenic disorder ($n = 11$), the lack of detail on the clarity or adequacy of the clinical assessments, and the use of inexperienced clinicians to make clinical diagnoses are major limiting factors. The number of subjects with clearly elicited clinical diagnoses of *DSM-III* schizophrenic disorder who have also been assessed with a DIS administered by a lay interviewer remains relatively small, and the sensitivity of the instrument in detecting *DSM-III* schizophrenic symptoms and diagnoses needs to be defined further.

Method

To test the sensitivity of the DIS in detecting schizophrenic disorders, we chose a clinical population of veterans for whom significant clinical and historical information was available. The sample consisted of 82 subjects with a clinical diagnosis of *DSM-III* schizophrenic disorder who were participating in a longitudinal, cross-cultural study of sociocultural factors and clinical phenomenology of schizophrenia. The study used a battery of instruments (Social Network Inventory, Symptom Checklist-90, Brief Psychiatric Rating Scale, Global Assessment Scale, and Social Adjustment Scale) in addition to the DIS. All subjects were outpatients at the Veterans Administration Medical Center, Brentwood Division, in Los Angeles, and all were on neuroleptic maintenance. Clinical diagnoses made by treating psychiatrists using *DSM-III* criteria were verified by the research psychiatrist (Javier I. Escobar) with a checklist of *DSM-III* criteria for schizophrenia (criteria A through F). Besides reinterviewing the patient, the psychiatrist also reviewed medical records information available on every subject to scrutinize further symptom duration, chronicity, impairment, and exclusion criteria. Screened for participation in the study were all subjects with a clinical diagnosis of schizophrenia who were registered at the outpatient clinic and who met the following criteria: ethnic background: Hispanic or Anglo; age: between 20 and 42; sex: males only; service era: Vietnam or post-Vietnam. After these pools were exhausted, new patients admitted to the clinic were recruited as they entered. Seventy-five percent of those subjects found eligible for the study signed the consent form and were inducted into the study. Most subjects had been chronically ill. About 80 percent had been ill for 5 years or longer and had been hospitalized at least three times. Most had residual symptoms and many had florid symptoms (e.g., hallucinations and delusions, in spite of their outpatient status.

DIS Interviews. The NIMH-DIS was administered by one of two lay interviewers at baseline. The DIS interviews were done within 1 to 2 weeks after the clinical diagnosis of schizophrenic disorder had been confirmed by the research psychiatrist. The lay interviewers had been rigorously trained by the Los Angeles Epidemiology Catchment Area (ECA) group in accordance with the Washington University group's DIS training guidelines. All the interviews were performed at the outpatient clinic. Although a clinically evaluated Spanish version of the DIS was available, more than 95 percent of the Hispanic subjects chose to be interviewed using the English version of the instrument. A randomly selected subsample ($n = 22$) balanced by ethnicity (11 Hispanic, 11 Anglo) was given the DIS again by the same interviewer who had administered the initial DIS. The range of intervals between the two DIS interviews was less than 6 months for 5 subjects, 7 to 21 months for 6 subjects, and more than 2 years for 11 subjects. The differing periods were intended to provide information on the effects of time elapsed on reporting of lifetime symptoms.

The DIS computer program generates lifetime and current *DSM-III* diagnoses of dysthymic, major depressive, bipolar, substance use, schizophrenic, schizophreniform, phobic, panic, and obsessive-compulsive disorder, as well as somatization, antisocial disorder, anorexia nervosa, tobacco dependence, compulsive gambling, and sexual dysfunction. It also provides a crude estimate of cognitive impairment based on the subject's performance on a "mini-mental" state evaluation (Folstein, Folstein, and McHugh 1975). Diagnoses are presented without hierarchical arrangement so that any diagnosis for which criteria were met is listed in the program's output. Because of the excessive length of the evaluations, the anticipated compliance problems, and the rather limited attention span of many subjects, we decided to omit sections from the DIS that we considered less significant for this patient population (these included phobic, panic, and obsessive-compulsive disorder; anorexia nervosa; tobacco dependence; compulsive gambling; and sexual dysfunction). Thus, *DSM-III* diagnoses for this sample are limited to those listed in table 1.

Table 1. Lifetime DIS-*DSM-III* diagnoses (n = 82)[1]

Diagnoses	Number	Percent
Schizophrenia, schizophreniform:		
A–F are positive; definite diagnosis	49	60
D and/or F are negative; definite diagnosis if hierarchies are ignored	10	12
C criterion is < 6 months but > 2 weeks (schizophreniform)	4	5
Cognitive impairment (definite but mild)	3	4
Manic episode	4	5
Major depressive episode	12	15
Major depressive episode (due to psychosis)	26	32
Bipolar disorder	4	5
Major depression (recurrent)	7	9
Alcohol abuse/dependence	47	57
Barbiturate/sedative/abuse/dependence	5	6
Opioid abuse/dependence	7	9
Amphetamine abuse	4	5
Hallucinogen abuse	7	9
Cannabis abuse	23	28
Somatization	2	2
Antisocial personality	3	4
Antisocial personality (due to schizophrenia)	12	15

[1] Percentages based on definite *DSM-III* diagnoses unless otherwise specified.

Results

DIS-*DSM-III* Diagnoses. The distribution of DIS-*DSM-III* diagnoses for the 82 subjects is presented in table 1. With the exception of schizophrenia (see following), all diagnoses are lifetime "definite" diagnoses. Since the DIS computer algorithm largely ignores hierarchical arrangement of *DSM-III* diagnoses, any disorder for which sufficient criteria are met is listed. As indicated in table 1, many subjects had multiple diagnoses. After schizophrenia, substance abuse/dependence and depression were the most frequently occurring diagnoses. Although many of these symptom clusters had been noted by the clinicians who examined the patients, they were judged, following *DSM-III* decision rules, to be hierarchically "secondary" to the "dominant" diagnoses of schizophrenia. Ethnic differences in the clinical presentation of schizophrenic disorders found in this patient sample have been reported elsewhere (Escobar and Randolph 1983; Escobar et al. 1983).

Seventy-seven percent (n = 63) of the 82 clinically diagnosed *DSM-III* schizophrenic subjects met *DSM-III* criteria for "lifetime" schizophrenic disorder in the DIS interview. DIS-*DSM-III* schizophrenia diagnoses are generated according to three different schemata. (1) Subjects who meet all six *DSM-III* criteria for schizophrenia are considered positive with hierarchies included. (2) Subjects who fail to meet the D criterion (if ever depressed or manic, symptoms of schizophrenia preceded the affective symptoms) or the F criterion (symptoms not due to organic mental disorder), but meet the other criteria, are considered positive with hierarchies ignored. (3) Subjects who meet all criteria except that their symptoms last less than 6 months, but at least 2 weeks, are considered to have schizophreniform disorder. In this sample, 10 subjects had a "definite with hierarchies ignored" diagnosis and 4 had a schizophreniform diagnosis. The rest (n = 49) met all six criteria for definite, lifetime schizophrenic disorder.

Nineteen subjects failed to meet the preceding criteria for a diagnosis of schizophrenia according to the DIS computer algorithm. These subjects will be called DIS-negative schizophrenics in contrast to the 63 subjects diagnosed as schizophrenic by the DIS ("DIS-positive" schizophrenics). A review of medical records showed no notable differences in clinical presentation or psychiatric history between the DIS-negative and DIS-positive schizophrenic subjects.

Of the six criteria required for a schizophrenia diagnosis, criterion A is the most inclusive and the one that lists "pathognomonic" symptoms of schizophrenia such as hallucinations and delusions. Six symptoms [A(1) through A(6)] alone or in combination are specified here, of which at least *one* must be present for a definite diagnosis. Criteria B through F deal with secondary aspects of the syndrome such as impairment, duration of symptoms, and presence of competing syndromes (exclusion criteria). The DIS-*DSM-III* criteria for schizophrenia are presented in table 2. These items are incorporated in the DIS computer algorithm used to derive a diagnosis of schizophrenia.

Table 2. DIS-*DSM-III* criteria for schizophrenia

Criterion	DIS Descriptor
A(1)–A(6)	Characteristic delusions, hallucinations, or thought disorder. One or more symptoms must be positive in the following list: (1) bizarre delusions; (2) somatic, grandiose, religious, or nihilistic delusions (counted only if volunteered); (3) persecutory delusions if accompanied by hallucinations; (4) voices commenting or conversing; (5) auditory hallucinations on several occasions; (6) thought disorder (detected only if current)
B	Deterioration of function
C	Current signs of illness as well as evidence for chronicity
D	Either no depressive or manic syndrome or the depression or mania (if present) developed after any psychotic symptoms or was brief relative to psychotic symptoms
E	Age of first characteristic symptom: criterion symptom occurred before age 45 (or had a 2-year prodromal period and subject was under 47)
F	Symptoms not due to organic mental disorder

Table 3 presents in detail the *DSM-III* criteria profiles of the 19 subjects who failed to meet the criteria for schizophrenia. As can be seen in the summary calculations at the bottom of the table, a majority of the subjects who missed a DIS diagnosis of schizophrenia met each criterion item except the A and B criteria. Nine of the 19 DIS-negative subjects (47 percent) missed a schizophrenic diagnosis because they failed to meet either the B criterion ($n = 6$) or the A criterion ($n = 3$) alone.

Table 4 presents a breakdown of the reasons (as specified in the DIS computer algorithm) for failure to meet the diagnostic criteria. Data in this table illustrate more clearly the manner in which subjects generally fail to meet the DIS-*DSM-III* criteria for schizophrenia. With regard to criterion B, nine subjects had no perception of a decline in personal functioning since symptoms first began. Four additional subjects had denied ever having any symptoms from criterion A, so their functional deterioration vis-à-vis is a specific symptom appearance could not be assessed. Clinicians generally decide on criterion B from historical information and observations derived from interviews with a patient but tend to rely less on the patient's perception of his or her own impairment in contrast to the DIS. A review of medical records of those subjects who scored negatively on this item showed clear evidence of a significant personal and functional deterioration since their symptoms first appeared.

In addition to criterion B, the other criterion item most often missed was criterion A. Less than 5 percent ($n = 4$) of the subjects, however, showed what may be called "massive" denial—that is, did not acknowledge having ever had a single A criterion symptom in spite of florid symptoms previously elicited by clinicians and documented in their medical records. Ninety-five percent of the subjects thus acknowledge at least one lifetime delusion or hallucination in the DIS interview. Seven subjects (8.5 percent of the total sample) only partially reported symptoms for criterion A; that is, they admitted having had paranoid delusions but reported no hallucinations, or they reported hallucinations other than auditory ones (see table 4).

The DIS-negative schizophrenics still reported significant symptomatology in the DIS. Eighty percent of those subjects who did not meet the full criteria for schizophrenia met at least five of the six criterion items. Only 11 percent (9 of 82) of the subjects in the total sample met four or fewer diagnostic criteria for schizophrenia. Moreover, of the DIS-negative subjects, 32 percent ($n = 6$) met all criteria except criterion B. If this criterion were omitted, 85 percent of the clinically diagnosed schizophrenic subjects (instead of 77 percent) would also have been diagnosed as having *DSM-III* schizophrenic disorders by the DIS computer algorithm.

Table 3. DIS-negative schizophrenic patients

Patient number	Criteria met						Total number of criteria met	Criteria not met
	A	B	C	D	E	F		
110	X		X	X	X	X	5	B
120		X	X	X	X	X	5	A
123	X		X	X	X	X	5	B
127		X	X		X	X	4	A,D
129	X		X	X	X	X	5	B
131		X		X	X	X	4	A,C
132						X	1	A,B,C,D,E
134		X	X	X	X	X	5	A
204		X	X	X	X	X	5	A
208						X	1	A,B,C,D,E
214	X		X	X	X	X	5	B
215						X	1	A,B,C,D,E
216	X		X	X	X	X	5	B
218			X	X	X	X	4	A,B
220			X	X	X	X	4	A,B
221	X			X	X	X	4	B,C
231						X	1	A,B,C,D,E
232	X		X	X	X	X	5	B
238	X	X	X		X	X	5	D
Total	8	6	13	13	15	19		
Percent	42	32	68	68	79	100		

A diagnostic change between the first and the second interviews was observed in only 2 of the 22 subjects. One of the original DIS-positive subjects became DIS-negative because of his inconsistent responses on criterion A; that is, he acknowledged having hallucinations and delusions at the first interview but denied such in the second interview, which took place only 30 days later. One of the DIS-negative subjects became DIS-positive in the second interview when he acknowledged symptoms from criterion A that he had denied the first time.

Three of four DIS-negative subjects remained negative after the second interview, and they missed a diagnosis of schizophrenia in both interviews because of their responses to criterion A. Although all three subjects acknowledged at least one psychotic symptom (paranoid delusions in two subjects and visual hallucinations in the third), they did not meet *DSM-III* criteria according to the DIS computer algorithm because criterion A [subcriteria A(1) through A(5)] requires nonpersecutory delusions, or paranoid delusions accompanied by hallucinations of any type, or hallucinations that are auditory unless they are accompanied by delusions. Since only one of the six subcriteria listed under criterion A is needed for a diagnosis of schizophrenia, even in the absence of reported symptoms necessary to elicit subcriteria A(1) through A(5), the clinician can often use criterion A(6): "incoherence, marked loosening of associations, markedly illogical thinking, or marked poverty of content of speech if associated with at least one of the following: (a) blunted, flat, or inappropriate affect, (b) delusions or hallucinations (any), or (c) catatonic or other grossly disorganized behavior" (pp. 188–189). This subcriterion can be largely impressionistic, and decisions on its presence are based on a mental status evaluation, behavioral observations, and all historical information available on the subject. The DIS administered by the lay interviewer, therefore, cannot properly elicit criterion A(6) on a lifetime basis, and this may be another major factor contributing to false-negative DIS diagnoses of schizophrenia.

Discussion

Psychiatric diagnosis remains in a state of flux, reflecting the need for more precise and replicable

Table 4. Clinically diagnosed *DSM-III* schizophrenics who did not meet *DSM-III* schizophrenia criteria in DIS interview

DSM-III criterion	Subjects not meeting criteria (*n*)		Reasons for not meeting criteria
A(1)–A(6)	11	4 →	Denied hallucinations and delusions or thought disorder was not detected; did not meet criteria A(1)–A(6)
		6 →	Acknowledged delusions with persecutory content but these were not accompanied by hallucinations; 2 reported visual hallucinations not accompanied by delusions; did not fully meet criterion A(3)
		1 →	Reported possible persecutory delusions but interviewer judged it to be realistically based
B	13	4 →	Criterion B could not be determined (no symptoms from criterion A)
		9 →	Felt they were not less able to work or socialize since criterion A symptoms began
C	6	4 →	Criterion C could not be determined (no symptoms from criterion A)
		2 →	Reported they went "back to normal" for 1 year or had no delusions/hallucinations during the past year
D	6	4 →	Criterion D could not be determined (no symptoms from criterion A)
		2 →	Delusions/hallucinations occurred only in relation to depression or mania
E	4	4 →	Criterion E could not be determined (no symptoms from criterion A)
F	0	—	

diagnostic labels. The advent of operational systems such as *DSM-III* brought hope that the days of intuitive, open-ended diagnostic formulations were numbered. In implementing *DSM-III*, however, a major and not sufficiently recognized problem is that *DSM-III* criteria require clinicians to conceptualize mental disorders in a way that often deviates from ways that have been taught through generations of psychiatric training. Because changes over preceding nomenclatures (e.g., *DSM-II;* American Psychiatric Association 1968) have been extensive, implementation of the new system is necessarily in a transition phase. Clinicians are just beginning to understand fully the new system's demands for careful evaluation, probing, and strict adherence to the operational criteria.

Structured interviews, such as the DIS, that incorporate the criteria and probing of *DSM-III* help make diagnoses more operational. The use of structured interviews in clinical settings increases reliability of psychiatric diagnoses, diminishes interviewer bias, and allows for a more systematic evaluation of symptoms than often occurs in "routine" clinical evaluations. Although the DIS was not originally devised as a clinical instrument, its use in clinical settings seems an effective way to enforce *DSM-III*'s probing requirements. The clinical relevance of the DIS is illustrated by recent studies of subjects with esophageal dysfunction (Clouse and Lutsman 1983), posttraumatic stress disorder (Escobar et al. 1983), and bulimia (Hudson, Pope, and Jones 1983). The systematic probing required by the DIS, however, with its rigidity, relative lengthiness, and repetitiousness may be met with resistance from clinicians. In the absence of structured probing of *DSM-III* symptoms, agreement among clinicians in the face of similar clinical phenomena may continue to be rather low, in spite of the availability of operational diagnoses. It is, therefore, questionable in our view to use clinically derived diagnoses as the absolute yardstick for evaluating the validity of instruments such as the DIS unless assurances are provided that the operational criteria are followed strictly by fully trained diagnosticians.

Assessment of disorders such as schizophrenia, in which the exploration of affect, thought content, behavioral deviance, and attitudes is so crucial, requires good rapport, astute observation, and probing systems that may vary depending on individual subjects, the nature of the interaction, and so forth. In sum, it requires training and clinical skills. This is particularly important when assessing criteria A and B for schizophrenia. Therefore, it is not surprising to find that these two criteria are the ones most frequently "missed" in the DIS interviews. In view of the lack of insight that is characteristic of schizophrenic disorders and the presence of traits such as suspiciousness, information provided by schizophrenic subjects is not always reliable. Moreover, in assessing "lifetime" symptoms, memory distortions and lack of recall are common difficulties even with nonpsychotic populations and more factual events (Jenkins, Hurst, and Rose 1979). In this context, the results of this study are quite supportive of the utility of a DIS administered by lay workers in eliciting symptoms and operational criteria for schizophrenic disorder in clinical settings.

Among potential shortcomings of this study is the possibility of observer's bias, since the interviewers knew that all subjects had been diagnosed as schizophrenic before the DIS interview. We believe such a possibility is remote not only because of the highly structured nature of the instrument, but also because interviewers were told that the interview's purpose was to distinguish between DIS-positive and DIS-negative subjects and to identify "problem" items from among those eliciting schizophrenic symptoms.

The observation that two of the criteria for schizophrenia (criteria A and B) account for most false-negative diagnoses provides the opportunity of improving the DIS diagnosis of schizophrenia through slight modifications in how diagnostic criteria are elicited by the instrument. Currently, a committee of ECA investigators is carefully considering modifications of the DIS that may improve its clinical validity in assessing schizophrenic and other *DSM-III* disorders.

References

American Psychiatric Association. *DSM-II: Diagnostic and Statistical Manual of Mental Disorders*. 2nd ed. Washington, DC: The Association, 1968.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: American Psychiatric Association, 1980.

Burnam, A.; Karno, M.; Hough, R.; Escobar, J.I.; and Forsythe, A. The Spanish Diagnostic Interview Schedule: Reliability and comparison with clinical diagnoses. *Archives of General Psychiatry*, 40:1189–1196, 1983.

Clouse, R.E., and Lutsman, P.J. Psychiatric illness and contraction abnormalities of the esophagus. *New England Journal of Medicine*, 309:1337–1342, 1983.

Eaton, W.W.; Regier, D.A.; Locke, B.Z.; and Taube, C.A. The Epidemiologic Catchment Area Program of the National Institute of Mental Health. *Public Health Reports*, 96:319–325, 1981.

Escobar, J.I., and Randolph, E.T. "Ethnicity and Symptoms of Schizophrenia in the U.S." Paper presented at the American Psychiatric Association's meeting, New York, NY, May 1983.

Escobar, J.I.; Randolph, E.T.; Puente, G.; Spiwak, F.; Asamen, J.; Hill, M.; and Hough, R. Posttraumatic stress disorder in Hispanic Vietnam veterans—Clinical phenomenology and socio-cultural factors. *Journal of Nervous and Mental Disease*, 171:585–596, 1983.

Feighner, J.P.; Robins, E.; Guze, S.B.; Woodruff, R.A.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 26:57–63, 1972.

Folstein, M.D.; Folstein, S.E.; and McHugh, P.R. Mini-Mental State: A practical method for grading cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12:189–198, 1975.

Helzer, J., and Robins, L.N. Lifetime psychotic symptoms assessed with the DIS. *Schizophrenia Bulletin*, 10(1):4–5, 1984.

Hendricks, L.E.; Bayton, J.A.; Collins, J.L.; Mathura, C.; McMillan, S.; and Montgomery, T. The NIMH Diagnostic Interview Schedule: A test of its validity in a population of black adults. *Journal of the National Medical Association*, 75:667–671, 1983.

Hudson, J.I.; Pope, H.G.; and Jones, J.M. Treatment of bulimia with antidepressants: Theoretical considerations and clinical findings. *Psychiatry Annals*, 13:965–969, 1983.

Jenkins, C.D.; Hurst, M.W.; and Rose, R.M. Life-changes: Do people really remember? *Archives of General Psychiatry*, 36:379–384, 1979.

Karno, M.; Burnam, A.; Escobar, J.I.; and Timbers, D. Lifetime psychotic symptoms assessed with the DIS. (Letter to the Editor) *Schizophrenia Bulletin*, 10:154–156, 1984.

Pulver, A.E., and Carpenter, W.T., Jr. Lifetime psychotic symptoms assessed with the DIS. *Schizophrenia Bulletin*, 9:377–382, 1983.

Robins, L.N.; Helzer, J.E.;

Croughan, J.; and Ratcliff, K.S. National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry,* 38:381-389, 1981.

Spitzer, R.L.; Endicott, J.; and Robins, E. Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry,* 35:773-782, 1978.

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Predictors of Outcome in Schizophrenia: The Process-Reactive Dimension

by Martin Harrow, Jerry F. Westermeyer, Marshall Silverstein, Billie S. Strauss, and Bertram J. Cohler

Abstract

Premorbid social-sexual competence and the process-reactive dimension, as assessed by the Phillips Scale, were investigated as predictors of posthospital adjustment in a prospective study of two samples of 141 young, early phase schizophrenics (a private hospital sample and a state hospital sample). The Phillips Scale predicted subsequent outcome at significant levels for one of the two samples of *DSM-II* schizophrenics (a "broad" construct of schizophrenia). It did not predict outcome as well for *DSM-III* schizophrenics (a "narrow" construct of schizophrenia). Mixed results emerged from separate analyses of first-admission schizophrenics only and for male schizophrenics only. The data indicated that marital status accounts for some of the positive relationship between the Phillips Scale and later outcome. A more focused measure of prehospital social adjustment successfully predicted social functioning at followup for both *DSM-II* and *DSM-III* schizophrenics ($p < .05$). There were some significant and near-significant relationships, but overall results suggest that when the influence of chronicity and marital status is reduced, the relationship between the Phillips Scale and subsequent outcome is less robust than was once thought.

Concepts about the role of premorbid social adjustment in schizophrenia have been central to theoretical formulations about this disorder. In particular, the process-reactive dimension, based on distinctions between good and poor premorbid social and sexual adjustment and measured by part I of the Phillips Scale (Phillips 1953), has been among the most important ways of categorizing schizophrenia in the psychological literature. Literally thousands of studies have been based on the process-reactive dimension and on good vs. poor premorbid social adjustment—two related, but not necessarily identical, concepts (DeWolfe 1968; Garmezy 1970; Fowles et al. 1970; Higgins 1971; Chapman and Chapman 1973; Evans, Goldstein, and Rodnick 1973; Bromet, Harrow, and Kasl 1974; Strauss and Carpenter 1974; Putterman and Pollack 1976; Quitkin, Rifkin, and Klein 1976).

The relationship between the process-reactive dimension and outcome bears directly on theories about the origin of schizophrenia. Formulations about the importance of the process-reactive dimension are based on theories about a very poor clinical course among process schizophrenics.

However, evidence on posthospital adjustment supporting the predictive utility of the distinction between good and poor premorbid adjustment using a prospective research design is surprisingly scant in recent studies. The current research was designed to provide new data in this area. There have been only a handful of modern outcome studies finding positive evidence (Gittleman-Klein and Klein 1969) or mixed evidence (Evans, Goldstein, and Rodnick 1973) in support of the process-reactive dimension, and to date none have used the new diagnostic systems (*DSM-III;* American Psychiatric Association 1980). A number of older investigations have found positive results (Farina and Webb

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1956; Farina et al. 1962; Query and Query 1964; Nuttal and Solomon 1965; Cancro and Sugarman 1968; DeWolfe 1968; Stephens, O'Connor, and Weiner 1969). These investigations used broad concepts of schizophrenia (*DSM-II;* American Psychiatric Association 1968); and research with modern, narrow concepts of schizophrenia (e.g., *DSM-III*) is needed as well.

A major problem in research on the process-reactive dimension is that the results from some studies may have been influenced by the use of retrospective research designs and/or the use of chronic, long-term patients who have already demonstrated years of poor outcome. In these cases, significant findings regarding the Phillips Scale may, in part, be influenced by characteristics portraying the severity of an already developed illness rather than *premorbid* adjustment (Strauss and Carpenter 1974). In contrast to older views, recent *prospective* studies using multiple measures of outcome and less chronic samples of schizophrenics have found less positive results (Bromet, Harrow, and Kasl 1974; Strauss and Carpenter 1974).

The current research studied the relationships between the process-reactive dimension and subsequent outcome, while reducing the potential confounding effects of previous chronicity, by analyzing young, early phase schizophrenics. Chronic, older schizophrenics with many years of poor functioning, who can contaminate prognostic results, were not included in the current sample. In addition, multiple measures of outcome were used (i.e., social, work, and symptoms) as well as a measure of rehospitalization. It is now widely recognized that various assessments of different types of symptoms and functioning are preferable for a complete, accurate outcome measure (Strauss and Carpenter 1972, 1974).

To increase the generalizability of the findings, the current research employed two samples of young, early phase schizophrenics—one from a state hospital type of setting and the other from a private hospital setting. The following questions were studied:

1. Does the process-reactive dimension, as defined by Phillips Scale, predict outcome in young, contemporary schizophrenics, including those defined according to both older, broader diagnostic concepts of schizophrenia (*DSM-II*) and more modern, narrow (*DSM-III*) concepts of schizophrenia?
2. Does the process-reactive dimension predict outcome for (a) a subsample of only schizophrenic men and (b) a subsample of first-admission schizophrenics?

Method

Patient Sample. The present research represents part of the Chicago Followup Study, a larger program studying schizophrenic thought pathology, prognosis, and outcome on a longitudinal basis by examining these features across different phases of the schizophrenic disorder (Westermeyer and Harrow 1984; Harrow, Carone, and Westermeyer 1985; Harrow and Marengo, in press; Pogue-Geile and Harrow 1985) and exploring mechanisms that may be involved in schizophrenic thought pathology and psychosis (Harrow and Miller 1980; Harrow and Quinlan 1985; Lanin-Kettering and Harrow 1985).

To assess the predictive utility of the process-reactive dimension using several different concepts of schizophrenia, two patient samples were studied. Each sample consisted of a group of patients meeting a broad concept of schizophrenia (*DSM-II*) and a group meeting a narrow concept of schizophrenia (*DSM-III*).

Sample 1. The first sample consisted of 95 young, early phase *DSM-II* schizophrenics from a private hospital, Michael Reese Hospital (MRH). Thirty-four of the 95 patients also fit the *DSM-III* criteria for schizophrenia (American Psychiatric Association 1980). Sixty-one of the patients who were classified as having schizophrenia using *DSM-II* did not meet the *DSM-III* criteria for the disorder. Previous research by the present investigators has suggested that many patients from this type of population are concurrently experiencing affective disturbances (Silverstein et al. 1982). The diagnoses of these 61 patients using *DSM-III* criteria were as follows: 4 patients had schizophreniform disorders, 4 patients had paranoid or atypical disorders, 9 patients had manic disorders, 28 patients had major depressive disorders, 10 patients had schizoaffective disorders, and 6 other patients had varied diagnoses.

The *DSM-II* diagnoses were assigned during hospitalization, based on a detailed admission interview, extensive hospital records, and an additional semistructured, tape-recorded interview described previously (Grinker and Holzman 1973). The major focus in this report is on the process and reactive schizophrenics using *DSM-II* criteria, since *DSM-II* schizophrenics have been the basis of almost all of the process-reactive literature, especially previous reports in this area. The *DSM-III* diagnoses were based on the admission interviews, the tape-recorded research interviews, hospital records, and the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978).

The 95 schizophrenic patients consisted of 50 men and 45 women. The mean age of this young, early phase sample was 22.7 years; 48 percent were first-admission patients. Using parental social class (Hollingshead and Redlich 1958), more than 75 percent of sample 1 were brought up in upper-middle-class homes. The mean educational level of sample 1 was 13.6 years at hospitalization. Only 13 of these young, early phase schizophrenics (14 percent) had ever married. The 95 schizophrenics in sample 1 included 35 "reactive" schizophrenic patients (Phillips score below 13), 24 schizophrenics with intermediate scores (scores from 13 to 17), and 36 "process" schizophrenics (scores above 17).

Sample 2. The second sample consisted of 46 *DSM-II* schizophrenics from the Illinois State Psychiatric Institute (ISPI). According to specific *DSM-III* criteria, the total sample of 46 *DSM-II* schizophrenics included 24 *DSM-III* schizophenics and 22 patients with other types of *DSM-III* diagnoses. These latter 22 patients included 7 patients with schizophreniform disorders or paranoid disorders, 8 with manic disorders, 5 with major depressive disorders, and 2 with schizoaffective disorders (American Psychiatric Association 1980). Diagnostic procedures and methods were the same in both hospital settings.

The patient group at ISPI also consisted of young, relatively early phase schizophrenics. The mean age at admission of sample 2 (ISPI) was 22.6 years, and the mean educational level was 12.5 years at hospitalization; 32 percent were first-admission patients. There were 30 men and 16 women. The 46 schizophrenic patients in sample 2 (ISPI) included 15 "reactive" schizophrenics (Phillips score below 13), 16 schizophrenics with intermediate scores (scores from 13 to 17), and 15 "process" schizophrenics (scores above 17).

The patients in sample 2 (ISPI) did not significantly differ from patients in sample 1 (MRH) in age, sex, previous hospitalizations, prior social adjustment (SADS scale), or process-reactive scores ($p > .10$).

In line with typical differences between private hospital and state hospital populations, sample 2 had fewer years of education than sample 1 ($p < .05$). In addition, a greater percentage of the state hospital schizophrenics were from parental social class 4 or 5 (using the Hollingshead and Redlich [1958] socioeconomic status system) than was the case for the schizophrenics from the private hospital (60 percent vs. 25 percent). Previous research, however, has suggested that parental social class may not be a strong predictor of overall outcome in similar types of schizophrenic samples (Westermeyer and Harrow 1984). Nevertheless, because of these differences and because the two populations represented samples from different types of institutions, with possibly different prognoses, the relationship between the process-reactive dimension and outcome was analyzed separately for each of the two samples.

For both sample 1 (private hospital) and sample 2 (state hospital), there were no significant differences between process and reactive schizophrenics in age, sex, education, or social class.

Medication Status. At the time of followup evaluation, 46 percent of the reactive schizophrenics and 52 percent of the process schizophrenics were receiving neuroleptic medications. The reactive schizophrenics receiving neuroleptics at followup had significantly poorer overall outcomes than the reactive schizophrenics not receiving neuroleptics ($p < .05$). The process schizophrenics receiving neuroleptics at followup showed a nonsignificant trend toward poorer overall outcomes than the process schizophrenics not receiving neuroleptics ($p < .06$). It is possible that results suggesting less adequate posthospital functioning among patients who were receiving neuroleptics may be a consequence of more negative-outcome schizophrenics being placed on medication because of their poorer clinical condition. Consequently, caution is warranted in interpreting the results comparing different medication groups, because patients were not assigned to their medication groups on a random basis.

Assessment of Prehospital Functioning and Outcome. All patients from sample 1 and sample 2 were studied prospectively as inpatients (Harrow et al. 1983; Marengo and Harrow 1985), and later followed up in detail as part of the Chicago Followup Study (Harrow et al. 1978; Harrow, Silverstein, and Marengo 1983; Harrow, Marengo, and McDonald 1986). Procedures for assessing predictors and outcome were the same in both hospital settings (sample 1 and sample 2).

Following traditional instrumentation in this area, part I of the Phillips Scale of Premorbid Social-Sexual Adjustment was used as the major predictor instrument of the process-reactive dimension (Phillips 1953). This scale has been modified slightly to make it more applicable to patients under 30 years of age and to women (Bromet, Harrow, and Kasl 1974). Scoring of the Phillips Scale was based on prospective administration of a modified version of the General Information Questionnaire, developed by DeWolfe to rate the

Phillips Scale (DeWolfe 1968) on extensive case history material and on the taped interview.

In addition, a 7-point scale assessing premorbid social relations was taken directly from the Schedule for Affective Disorders and Schizophrenia (SADS), a widely used and standardized schedule (Endicott and Spitzer 1978). Although most of the literature in this area has not reported evidence on interrater reliability, we assessed this factor on our major measures of premorbid adjustment. The results showed satisfactory interrater reliability for these measures ($r = .81$ for Phillips Scale, and $r = .72$ for the SADS Premorbid Social Adjustment Scale).

Outcome assessments were made an average of 2.4 years after hospital discharge. Posthospital adjustment was assessed in a structured interview. The followup schedule involved detailed evaluation of the following: (1) social, work, and family functioning; (2) patients' use of medication, psychotherapy, and other possible treatment; (3) neurotic, depressive, and psychotic symptomatology; and (4) rehospitalization.

Two scales of overall adjustment were used. The first was an overall scale (the LKP Scale) developed and used by Klein and associates (Levenstein, Klein, and Pollack 1966) that has also been used by our research team and others (Harrow et al. 1978; Summers, Harrow, and Westermeyer 1983; Pogue-Geile and Harrow 1985). This scale is based on work functioning, life disruptions, self-support, symptomatology, potential suicide, relapses, and rehospitalizations. The second scale was an overall measure, based on four separate scales, developed by Strauss and Carpenter (1972, 1974) in their investigations of posthospital functioning. Results are also reported on the four separate scales that measure posthospital work functioning, social functioning, rehospitalization, and symptoms.

Results

Results for Private Hospital Schizophrenics (Sample 1). Table 1 and table 2 present the results for the private hospital (sample 1) and the state hospital (sample 2) schizophrenics. These tables present the correlations between the two major prediction measures (the Phillips Scale and the SADS Scale of Prehospital Social Adjustment) and the various measures of outcome for the full sample of schizophrenics. Separate statistical analyses for the Phillips Scale are also presented for (1) schizophrenic men only, (2) first-admission schizophrenics only, and (3) the sample of *DSM-III* schizophrenics. The following results emerged for sample 1:

- The data for the total sample of schizophrenics in table 1 indicate that the Phillips Scale did not have a close relationship with any measure of outcome at followup ($p > .10$). For *DSM-II* schizophrenics from a private hospital (sample 1), process schizophrenics as defined by the Phillips Scale did not have a significantly poorer outcome than reactive schizophrenics.
- A more focused measure of prehospital social adjustment, the scale from the SADS, did predict good social functioning at followup ($p < .05$) for both the total sample of *DSM-II* schizophrenics and the more narrowly defined sample of *DSM-III* schizophrenics (see table 1).
- Some researchers have suggested the process-reactive dimension may be a better predictor of outcome among men than among women. Analyses using only *DSM-II* schizophrenic men in sample 1 failed to show a significant association between the Phillips Scale and subsequent outcome. Similar nonsignificant relationships were found when schizophrenic women were analyzed separately.
- Analyses of the subsample of first-admission schizophrenics in sample 1 did show a significant relationship between the Phillips Scale and the LKP Scale of overall outcome ($p < .05$). This indicates some limited prognostic utility for the process-reactive dimension, even with young, early phase samples.
- The Phillips Scale was also studied for the subgroup of patients from sample 1 who met the *DSM-III* criteria for schizophrenia. It did not predict outcome at significant levels within this sample of *DSM-III* schizophrenics. The SADS measure of prehospital social adjustment did predict posthospital social functioning for the *DSM-III* schizophrenics ($p < .05$).

Table 3A presents a more detailed breakdown of the outcome data for process and reactive schizophrenics in sample 1 if one looks separately (1) at both the first- and multiple-admission schizophrenics, and (2) at both the *DSM-III* schizophrenics and only the *DSM-II* schizophrenics who did not meet the *DSM-III* criteria for schizophrenia. A three-way ANOVA was conducted for sample 1 (process-reactive dimension, first-admission–multiple-admission schizophrenics, *DSM-III* schizophrenics–*DSM-III* nonschizophrenics). The overall *F* for the process-reactive comparison in this early phase, young schizophrenic sample was not significant for these patients in a private hospital (F (1, 87) = 1.8 ns). This analysis is in accord with the earlier analyses presented. It suggests that, within the private hospital sample, the differences in outcome between process and reactive schizophrenics are not significant, although there is a trend toward poorer outcome among the process schizophrenics.

Table 4 presents detailed data on the Phillips Scale and the LKP Scale of overall outcome for the total sample of schizophrenics in sample 1. The data indicate that a large percent of the process schizophrenics (51 percent) showed very poor outcomes. Among the reactive schizophrenic patients, however, 37 percent also showed very poor outcome. Overall, a smaller percent of the reactive than of the process schizophrenics in sample 1 showed very poor outcome, but group differences were not significant.

Results for State Hospital Schizophrenics (Sample 2). The results in table 2, which reports the correlations between outcome and both the Phillips Scale and the Scale of Prehospital Social Adjustment for the state hospital sample (sample 2), were as follows:

- The data in table 2 indicate that the Phillips Scale predicted, at statistically significant levels, several key aspects of outcome among the full sample of 46 state hospital schizophrenics. Thus, the Phillips Scale successfully predicted both measures of overall outcome ($p < .05$), rehospitalization ($p < .05$), and social adjustment ($p < .05$) at followup among the 46 *DSM-II* schizophrenics in sample 2.
- The Prehospital Social Functioning Scale from the SADS successfully predicted social adjustment at followup (without predicting overall outcome) for the 46 *DSM-II* schizophrenics and the 24

Table 1. Correlations of Phillips Scale with outcome for different types of schizophrenics at private hospital (sample 1)

	Overall outcome at followup		Specific areas of outcome functioning			
Sample assessed	Overall outcome: (LKP Scale)	Overall outcome: (Strauss-Carpenter Scale)	Rehospitalization	Posthospital social functioning	Posthospital work functioning	Posthospital symptoms
Total sample of *DSM-II* schizophrenics ($n = 95$)						
Phillips Scale	.13	.06	.09	.08	.06	−.03
Prior Social Adjustment (SADS)	.04	.13	.14	.24[1]	.08	−.04
First-admission schizophrenics ($n = 46$)						
Phillips Scale	.32[1]	.22	.20	.22	.09	.17
Prior Social Adjustment (SADS)	.22	.32[1]	.26	.30[1]	.17	.22
Male schizophrenics only ($n = 50$)						
Phillips Scale	.05	.02	.06	.16	.01	−.17
Prior Social Adjustment (SADS)	−.02	.12	.10	.21	.09	−.05
DSM-III schizophrenics ($n = 34$)						
Phillips Scale	−.01	.05	.06	.05	.04	.09
Prior Social Adjustment (SADS)	−.14	.13	−.02	.34[1]	.07	.04

[1] $p < .05$.

Note.—Full range of Phillips scores used for all *r*'s. A positive correlation indicates that the relationship is in the direction hypothesized.

DSM-III schizophrenics in sample 2 ($p < .05$). These positive results were similar to those for the private hospital (sample 1) *DSM-II* and *DSM-III* schizophrenics.

- The Phillips Scale successfully predicted three of the six outcome measures among *DSM-II* schizophrenic men in sample 2 ($p < .05$). The Phillips Scale did not predict any of the six outcome variables for schizophrenic women at statistically significant levels, although it did show a nonsignificant trend toward predicting symptom outcome for schizophrenic women ($p < .10$).
- Analyses of the subsample of first-admission schizophrenics in sample 2 revealed that the Phillips Scale and the SADS Scale failed to predict any aspect of outcome at followup. Nine of the 12 correlations studied for the first-admission schizophrenics were $r = .20$ or higher, and 4 were $r = .30$ or higher; but because of the small sample size, these correlations were not significant.

Table 3B also presents a detailed breakdown of the outcome data for process and reactive schizophrenics in sample 2 if one looks separately (1) at the first- and multiple-admission schizophrenics and (2) at both the *DSM-III* schizophrenics and the *DSM-II* schizophrenics who were *DSM-III* nonschizophrenics. The *F* for the main effect involving the process-reactive comparison in this young, early phase schizophrenic sample was significant ($F(1,38) = 6.0$, $p < .05$). The results support the

Table 2. Correlations of Phillips Scale with outcome for different types of schizophrenics at state hospital (sample 2)

	Overall outcome at followup		Specific areas of outcome functioning			
Sample assessed	**Overall outcome: (LKP Scale)**	**Overall outcome: (Strauss-Carpenter Scale)**	**Rehospitalization**	**Posthospital social functioning**	**Posthospital work functioning**	**Posthospital symptoms**
Total sample of *DSM-II* schizophrenics ($n = 46$)						
Phillips Scale	.29[1]	.41[2]	.31[1]	.33[1]	.26	.27
Prior Social Adjustment (SADS)	.15	.24	.03	.32[1]	.20	.12
First-admission schizophrenics ($n = 15$)						
Phillips Scale	.24	.36	.38	.21	.22	.32
Prior Social Adjustment (SADS)	.20	.21	.09	.08	.17	.30
Male schizophrenics only ($n = 30$)						
Phillips Scale	.26	.39[1]	.36[1]	.41[1]	.14	.19
Prior Social Adjustment (SADS)	.12	.15	.12	.26	.06	−.01
DSM-III schizophrenics ($n = 24$)						
Phillips Scale	.12	.40[1]	.32	.57[2]	.24	−.04
Prior Social Adjustment (SADS)	.11	.35	.14	.46[1]	.46[1]	−.16

[1] $p < .05$.

[2] $p < .01$.

Note.—Full range of Phillips scores used for all *r*'s. A positive correlation indicates that the relationship is in the direction hypothesized.

earlier analysis and suggest poorer outcome for process schizophrenics than for reactive schizophrenics from the state hospital setting.

Table 5 presents detailed data on the Phillips Scale of overall outcome for the total sample of state hospital schizophrenics. As can be seen from this table, a significantly larger percent of the process schizophrenics (60 percent) showed a poor outcome than was the case for the reactive schizophrenics. However, one-third of the reactive schizophrenics also showed a poor outcome.

For both sample 1 and sample 2, only a relatively small number of both reactive and process schizophrenics showed a very favorable outcome. Of the reactive schizophrenics in sample 2, however, five (24 percent) showed a very favorable outcome while none of the *DSM-II* process schizophrenics in sample 2 showed a very favorable outcome at followup.

Marriage and Sex as Influences on the Efficacy of the Phillips Scale as a Predictor of Outcome. A separate analysis was conducted to determine whether marriage was an influence on the posthospital results that emerged for the Phillips Scale as a predictor of outcome for the state hospital schizophrenics. Table 6 presents detailed data comparing ever married with never married state hospital schizophrenics according to their overall outcome at followup.

The data in table 6 indicate marital status was a successful predictor of overall outcome among state hospital (sample 2) schizophrenics. All five remitting schizophrenics in sample 2 were previously married. The never married state hospital schizophrenics had significantly poorer outcomes than the once married schizophrenics ($t = 2.83$, $p < .01$). In addition, all five previously married remitting schizophrenics in sample 2 were *reactive* schizophrenics. Thus, the predictive power of the Phillips Scale was strongly associated with marital status for the state hospital schizophrenics.

Table 3. Outcome for process and reactive schizophrenics, according to *DSM-III* diagnosis and first-admission vs. multiadmission patients

3A. Mean outcome scores for private hospital patients (sample 1)[1]

	DSM-III schizophrenics						*DSM-III* nonschizophrenics					
	First-admission			Multiadmission			First-admission			Multiadmission		
Phillips Scale	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD
Reactive schizophrenics (1–14)	10	4.1	3.0	8	6.8	1.8	17	4.5	2.1	11	4.5	2.3
Process schizophrenics (15–30)	5	6.8	2.2	11	5.2	2.5	14	5.8	2.3	19	5.0	2.4

3B. Mean outcome scores for state hospital patients (sample 2)[1]

	DSM-III schizophrenics						*DSM-III* nonschizophrenics					
	First-admission			Multiadmission			First-admission			Multiadmission		
Phillips Scale	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD
Reactive schizophrenics (1–14)	3	6.3	1.2	8	6.5	1.1	3	2.3	2.3	7	3.3	2.1
Process schizophrenics (15–30)	2	7.5	0.7	11	6.3	1.4	7	5.4	2.3	5	6.0	2.0

[1] Higher scores indicate poorer outcome, using the LKP Scale.

Table 4. Overall outcome at followup for process and reactive schizophrenics from private hospital (sample 1)[1]

	Overall outcome		
Phillips Scale	**Good outcome (1,2)**	**Equivocal outcome (3-6)**	**Poor outcome (7,8)**
Reactive schizophrenics (0-14)	8 (17%)	21 (46%)	17 (37%)
Process schizophrenics (15-30)	6 (12%)	18 (37%)	25 (51%)

[1] $r = .13$, NS. (Full range of Phillips scores used for correlation, rather than category scores.)

When the ever married schizophrenics alone were considered, the correlation between the Phillips Scale scores and overall outcome for the state hospital patients was $r = .25$ (NS). The similar correlation for the never married state hospital schizophrenics considered alone was $r = .02$ (NS). Thus, when the influence of marital status was controlled by analyzing once married and never married patients separately, the Phillips Scale's power to predict outcome was reduced.

Marital status failed to predict the LKP Scale of overall outcome for the private hospital schizophrenic sample (sample 1), in contrast to sample 2. It is not altogether clear why marital status was not a good predictor for this sample, although one possible influence may be the smaller percentage of married patients in the private hospital sample (only 14 percent).

Sex (males vs. females) had a minor influence on the relationship between process-reactive schizophrenics and outcome. There was a trend for more male schizophrenics to fit into the process group and more female schizophrenics to fit into the reactive group. This trend was most pronounced among the *DSM-II* schizophrenics and was not prominent for the *DSM-III* schizophrenics. The male process schizophrenics tended to show poorer outcomes than the female process schizophrenics. Among the reactive schizophrenics, the male-female differences in outcome were negligible.

Paranoid vs. Nonparanoid Status. Evans, Goldstein, and Rodnick (1973) have looked at data on outcome in process and reactive schizophrenics in terms of whether the process and reactive schizophrenics were paranoid or nonparanoid. We analyzed our data using this two-dimensional scheme, dividing the combined sample from both hospital groups according to whether the patients were (1) good premorbid paranoids, (2) poor premorbid paranoids, (3) good premorbid nonparanoids, or (4) poor premorbid nonparanoids. The results, using the total *DSM-II* sample of 141 patients, showed a trend similar to that found by Evans, Goldstein, and Rodnick (1973). The good premorbid nonparanoid patients showed the most favorable outcomes, and the poor premorbid paranoid patients showed the poorest overall outcomes. The only overall outcome differences that reached statistical significance, however, involved the more negative outcomes of the poor premorbid paranoid schizophrenics. This group of poor premorbid patients showed significantly poorer outcomes than the poor premorbid nonparanoids ($p < .05$) and the good premorbid nonparanoid groups ($p < .05$), and it tended to show poorer outcomes than the good premorbid paranoid group ($p < .10$).

Analyses of the data for the smaller *DSM-III* sample ($n = 58$) produced similar results. The good premorbid nonparanoid patients had the best overall outcome, and poor premorbid paranoid patients showed the poorest overall outcome,

Table 5. Overall outcome at followup for process and reactive schizophrenics from state hospital (sample 2)[1]

	Overall outcome		
Phillips Scale	**Good outcome (1,2)**	**Equivocal outcome (3-6)**	**Poor outcome (7,8)**
Reactive schizophrenics (0-14)	5 (24%)	9 (43%)	7 (33%)
Process schizophrenics (15-30)	0	10 (40%)	15 (60%)

[1] $r = .29$, $p < .05$. (Full range of Phillips scores used for correlation, rather than category scores.)

Table 6. Overall outcome at followup for ever married and never married schizophrenics (sample 2)[1]

	Overall outcome		
Marital status	**Good outcome (1,2)**	**Equivocal outcome (3-6)**	**Poor outcome (7,8)**
Ever married	5 (42%)	4 (33%)	3 (25%)
Never married	0	15 (44%)	19 (56%)

[1] $t = 2.83$, $p < .01$

although none of the group differences were close to significant. The lack of significant differences occurred partly because the different types of groups were more homogeneous in outcome, in part because some good-outcome *DSM-II* schizophrenics, no longer viewed as "schizophrenic" using *DSM-III* criteria (Pawelski, Harrow, and Grossman, in press), were eliminated. This two-dimensional system of categorizing schizophrenics, which has showed some initial promise, will be looked at more closely in subsequent followups, as the followup period lengthens.

Discussion

From a theoretical standpoint, the most important finding was a suggestion of some relationship between prehospital social-sexual adjustment and later outcome; it seems not a very strong one, however—either just barely significant or nonsignificant. The Phillips Scale was a strong predictor of several aspects of outcome for state hospital *DSM-II* schizophrenics, but it failed to predict all aspects of outcome. These data are in general agreement with our recent results and those of others (Bromet, Harrow, and Kasl 1974; Strauss and Carpenter 1974). For both these other samples, as with the present two samples, the Phillips Scale showed nonsignificant or just significant correlations with overall outcome. Thus, the process-reactive dimension may have only limited predictive validity, or it may have prognostic significance only for specific types of outcome and/or particular groups of patients.

One factor that may play a role in the failure of the Phillips Scale to predict a very high percentage of the variance in modern outcome studies is better control of key factors such as chronic duration of prior schizophrenic illness. When such factors are better controlled, the predictive utility previously reported for this major index of the process-reactive dimension may be markedly reduced. The hypothesis here is that some previously reported positive findings regarding the Phillips Scale, based on older, more chronic samples, may have been influenced by characteristics of the schizophrenics at hospital admission that are a function of the severity of an already developed illness rather than of *premorbid* adjustment. Strauss and Carpenter (1974, 1977) and Carpenter et al. (1978) also note this possibility and cite the powerful effects of chronicity, or prior length of illness, in predicting outcome.

Thus, it is possible that in some previous research the patients showing less adequate premorbid social adjustment were partially deteriorated, chronic schizophrenics. The seemingly poorer adolescent social functioning may not have been an accurate measure of premorbid competence. Rather, it may have been a measure of an *early morbid phase*, representing deficits from early stages of illness.

The current research design also included multiple aspects of outcome rather than relying on a single, global outcome rating or a measure of rehospitalization. It is harder for a prognostic index to predict *multiple* aspects of outcome and adjustment, some of which demand different skills and abilities. It is of some importance that there was a significant relationship between the measure of prehospital social functioning and social adjustment at followup for all groups of privately hospitalized and state hospitalized *DSM-II* and *DSM-III* schizophrenics. Thus, although the SADS Scale of Prehospital Social Functioning (a 7-point continuous scale) does not predict overall adjustment for these schizophrenic groups, it does show significant correlation with the measure of social relations at followup for *DSM-II* and *DSM-III* schizophrenics at both institutions.

These findings are consistent with those of Strauss and Carpenter (1974), who show that prehospital social relations were the most powerful predictors of social adjustment at followup. Strauss and Carpenter's suggestion, supported by the present data, is that one factor with predictive power is the ability of prehospital competence or adjustment in a particular area to predict hospital functioning in the *same* area of adjustment. One could propose that the already present skills and competence developed previously in some area should help a patient show some level of adequacy in that same area after hospital discharge.

The current study is the first prospective research effort (using a variety of outcome measures) to explore the predictive utility of the process-reactive dimension in patient groups using modern, narrow concepts of schizophrenia. It is possible that the Phillips Scale may have less predictive utility in narrowly defined *DSM-III* schizophrenics than in more broadly defined *DSM-II* schizophrenics. Other research by the present investigators has shown *DSM-III* schizophrenics to be a more homogeneous group of patients with a poor prognosis in comparison with *DSM-II* schizophrenics and has indicated that the predictive power of various factors may shift in each group (Westermeyer and Harrow 1984).

The predictive utility of the Phillips Scale varied by private versus state hospital samples. Differences were explored in terms of marital status. A larger percentage of the state hospital patients were married, with much of the significant relationship being influenced by the more positive outcome of the married, reactive schizophrenics.

It is possible that marriage is an important factor that accounts for some positive results previously reported in other studies regarding the process-reactive dimension, since married patients automatically attain better scores than unmarried patients on the Phillips Scale. Two of the five Phillips subscales are heavily weighted by marital status, and several researchers have reported a very high correlation between marital status and the total Phillips score (Farina et al. 1962; Cancro and Sugarman 1968; McCreary 1974). Consequently, marital status may be one important determinant of prior findings, both with the Phillips Scale and the process-reactive dimension and outcome (Klorman, Strauss, and Kokes 1977).

Sample 1 was predominantly an upper-middle-class sample. An upper-middle-class sample is relatively rare in the literature in this area, and especially so in research relating the process-reactive dimension to outcome. Thus, although upper-middle-class patients are one important type of sample, they have been relatively neglected in much of the empirical literature on schizophrenia and on major psychotic disorders.

Although social class and marital status may play some role in the predictive utility of the Phillips Scale, predictive differences for the process-reactive dimension among various groups cannot be explained adequately by these factors alone. A host of factors that influence later outcome may also be confounded with institution and diagnostic categories. One of the difficulties in predicting posthospital adjustment is that premorbid personality is only one of the factors that may influence later posthospital adjustment. Other variables that may be important in outcome are the patients' social network, type of family, type of ecological milieu, types of treatment they receive, chance events in their lives, and other unknown factors.

We should note that although the Phillips Scale showed a moderate relationship to outcome, other types of measures of prehospital competence, such as that of Zigler and Phillips (1961) and of Zigler, Glick, and Marsh (1979), could be better predictors of outcome. The present investigators, using this approach of Zigler and associates, have found evidence that it is a better predictor of outcome for schizophrenics (Westermeyer and Harrow, in press).

The process-reactive dimension does seem to provide a useful classification system in studies of schizophrenic performance on psychomotor tests, in studies on perceptual and cognitive processes, and in several other areas. Because of these positive results, which are based on a number of research reports from a variety of settings, the process-reactive dimension would still appear to have considerable utility in reducing heterogeneity in schizophrenic samples. In addition, the positive findings in other areas suggest that the process-reactive dimension could be linked to theory in important ways.

It has often been overlooked, however, that there has never been strong empirical evidence to support the underlying assumptions about the positive clinical course of adjustment expected for reactive schizophrenics. Higgins (1971), in an important review article, has begun to express some doubts about the overall importance of this dimension, citing less positive results for process versus reactive schizophrenics in some areas with recent, as opposed to older, research. At the time of Higgins' review, however, he was not able to cite many prospective, long-term outcome studies because of the dearth of empirical research in this area.

Current results suggest that there probably is a relationship between the process-reactive dimension and later posthospital outcome but that it is less robust than was once thought. When the potential contaminating effects of an already chronic course of illness are eliminated, it may be more difficult to fit the positive results on the process-reactive dimension concerning performance on various psychological tasks into a larger theoretical network that relates these data to subsequent clinical course and outcome.

References

American Psychiatric Association. *DSM-II: Diagnostic and Statistical Manual of Mental Disorders*. 2nd ed. Washington, DC: The Association, 1968.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

Bromet, E.; Harrow, M.; and Kasl, S. Premorbid functioning and outcome in schizophrenics and nonschizophrenics. *Archives of General Psychiatry*, 30:203–207, 1974.

Cancro, R., and Sugerman, A.A. Classification and outcome in process-reactive schizophrenia. *Comprehensive Psychiatry*, 9:227–232, 1968.

Carpenter, W.T.; Bartko, J.J.; Strauss, J.S.; and Hawk, A.B. Signs and symptoms as predictors of outcome: A report from the International Pilot Study of Schizophrenia. *American Journal of Psychiatry*, 135:940–944, 1978.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia*. New York: Appleton Century Crofts, 1973.

DeWolfe, A.S. Self-reports and case histories of schizophrenic patients: Reliability and validity of Phillips Scale ratings. *Journal of Clinical Psychology*, 24:415–418, 1968.

Endicott, J., and Spitzer, R. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35:837–844, 1978.

Evans, J.R.; Goldstein, M.J.; and Rodnick, E.H. Premorbid adjustment, paranoid diagnosis, and remission: Acute schizophrenics treated in a community mental health center. *Archives of General Psychiatry*, 28:666–672, 1973.

Farina, A.; Garmezy, N.; Zelusky, M.; and Becker, J. Premorbid behavior and prognosis in female schizophrenic patients. *Journal of Consulting Psychology*, 26:56–60, 1962.

Farina, A., and Webb, W.W. Premorbid adjustment and subsequent discharge. *Journal of Nervous and Mental Disease*, 124:612–613, 1956.

Fowles, D.C.; Watt, N.F.; Maher, B.A.; and Grinspoon, L. Automatic arousal in good and poor premorbid schizophrenics. *British Journal of Social and Clinical Psychology*, 9:135–147, 1970.

Garmezy, N. Process and reactive schizophrenia: Some conceptions and issues. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 2):30–74, 1970.

Gittleman-Klein, R., and Klein, D. Premorbid asocial adjustment and prognosis in schizophrenia. *Journal of Psychiatric Research*, 7:35–53, 1969.

Grinker, R.R., Sr., and Holzman, P.S. Schizophrenic pathology of young adults: A clinical study. *Archives of General Psychiatry*, 28:168–175, 1973.

Harrow, M.; Carone, B.J.; and Westermeyer, J.F. The course of psychosis in early phases of schizophrenia. *American Journal of Psychiatry*, 142:702–707, 1985.

Harrow, M.; Grinker, R.R.; Silverstein, M.D.; and Holzman, P.S. Is modern-day schizophrenic outcome still negative? *American Journal of Psychiatry*, 135:1156–1162, 1978.

Harrow, M.; Lanin-Kettering, I.; Prosen, M.; and Miller, J.G. Disordered thinking in schizophrenia: Intermingling and loss of set. *Schizophrenia Bulletin*, 9:354–367, 1983.

Harrow, M., and Marengo, J. Schizophrenic thought disorder at followup: Its persistence and prognostic significance. *Schizophrenia Bulletin*, in press.

Harrow, M.; Marengo, J.; and McDonald, C. The early course of schizophrenic thought disorder. *Schizophrenia Bulletin*, 12:208–224, 1986.

Harrow, M., and Miller, J.G. Schizophrenic thought disorders and impaired perspective. *Journal of Abnormal Psychology*, 89:717–727, 1980.

Harrow, M., and Quinlan, D.M. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, 1985.

Harrow, M.; Silverstein, M.; and Marengo, J. Disordered thinking: Does it identify nuclear schizophrenia? *Archives of General Psychiatry*, 40:756–771, 1983.

Higgins, J. Process-reactive schizophrenia: Recent developments. In: Cancro, R., ed. *The Schizophrenic Syndrome: An Annual Review*. New York: Brunner/Mazel, 1971.

Hollingshead, A.B., and Redlich, R.C. *Social Class and Mental Illness*. New York: John Wiley & Sons, 1958.

Klorman, R.; Strauss, J.S.; and Kokes, R.F. Premorbid adjustment in schizophrenia: Concepts, measures, and implications. Part III. The relationship of demographic and diagnostic factors to premorbid adjustment. *Schizophrenia Bulletin*, 3:214–225, 1977.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorders. *Schizophrenia Bulletin*, 11:2–7, 1985.

Levenstein, S.; Klein, D.F.; and Pollack, M. Followup study of formerly hospitalized voluntary psychiatric patients: The first two years. *American Journal of Psychiatry*, 10:1102–1109, 1966.

Marengo, J., and Harrow, M. Thought disorder: A function of schizophrenia, mania, or psychosis? *Journal of Nervous and Mental Disease*, 173:35–41, 1985.

McCreary, C.P. Comparison of measures of social competency in schizophrenics and the relation of social competency to socioeconomic factors. *Journal of Abnormal Psychology*, 83:124–129, 1974.

Nuttal, R.L., and Solomon, L.F. Factorial structure and prognostic significance of premorbid adjustment in schizophrenia. *Journal of Consulting Psychology*, 29:362–372, 1965.

Pawelski, T.J.; Harrow, M.; and Grossman, L. The construct of schizophrenia: Should a broad or narrow construct be used? In: Grinker, R.R., Sr., and Harrow, M., eds. *Clinical Research in Schizophrenia: A Multidimensional Approach*, in press.

Phillips, L. Case history data and prognosis in schizophrenia. *Journal of Nervous and Mental Disease*, 117:515–525, 1953.

Pogue-Geile, M.F., and Harrow, M. Negative symptoms in schizophrenia: Their longitudinal course and prognostic importance. *Schizophrenia Bulletin*, 11:427–439, 1985.

Putterman, A.H., and Pollack, H.B. The developmental approach and process-reactive schizophrenia: A review. *Schizophrenia Bulletin*, 2:198–208, 1976.

Query, J., and Query, W. Prognosis and progress: A five-year study of forty-eight schizophrenic men. *Journal of Consulting Psychology*, 28:501–505, 1964.

Quitkin, F.; Rifkin, A.; and Klein, D.F. Neurologic soft signs in schizophrenia and character disorders: Organicity in schizophrenia with premorbid asociality and emotionally unstable character disorders. *Archives of General Psychiatry*, 33:845–853, 1976.

Silverstein, M.L.; Warren, R.A.; Harrow, M.A.; Grinker, R.R.; and Pawelski, T. Changes in diagnosis from *DSM II* to *DSM III*. *American Journal of Psychiatry*, 139:366–368, 1982.

Stephens, H.J.; O'Connor, G.; and Weiner, G. Long-term prognosis in schizophrenia using the Becker-Wittman Scale and the Phillips Scale. *American Journal of Psychiatry*, 126:498–504, 1969.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: I. Characteristics of outcome. *Archives of General Psychiatry*, 27:739–746, 1972.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: II. Relationships between predictors and outcome variables. *Archives of General Psychiatry*, 31:37–42, 1974.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia: III. Five year outcome and its predictors. *Archives of General Psychiatry*, 34:159–163, 1977.

Summers, F.; Harrow, M.; and Westermeyer, J. Neurotic symptoms in the postacute phase of schizophrenia. *Journal of Nervous and Mental Disease*, 171:216–221, 1983.

Westermeyer, J.F., and Harrow, M. "Does the Social Competence Scale Predict Outcome in Schizophrenia?" Paper presented at the 91st Annual Meetings of the American Psychological Association, Predicting outcome in schizophrenics and nonschizophrenics of both sexes. *Journal of Abnormal Psychology*, in press.

Westermeyer, J.F., and Harrow, M. Prognosis and outcome using broad (*DSM-II*) and narrow (*DSM-III*) concepts of schizophrenia. *Schizophrenia Bulletin*, 10:624–637, 1984.

Zigler, E.; Glick, M.; and Marsh, A. Premorbid social competency and outcome among schizophrenics and nonschizophrenic patients. *Journal of Nervous and Mental Disease*, 167:478–483, 1979.

Zigler, E., and Phillips, L. Social competence and outcome in psychiatric disorder. *Journal of Abnormal Social Psychology*, 63:264–271, 1961.

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Family Care of Schizophrenia

Family Care of Schizophrenia, authored by Ian R.H. Falloon, Jeffrey L. Boyd, and Christine W. McGill, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). In an ongoing search for the cause (causes) of schizophrenia, the family has often been identified as a prime candidate. Focus on the harmful effects of critical or rejecting family members and deviant communication patterns has obscured the potentially beneficial role of many families in providing support for their schizophrenic members. Though available evidence suggests that intolerance and emotional overinvolvement do heighten the risk of relapse, the authors' thorough review of the literature reveals that only half of the families studied exhibit such attitudes.

Family Care of Schizophrenia focuses on a model developed by the authors for the broad-based community treatment of schizophrenia and other severe forms of mental illness that taps this underutilized potential. Based on the hypothesis that environmental stress is a major factor in the onset and severity of schizophrenic episodes, the model incorporates well-established behavioral techniques to enhance the coping mechanisms and problem-solving abilities of the family. The goal of the program is not merely the reduction of stress that can trigger florid episodes, but also the restoration of the patient to a level of effective social functioning that permits employment and socialization with persons outside the family. Following a thoughtful and highly readable discussion of the rationale behind their approach, the authors present a detailed description of their behavioral family model, buttressed by illustrative transcripts from actual therapy sessions. Central to their strategy is the development of problem-solving skills and social supports for the patient and his or her family—including education about the illness and the effects of neuroleptics—that will enable them to cope not just with potentially threatening behavioral disturbances and other traumatic life events, but also with the stressors of daily life.

As the authors persuasively demonstrate, families can, with proper guidance, be taught to modulate the level of intrafamilial stress, regardless of whether it derives from family tensions or external life events. Their careful exposition of the family care model, coupled with case studies and results from a controlled outcome study, reveal the family to be an important resource in the community management of mental illness. A major contribution to the treatment of schizophrenia, this unique synthesis of systems theory and behavioral techniques will be of interest to psychiatrists, who are familiar with the limitations of present treatment strategies, as well as to family and behavior therapists concerned with the problems posed by major mental illness.

The Early Course of Schizophrenic Thought Disorder

by Martin Harrow, Joanne Marengo, and Cathleen McDonald

Abstract

The current investigation studied the early course of positive thought disorder and its relationship to other aspects of schizophrenic outcome. Forty-eight schizophrenics diagnosed using the Research Diagnostic Criteria (RDC), 51 psychotic nonschizophrenics, and 67 nonpsychotic patients were studied at index hospitalization and at a 1.5-year followup. Most thought-disordered schizophrenics and other psychotic patients showed some reduction in thought pathology between index hospitalization and followup ($p < .02$). A larger percentage of schizophrenics than other psychotic and nonpsychotic showed thought disorder at the acute phase and at followup assessment ($p < .05$), although only a subgroup of 27 percent of the schizophrenics showed *severe* thought disorder at followup. At followup, severely thought-disordered schizophrenics showed residual signs of psychosis and poor functioning in other areas. Positive thought disorder in schizophrenics at followup most often occurred within the context of an unremitted illness and was less frequently due to a new, acute episode of disturbance. Continuously thought-disordered schizophrenics exhibited more severe impairments in life adjustment at 1.5-year followup than did episodic or nonthought-disordered schizophrenics ($p < .02$). The results suggest that severe thought disorder is a persistent characteristic in a subgroup of early schizophrenics.

The present investigation was designed to study the longitudinal course of positive thought disorder in early schizophrenia and to examine its relation to life adjustment. The course of symptoms is of major importance in formulations about the nature of schizophrenia. Longitudinal research tracking the course of schizophrenia and its associated symptoms, however, remains rare. In particular, recent theory has raised questions about the frequency, course, and prognostic significance of positive thought disorder and other positive symptoms in schizophrenia.

Initial research studying positive thought disorder during the acute, in-hospital phase has provided a first step toward defining the course of positive thought disorder in schizophrenia. Hospitalized schizophrenic patients who are in acute states of disorder have shown more severe and pervasive thought pathology than nonschizophrenics in the form of overinclusive thinking, bizarre-idiosyncratic thinking, derailment, neologisms, autism, and other types of deviant verbalizations (Buss and Lang 1965; Chapman and Chapman 1973; Arieti 1974; Harrow and Quinlan 1977, 1985; Andreasen 1979; Johnston and Holzman 1979). More than 50 percent of hospitalized, young adult schizophrenics assessed at acute phases of disturbance show severe positive thought disorder (Marengo and Harrow 1985).

By contrast, studies of the early course of schizophrenia that compare acute phases of disorder with periods of partial recovery (5 to 7 weeks after hospital admission) suggest a reduction in positive thought pathology as acute disorganization diminishes (Shimkunas 1970; Adler and Harrow 1974; Harrow et al. 1982) These data suggest that the acute phase of schizophrenic disorders is associated with the

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severity of the thought disorder observed (Harrow and Quinlan 1977). Decreases in disturbed thinking between acute and partial recovery phases have not always been significant, however, and either some or many schizophrenics continue to evidence positive thought disorder during the partial recovery period (Adler and Harrow 1974; Harrow et al. 1982).

Our followup research on young schizophrenics studied early in the course of disorder has subsequently extended the assessment of thought-disorder symptoms into posthospital periods ranging from 11 months posthospital admission to 3 years after discharge (Harrow et al. 1973; Harrow and Silverstein 1980; Harrow, Silverstein, and Marengo 1983). The results of this research begin to suggest that while many schizophrenics do not show severe thought pathology during the posthospital or less active stages of the disorder, severe positive thought pathology may be found in a subgroup of young schizophrenics at followup. This phenomenon of a subgroup with severe positive thought disorder within the larger group of schizophrenics during a postacute period underlies the reports of significant schizophrenic-nonschizophrenic differences in thought disorder over time (Adler and Harrow 1974; Harrow, Silverstein, and Marengo 1983). As the mixed data at the phase of partial recovery also indicate, however, a large proportion of schizophrenics may not be thought disordered after the acute phase.

These results begin to raise questions regarding differences in the course of thought pathology *within* schizophrenia over time. The research just reported leads to the hypothesis that for a certain percentage of schizophrenics, positive thought disorder is a permanent part of their thinking; for others, severe thought pathology is more strongly associated with periods of acute psychosis and disorganization (Harrow and Quinlan 1977; Holzman 1978). It has not been determined, however, whether the schizophrenics who show severe thought disorder during posthospital followup are reexperiencing severe thought pathology in conjunction with a new, acute illness episode (i.e., remission, followed by relapse, leading to acute disorder and possibly hospitalization) or whether such symptoms appear at followup within the context of a more permanent, unremitted course. Given that views regarding the episodic versus chronic nature of positive schizophrenic symptoms have been increasingly advanced (Strauss, Carpenter, and Bartko 1974; Bleuler 1978; Zubin, Magaziner, and Steinhauer 1983), more careful assessments of within-group thought disorder patterns over the early course of schizophrenia are needed.

In addition, most of the published research on thought disorder in schizophrenia has used a broad concept of schizophrenia rather than modern, narrow concepts, such as those of the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978) and *DSM-III* (American Psychiatric Association 1980). Documenting the course of thought pathology in schizophrenics defined according to modern, narrow concepts of this disorder and studying differences in the course of thought pathology between RDC/*DSM-III* schizophrenics and other psychotic disorders would seem timely. Recent research has suggested that pervasive and severe thought disorders may be particularly characteristic of manic patients at the acute, in-hospital phase (Andreasen and Powers 1975; Andreasen 1979; Harrow et al. 1982; Resnick and Oltmanns 1984). More diverse groups of nonschizophrenic *psychotic patients* have not been focused upon, however, and differences in the longitudinal course of positive thought disorder symptoms of RDC/*DSM-III* schizophrenics and a wide range of *other psychotic patients* have not been documented.

The current article presents the 1.5-year followup results of a multidimensional, longitudinal study of the early course of positive thought disorder in schizophrenia and other types of psychoses. Using a new and larger population of schizophrenics than has been assessed in prior studies, this research examined the course of positive thought disorder in young schizophrenic patients between an acute-phase hospitalization and a followup assessment 1.5 years after hospital discharge. Employing RDC and *DSM-III* criteria in the diagnosis of schizophrenia and comparison groups, other psychotic and nonpsychotic patients were also followed up to assess the specificity of thought disorder patterns over the early course of disturbance. Thought disorder was examined within the context of other aspects of the schizophrenic course, and rehospitalized and community-dwelling patients were compared to assess the contribution of acute psychiatric conditions to the severity of disturbed thinking at followup.

The primary research questions were as follows:

- Are there differences in the presence and severity of positive thought disorder among RDC/*DSM-III* schizophrenic, other psychotic, and nonpsychotic patients at the acute phase of hospitalization and at followup; and is positive thought disorder more persistent in schizophrenia than in other psychiatric disorders?

- Does thought pathology occur in schizophrenia as part of a persistent, unremitting course of illness during the posthospital period or as part of a new episode of illness?
- What is the relationship between the *course* of positive thought disorder and life adjustment at followup in schizophrenia?

Method

Patient Setting and Population. The present sample, derived from the Chicago Followup Study, consisted of 166 patients admitted to two Chicago area facilities—Michael Reese Medical Center (MRH) and the Illinois State Psychiatric Institute (ISPI)—and then followed up 1.5 years later. The overall goals of this longitudinal research program were to (1) study schizophrenic thought disorders, psychosis, and outcome on a longitudinal basis over different phases of the schizophrenic disturbance (Harrow and Silverstein 1977; Harrow et al. 1978; Harrow, Silverstein, and Marengo 1983; Pogue-Geile and Harrow 1984) and (2) to explore mechanisms that may be involved in schizophrenic thought pathology and psychosis (Harrow and Prosen 1979; Harrow and Miller 1980; Harrow et al. 1983; Lanin-Kettering and Harrow 1985).

The Research Diagnostic Criteria (RDC) were used for the primary classification of diagnostic groups (Spitzer, Endicott, and Robins 1978). The sample included 48 RDC schizophrenics, 51 psychotic nonschizophrenic patients, and 67 nonpsychotic patients. *DSM-III* classifications of the same patient sample yielded 40 schizophrenics, 59 psychotic nonschizophrenics, and 67 nonpsychotic patients. For this research, psychotic patients were defined in terms of the presence of delusions and/or hallucinations. To avoid circularity in the current study of pathological thinking, the presence of positive thought disorder in the absence of delusions and hallucinations was not used as a criterion to label patients as psychotic at index hospitalization.

Information for RDC and *DSM-III* diagnoses were obtained from standardized interviews using a modified version of the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978) and the Schizophrenia State Inventory (SSI); a tape-recorded, semistructured research interview obtained shortly after index admission (Grinker and Holzman 1973); and from detailed information on the patient's clinical picture and previous history obtained at hospitalization. Further diagnostic information relating to schizophrenic and comparison samples is presented in table 1.

Patient Characteristics. Patients were assessed early in the course of their disorders to minimize the potential contaminating effects of long-term hospitalization and/or years of chronic illness. All patients ranged from 17 to 32 years of age at index hospitalization (mean = 22.7 years, SD = 3.7 years). Fifty-four percent of the patients were first hospital admissions, and a total of 77 percent of them had no or only one previous admission at index hospitalization.

Fifty-four percent of the sample was female and 46 percent was male. Using the 5-point Hollingshead and Redlich (1958) system to assess socioeconomic class, 59 percent of the sample fell into higher socioeconomic classes (categories 1, 2, 3) and 41 percent clustered into lower socioeconomic classes (categories 4 and 5). Seventy-five percent of the patients were white, 24 percent were black, and 1 percent was composed of other groups. The mean educational level of the sample was 12.9 years of schooling (SD = 2.1), and the mean level of intelligence, using the Wechsler Adult Intelligence Scale (WAIS) Information Test, was 11.0 (SD = 2.9), falling within the

Table 1. Diagnoses of patient population

Sample	RDC diagnosis	n
Schizophrenic	Paranoid	23
	Undifferentiated	25
Psychotic nonschizophrenic (other psychotic)	Major depressive disorder	13
	Manic disorder	9
	Schizoaffective depressed	14
	Schizoaffective manic	5
	Unspecified functional disorder	7
	Drug abuse disorder, psychotic	3
Nonpsychotic	Major depressive disorder	38
	Minor depressive disorder	9
	Manic disorder	1
	Hypomanic disorder	2
	Bipolar affective disorder, mixed type	1
	Other (other psychiatric disorders—labile personality, etc.)	16

Average Range of Intelligence (Wechsler 1955).

One-way ANOVA revealed that the schizophrenics had completed significantly fewer years of education than the psychotic and nonpsychotic patient groups ($F = 4.76$, $df = 2, 163$, $p < .01$). However, 72 percent of the schizophrenics had completed at least 12 years of education. Within the schizophrenic sample, the severity of disturbed thinking showed no significant relationship to years of education. In addition, in the subsequent analyses of the results, analysis of covariance was conducted to control for any possible effects of education on thought disorder.

Eighty-three percent of the schizophrenic, 67 percent of the psychotic nonschizophrenic, and 23 percent of the nonpsychotic patients were receiving neuroleptic medications at index assessment. At posthospital assessment, 52 percent of the schizophrenic, 36 percent of the psychotic, and 13 percent of the nonpsychotic patients were receiving neuroleptic treatment. Data on the relationship between medication status and thought disorder are presented in the "Results" section.

Assessment of Thought Disorder. The term *positive thought disorder* has been applied to various types of disordered cognition and has been used to describe disordered thinking that is loose, bizarre, incoherent, idiosyncratic, illogical, and/or peculiar (Chapman and Chapman 1973). Current use of the terms *thought disorder* and *disordered thinking* is based on a comprehensive measure of positive types of thought disorder (see Fish 1962, for a discussion of positive vs. negative types of thought disorder). This comprehensive measure of bizarre-idiosyncratic thinking or positive thought disorder, which has been used successfully in previous research, is based on an overall score derived from three cognitive tests (Harrow et al. 1982; Harrow, Silverstein, and Marengo 1983).

These three tests, administered at index and followup assessments, are the Goldstein-Scheerer Object Sorting Test (Goldstein 1944), the Gorham Proverbs Test (Gorham 1956), and the Social Comprehension Test of the WAIS (Wechsler 1955). Each of these tests provides a separate measure of bizarre-idiosyncratic thinking. Patients' responses to each of the tests were scored "blind" by raters who were not informed of patient diagnosis or whether they were rating inpatient or followup records; and they were scored without knowledge of the specific hypotheses being investigated.

The individual proverb and comprehension test responses were scored for bizarre-idiosyncratic thinking along 4-point scales ranging from no bizarre thinking to severe bizarre or idiosyncratic thought.

An example of a response with *clear* bizarre-idiosyncratic features follows:

> (Q) Rome was not built in a day.
> (A) It's love. I think of it as love. I have to work towards love and love has to work towards me. And this has to gradually come.

A response example with very *severe* bizarre-idiosyncratic features follows:

> (Q) The wife is the key to the house.
> (A) Faults are true, equal, the same. The wife is equal to the husband only in name. But if names are not the same, it cannot be true.

This index of bizarre-idiosyncratic thinking encompasses many of the classic qualities of thought disorder considered important by diverse theorists. It also encompasses almost all the positive types of thought disorder described in the RDC and *DSM-III* diagnostic manuals as symptoms that can be used as partial criteria for a diagnosis of schizophrenia. These types of thought disorder include incoherence, marked loosening of association or derailment, marked illogical thinking, and neologisms. Other examples of bizarre responses have been presented in previous reports (Harrow et al. 1982; Harrow et al. 1983) and in a detailed manual (Marengo et al. 1985). Recent interrater reliabilities of $r = .84$ to $r = .93$ were obtained for the scoring system.

The individual Object Sorting Test responses were scored for positive thought disorder along a 3-point scale, according to a continuum from none to severe, bizarre, or idiosyncratic features. The final score represents the sum of a patient's individual bizarre responses. The specific criteria for the current system of assessing object sorting items have been outlined in an earlier scoring manual (Himmelhoch et al. 1973). Satisfactory interrater reliability ($r = .87$) has been obtained for the scoring system, and the current system has been used in recent reports (Harrow and Silverstein 1980; Harrow, Silverstein, and Marengo 1983).

The total scores from each of these three tests were categorized into five major levels of bizarre-idiosyncratic thinking, varying from no thought disorder to severe thought disorder, as follows: (1) no bizarre-idiosyncratic thinking, (2) minimal to mild levels, (3) moderate levels, (4) severe levels, and (5) extremely severe levels of bizarre-idiosyncratic thinking. Even level 3 reflects definite signs of abnormal thinking. The combined number of patients at levels 3 through 5 gives the percentage of thought-disordered patients. The grouping of patients at levels 4 and 5 provides an index of *severely* thought-disorderd patients.

The major measure of thought disorder used in the current research is the composite or overall index of bizarre-idiosyncratic thinking. This overall index is based on patients' scores from the three separate measures of thought disorder. For this overall index, each patient was assigned to one of the five categories, or levels, ranging from no bizarre-idiosyncratic thinking (level 1) to very severe disturbance (level 5) according to the highest level achieved on any of the three tests. This classification system categorizes patients according to whether they show any bizarre-idiosyncratic thinking, given three separate opportunities to do so. The overall score encompassed a broad sample of patients' behavior and is more comprehensive than indices that include only one or two tests.

Assessment of a New Episode or Course of Continuing Illness in Schizophrenia Between Index and Followup Assessments. Positive thought disorder at followup was also related to the course of schizophrenia during the period between index hospitalization and followup. The *course* of disturbance between index and followup assessment was assessed to determine if, at followup, thought disorder symptoms were associated with (1) a new, acute episode of illness; (2) a continuing, unremitted condition; (3) a course of continuing psychosis but adequate social and work functioning; or (4) a remission in the disturbance or illness.

The course of disturbance during the year between hospital discharge and followup was assessed in a structured functioning interview (Harrow et al. 1978), and by the modified SADS (Endicott and Spitzer 1978). These two followup interviews assessing functioning involved detailed evaluations of the following: (1) social, work, and family functioning; (2) intellectual functioning; (3) patients' use of medication, psychotherapy, and other posthospital treatment; (4) anxiety, depressive, and psychotic symptoms; and (5) rehospitalization.

Each patient's functioning in the year before followup was compared with their prehospital functioning and to their clinical condition at index hospitalization to determine the course of illness between hospitalization and followup assessment.

At followup, patients whose functioning was adequate (i.e., they had returned to work or school, were able to maintain household duties, or showed increasing social relations) and whose symptom picture was similar to their premorbid state were judged to be in remission from their index episode. Patients were judged to be in a new episode of illness at followup if their functioning and symptom picture indicated a remission for 6 months or more in the year following hospital discharge but reflected a deterioration in work and social functioning and a return of psychosis at the 1.5-year followup.

In general, ratings for continued illness were based on the patient's having continuous low levels of functioning and adjustment throughout the followup year and also showing continuing symptoms (usually psychotic symptoms but, in some cases, severe anxiety or depressive symptoms). The fourth category (adequate functioning but psychotic) was reserved for the rare patient who was psychotic but who managed to be self-supporting and engaged in some social activities. For the purposes of this research studying pathological thinking, assessments of the course of illness were independent of and did not include thought disorder ratings.

Satisfactory agreement between two raters was obtained when the 49 schizophrenics were classified into one of these four groups using the above criteria (*Kappa* = .87).

Assessment of Instrumental Adjustment at Followup. Thought disorder courses were compared to outcome states at 1.5-year followup in each of the patient groups. Two scales of overall adjustment were used: (1) an overall scale based on work and social functioning, life disruptions, self-support, symptoms, suicide, relapses, and rehospitalization (Levenstein, Klein, and Pollack 1966); and (2) an overall measure that included four individual scales covering work functioning, social functioning, rehospitalization, and symptoms (Strauss and Carpenter 1972). The presence of psychotic and affective symptoms was assessed using a modified version of the Schedule for Affective Disorders and Schizophrenia (Endicott and Spitzer 1978) and the Katz Adjustment Scales (Katz and Lyerly 1963).

Procedure. All patients were tested during the acute phase of disturbance within the first 2 weeks of index hospitalization. Followup assessments took place an average of 1.5 years after hospital discharge. Patient protocols were scored for positive thought disorder by research assistants who were blind to diagnosis and other outcome data. Information regarding medication use, sociodemographic characteristics, and instrumental adjustment were obtained at the time of assessment.

Results

Thought Disorder at the Acute Phase of Disturbance. The initial assessment of positive thought disorder was conducted within the first 2 weeks of psychiatric hospitalization. Previous research suggests that this represents an acute phase of disorder for most patients.

Tables 2 and 3 present data on positive thought disorder in the three major RDC diagnostic groups at index assessment and at followup. A one-way ANOVA of the data in table 3 on the severity of bizarre-idiosyncratic thinking at index admission across the RDC patient groups showed significant diagnostic differences ($F = 12.62$, $df = 2, 163$, $p < .001$). Analyses (ANOVA) controlling for the possible effects of educational achievement and first-admission status on the severity of thought disorder showed similar results ($F = 7.03$, $df = 2, 161$, $p < .001$). Post hoc comparisons (Neuman-Keuls) indicated that schizophrenics showed more severe thought pathology than psychotic nonschizophrenic and nonpsychotic patients at the index hospitalization ($p < .05$). No significant differences in bizarre-idiosyncratic thinking emerged between the psychotic nonschizophrenic and nonpsychotic patient groups, although there were trends toward more severe thought disorder in psychotic nonschizophrenic patients ($p < .10$).

Similar diagnostic differences in the severity of positive thought disorder at in-hospital assessment were demonstrated in patients classified according to *DSM-III* criteria ($F = 13.23$, $df = 2, 163$, $p < .001$). Post hoc comparisons of the *DSM-III* groups yielded significant differences in disordered thinking between schizophrenic and both groups of nonschizophrenic patients ($p < .05$) and between psychotic nonschizophrenic and nonpsychotic patients ($p < .05$). Larger differences in thought disorder severity emerged between *DSM-III* psychotic nonschizophrenic and nonpsychotic patients than between RDC psychotic nonschizophrenic and nonpsychotic patients. A number of severely thought-disordered RDC schizophrenics were categorized as having other types of psychotic disorders in *DSM-III*, increasing the mean thought disorder score of *DSM-III* psychotic nonschizophrenic patients (compared with RDC psychotic nonschizophrenics) and resulting in greater group differences between *DSM-III* psychotic nonschizophrenic and nonpsychotic patients. In the *DSM-III* system, psychotic nonschizophrenics fell midway between, and were significantly different from, schizophrenic and nonpsychotic patients in the severity of their disordered thinking.

Diagnostic differences were partly a function of the presence of severe positive thought disorder in over 50 percent of the schizophrenic patients (table 2). This portion of schizophrenics showing severely disordered thought can be contrasted with a significantly lower frequency (only 23 percent) of severe thought disorder in the combined sample of nonschizophrenic patients ($\chi^2 = 17.3$, $df = 1$, $p < .001$). Other data of ours indicate that in addition to the severe thought pathology shown in schizophrenics at the acute phase, manic patients are also severely thought disordered at acute phases of disturbance and show significantly more thought pathology than other psychotic patients (Harrow et al. 1982; Marengo and Harrow 1985).

The data indicate that thought disorder is not unique to schizophrenia and that moderate signs of cognitive disruption appear across all diagnostic groups at the in-hospital phase. While psychotic nonschizophrenic patients tended to show more positive thought disorder than nonpsychotic patients, not all psychotic patients were thought

Table 2. Distribution of patients on thought disorder at index hospitalization and at followup

	Level of thought disorder at index hospitalization			Level of thought disorder at followup		
Patient group	No thought disorder (scores: 1 or 2)	Signs of abnormal thinking (score: 3)	Severe thought disorder (scores: 4 or 5)	No thought disorder (scores: 1 or 2)	Signs of abnormal thinking (score: 3)	Severe thought disorder (scores: 4 or 5)
Schizophrenic ($n = 48$)	19%	25%	56%	56%	17%	27%
Psychotic ($n = 51$)	43%	28%	29%	61%	25%	14%
Nonpsychotic ($n = 67$)	58%	24%	18%	74%	24%	2%

Table 3. Means and standard deviations of the three patient groupings on thought disorder at index admission and followup[1]

Patient group	Thought disorder at index hospitalization		Thought disorder at followup	
	Mean	SD	Mean	SD
Schizophrenic ($n = 48$)	3.54	1.22	2.70	1.34
Other psychotic ($n = 51$)	2.92	1.13	2.35	1.11
Nonpsychotic ($n = 67$)	2.46	1.07	2.02	0.75

[1] Range: 1 (no thought disorder) to 5 (very severe thought disorder).

disordered. Severe thought disorder is not only a function of psychosis (Marengo and Harrow 1985). In addition, schizophrenic patients are distinct from other psychotic and nonpsychotic patient groups in that a larger percentage of schizophrenics exhibit *severely* disordered cognition at acute phases of their disorder.

Thought Disorder at Followup: Schizophrenic Versus Other Psychotic and Nonpsychotic Patients. Followup assessments took place 1.5 years after hospital discharge (1.8 years after the initial, acute phase, thought disorder assessment). For the majority of these young patients, the followup assessment represents a postacute period. A major issue is whether cognitive disorders, as well as other types of major symptoms, are more severe in schizophrenia than in other types of psychiatric disorder at followup.

One-way ANOVA of the data on the severity of bizarre-idiosyncratic thinking at followup (table 3) indicated significant diagnostic differences ($F = 5.96$, $df = 2, 163$, $p < .01$). Post hoc comparisons of the severity of disordered thinking at followup in RDC schizophrenic, psychotic, and nonpsychotic patient groups showed significantly more positive thought disorder in schizophrenics than in the other two patient groups ($p < .05$). Similar results were obtained using *DSM-III* criteria. Nonschizophrenic patients appeared more homogeneous and showed, on average, less frequent signs of positive thought disorder. Mean thought disorder levels suggest that disordered thinking is a more frequent and enduring problem for schizophrenic than for nonschizophrenic patients 1.5 years after hospital discharge.

As indicated in table 2, 27 percent of the schizophrenic patients showed *severe* thought pathology and an additional 17 percent exhibited evidence of abnormal thinking at followup. In contrast to the 27 percent rate of severe thought disorder at followup in the schizophrenics, less than 10 percent of the combined sample of nonschizophrenic patients were severely thought disordered at followup ($\chi^2 = 12.7$, $df = 1$, $p < .001$).

Previous research of ours, using the Object Sorting Test only, suggested that much of the difference between *DSM-II* schizophrenic and nonschizophrenic patients at followup could be attributed to a subgroup of schizophrenics who show severe positive thought disorder (Harrow and Silverstein 1980). Our current results, based on a broader index of positive thought disorder and using modern diagnostic systems, reaffirm this finding. Schizophrenic-nonschizophrenic differences in early, young patients are primarily confined to the most pathological end of the thought disorder spectrum, and more schizophrenic than nonschizophrenic patients are severely thought disordered. Overall, the results reveal persistent differences between early schizophrenic and nonschizophrenic patient groups at followup. Severe positive thought disorder is persistent, however, in only a subgroup of early schizophrenics after the acute phase.

Neuroleptic Treatment During the Acute and Posthospital Phases. A 3 × 2 ANOVA (diagnostic group by medication status) on followup thought disorder yielded no significant differences between medicated and unmedicated patients. The interaction between diagnosis and medication status, however, was significant ($F = 3.00$, $df = 2, 160$, $p < .05$). Medicated schizophrenic and nonpsychotic patients were slightly more thought disordered than their unmedicated counterparts. In contrast, unmedicated, psychotic nonschizophrenic patients were more thought disordered than the medicated psychotic nonschizophrenic group.

Caution should be exercised in interpreting these results, since patients were not administered neuroleptic medication on a random basis. Rather, the administration of neuroleptics was based, in part, on the severity of symptoms. Those patients experiencing psychotic symptoms were more likely to receive neuroleptic treatment than patients who were not psychotic or not symptomatic. A study that randomly assigns patients to treatment conditions and monitors posthospital treatment carefully is needed to assess adequately the effects of neuroleptic medications on thought disorder during the followup period.

Does Thought Pathology Occur in Schizophrenia at Followup as Part of a Persistent, Unremitting Course of Illness During the Posthospital Period or as Part of a New Episode of Illness? One factor that may be involved in the observed schizophrenic-nonschizophrenic differences in positive thought disorder at followup is that the schizophrenic sample may include a substantial proportion of patients who are still or again in an acute phase of illness. This possibility would suggest that many of the schizophrenic patients who are experiencing thought pathology at followup are in an active, acute phase of disorder and are experiencing thought disorder concomitant with acute psychosis or rehospitalization. The differences in the prevalence of bizarre-idiosyncratic thinking between schizophrenic and nonschizophrenic groups, according to this view, would be due to a greater relapse rate, and perhaps rehospitalization, in the schizophrenic group. This possibility was examined, and our analyses are now presented.

Positive thought disorder in relation to a new episode of illness in schizophrenia or to a continuing course of disturbance. We assessed the subsample of schizophrenics with *severe* thought pathology at followup using data on other aspects of their functioning during the year preceding followup to examine whether these patients showed severely disturbed thinking as part of a continuing course of disturbance during the posthospital period, as part of a new episode of illness, or as an isolated symptom in an otherwise healthy patient. The posthospital course of disturbance in these patients was also compared to the posthospital course of schizophrenics who exhibited no or only moderate signs of abnormal thinking at followup assessment. The data that emerged from this analysis are presented in table 4.

For the schizophrenic sample, severe positive thought disorder did not occur in isolation from other aspects of disturbance. Severely disordered thinking occurred as part of either a larger picture of continuing illness or a new acute episode of disturbance. The most prominent finding in this area was the co-occurrence of severe thought disorder with residual signs of schizophrenia and poor functioning in other areas. Evidence of psychosis (i.e., delusions and/or hallucinations) accompanied severe thought disorder signs in all but one of the severely thought-disordered schizophrenics at followup assessment. Severe signs of positive thought disorder and abnormal thinking most often occurred within the context of a chronic, unremitting illness during the followup period.

More of the nonthought-disordered schizophrenics were in remission in other areas of functioning than was the case for the severely thought-disorderd schizophrenics ($\chi^2 = 4.67$, $df = 1$, $p < .05$). Despite a small subgroup of schizophrenics who were in remission in all areas, however, the majority of both thought-disordered and nonthought-disordered schizophrenics showed a course of continuing dysfunction during the year following hospital discharge (see table 4). This was especially prominent for the schizophrenics who were severely thought disordered at followup. None of these 13 severely thought-disordered schizophrenics were in complete remission in other areas during the followup period, in contrast to a 29 percent remission rate among the 27 schizophrenics with minimal or no thought disorder at followup.

While the thought disorder of most of these young schizophrenics fit into a more general picture of chronic, unremitting illness during the followup period, there also was a much smaller group of schizophrenics whose thought disorder seemed to be part of a new episode of disturbance,

Table 4. Thought disorder in schizophrenia at followup as a function of continual illness or a new episode of illness

	Status of schizophrenic illness at followup			
Level of thought disorder at followup	In remission	Adequate functioning but psychotic	In new episode	Continual illness
No thought disorder ($n = 27$)	29%	4%	0%	67%
Signs of abnormal thinking ($n = 8$)	13%	12%	0%	75%
Severe thought disorder ($n = 13$)	0%	8%	15%	77%
Total sample ($n = 48$)	19%	6%	4%	71%

in which posthospital remission was followed by a relapse of psychosis, job loss, and increased disturbance in social relationships during the period immediately preceding followup. Two of the 13 severely thought-disordered schizophrenics exhibited such a course.

One possible view of the early young schizophrenic during the posthospital period is of a patient who might be seen as a strange, eccentric, thought-disordered person but who is adequately adjusted in other areas. This type of picture was not found in any of the current relatively young sample of thought-disordered schizophrenics. Only 1 of the 13 schizophrenics with severe thought disorder showed good work and social functioning during the followup period, and even this thought-disordered patient showed evidence of psychosis accompanying her thought disorder symptoms. The remaining 12 schizophrenics showed thought disorder as part of a chronic illness during the 1.5-year followup period or as part of a new acute illness.

Overall, the data in this area indicate that for those schizophrenics who are thought disordered at followup, their thought disorder is not an isolated symptom in an otherwise healthy patient; nor did thought disorder occur as part of a new episode of illness for the majority of patients. Rather, most of these thought-disordered schizophrenics showed a more chronic course of illness during the 1.5-year followup period.

Positive thought disorder in relation to rehospitalization at followup. To determine whether the severity of disturbed thinking was related to rehospitalization at followup, the sample was divided into three groups: (1) patients who were back in the hospital for a psychiatric disorder at followup; (2) patients who had been hospitalized during the year between index and followup but who were not in the hospital at followup; and (3) patients who had not been hospitalized in the past year. At followup assessment, 8 schizophrenic, 7 psychotic nonschizophrenic, and 4 nonpsychotic patients were in the hospital for psychiatric disorders. Twenty-two schizophrenics, 13 other psychotic patients, and 19 nonpsychotic patients had been rehospitalized since index admission but were not in the hospital at followup assessment.

A 3 × 3 ANOVA (diagnostic group × hospital status) of followup thought disorder revealed no significant main effect for hospital status and no significant interaction. Schizophrenic patients who were currently hospitalized, however, showed a tendency to be more thought disordered than currently hospitalized psychotic nonschizophrenic and nonpsychotic groups ($F = 4.43$, $df = 2, 18$, $p < .05$). Currently hospitalized schizophrenics tended to have more severe positive thought disorder than schizophrenics not hospitalized at followup ($t = 2.14$, $df = 45$, $p < .05$).

Is There a Reduction in Positive Thought Disorder at Followup? The current research assessed positive thought disorder at two points over the early course of schizophrenic and nonschizophrenic disorders. The prospective nature of this investigation afforded an opportunity to assess changes in positive thought disorder over time, as patients moved from acute to postacute phases of disturbance.

A two-way repeated measures ANOVA (diagnosis × phase of disorder) of the data in table 3 on the severity of positive thought disorder at acute and followup phases showed significant remission in thought disorder for the total sample ($F = 27.82$, $df = 1, 163$, $p < .01$), with no significant interactions.

Positive thought disorder, on average, decreased from the acute phase to followup in each diagnostic group, although some select patients showed more severe levels of thought disorder at followup. This remission in positive thought disorder between index and followup assessments was significant in schizophrenic ($t = 3.60$, $df = 47$, $p < .001$), psychotic nonschizophrenic ($t = 2.48$, $df = 50$, $p < .02$), and nonpsychotic patients ($t = 2.70$, $df = 66$, $p = < .009$).

Some earlier views had suggested that there is severe thought disorder in acute schizophrenia and that further deterioration in schizophrenic thinking occurs over time. The current data indicate that most early young schizophrenics show evidence of disordered thinking at the acute phase but do not show further deterioration in positive thought disorder during the early posthospital period; they even tend to show some reduction after the acute phase.

Does the Presence of Thought Disorder at the Acute Phase Predict Subsequent Thought Disorder at Followup? The acute phase of the disorder is in some ways the least stable phase of disturbance. Thus, the question has been raised of whether schizophrenics (as well as patients from other diagnostic groups) who initially show signs of thought disorder at the acute phase are more likely to show thought disorder at followup compared with patients who do not show initial evidence of disordered thinking.

Table 5 presents data bearing on whether positive thought disorder at the acute phase is predictive of

Table 5. Relationship between positive thought disorder at hospitalization and at followup

			Percent of patients with thought disorder at followup		
Patient group	**Thought disorder at index hospitalization**	*n*	**No thought disorder**	**Signs of abnormal thinking**	**Severe thought disorder**
Schizophrenic	Absent	9	89%	0%	11%
	Present	39	49%	20%	31%
Other psychotic	Absent	22	59%	27%	14%
	Present	29	62%	24%	14%
Nonpsychotic	Absent	38	84%	16%	0%
	Present	29	59%	38%	3%

thought disorder at followup for each diagnostic group. Schizophrenics who were thought disordered at the acute phase showed significantly more thought disorder at followup than did schizophrenics who did not have acute phase thought disorder ($t = 2.44$, $df = 46$, $p < .02$). Twelve of the 39 schizophrenics who showed evidence of thought disorder at the acute phase showed severe thought disorder at followup, and an additional 8 of these 39 showed signs of abnormal thinking at followup.

All patient groups showed overall reductions in thought disorder at followup. From among the patients who showed signs of thought disorder at the acute phase, however, a significantly larger percentage of schizophrenics showed severe thought disorder at followup than was the case for nonschizophrenic patient groups ($\chi^2 = 7.91$, $df = 1$, $p < .01$). Overall, the presence of thought disorder for schizophrenics at the acute phase was predictive of thought disorder at followup, although it was not a powerful predictor.

The data derived from two phases of the disorder in psychotic nonschizophrenic patients showed surprising inconsistency in the course of positive thought disorder. This group showed the greatest variation in thought pathology from the acute phase to followup. There were no significant or near-significant differences in thought disorder at followup between psychotic patients who were initially thought disordered and those who were not ($p > .40$). Almost two-thirds of the other psychotic patients who were thought disordered at the acute phase showed remissions in cognitive symptoms at followup. On the other hand, 41 percent of the other psychotic patients who were not thought disordered initially showed thought disorder at followup. Nine of the 22 psychotic patients who were *not* thought disordered at the acute phase showed at least some signs of positive thought disorder at followup. Included among these nine other psychotic patients were four psychotic depressed patients, two schizoaffective patients, one patient with unspecified functional psychotic disorder, and two drug abuse patients.

No specific diagnostic constellation characterized these psychotic patients who showed increases in positive thought disorder over time. Overall, the other psychotic patient group showed the greatest heterogeneity in terms of their thought pathology, with the suggestion that they show a different clinical course than that of schizophrenics. Some or many of these other psychotic patients may be vulnerable to future thought disorder and/or other psychotic symptoms.

In general, the presence of acute phase thought disorder did suggest a greater potential for thought disorder at followup for the schizophrenics, but this relationship did not hold among the other psychotic patients. These data suggest that despite the potential instability of acute phase thought disorder, it does tend to predict subsequent thought disorder at followup for schizophrenics. The relationship, while significant, is only a modest one.

The Early Course of Thought Pathology: Subgroups of Patients With Reductions and With Persistent Thought Disorder and Their Life Adjustment. An alternate way of looking at the data at the two early phases of disorder is by focusing on subgroups of patients defined according to their course of thought pathology. One of the goals is to determine whether the course of thought disorder, especially for schizophrenia, differentiates patients in terms of other aspects of their adjustment at followup. Table 6 presents data on the frequency of

Table 6. Frequency of four courses of thought disorder between index hospitalization and followup

	Not thought disordered at index hospitalization		Thought disordered[1] at index hospitalization	
Patient group	**And not thought disordered at followup**	**But thought disordered at followup**	**But not thought disordered at followup**	**And thought disordered at followup**
Schizophrenic (n = 48)	17%	2%	39%	42%
Other psychotic (n = 51)	25%	18%	35%	22%
Nonpsychotic (n = 67)	48%	9%	25%	18%

[1] Patients were rated as thought disordered if clear signs were present: composite index scores = 3 to 5.

each type of thought disorder course for schizophrenics and for the other groups of psychotic and nonpsychotic patients.

A larger percentage of schizophrenics than nonschizophrenics showed signs of thought disorder at both the acute phase and the followup assessment ($\chi^2 = 8.74$, $df = 1$, $p < .01$). More than 40 percent of the schizophrenics showed at least some signs of positive thought disorder during both these early phases of disorder, as contrasted with about 20 percent of the other patients. While a larger percentage of schizophrenics who were thought disordered at the acute phase again showed signs of thought disorder at followup, almost none of the small subgroup of schizophrenics who did not show evidence of thought disorder at the acute phase showed evidence of thought disorder at followup (see table 6). A course of illness characterized by *no* signs of positive thought disorder at either the acute phase or followup was not common in schizophrenia and was found in only 17 percent of the total schizophrenic sample.

As expected, nonpsychotic patients were the least likely to show cognitive disorder over time. About half of the nonpsychotic patients showed no evidence of abnormal thinking at either acute phase or followup assessment.

As a group, the other psychotic patient group showed the most variation in the course of positive thought disorder over time. Thus, a larger percentage of the other psychotic patients showed episodic patterns, in terms of either a reduction or an increase in thought disorder, as they moved into the posthospital phase. Fewer of them showed the same level of thought disorder at both phases of disorder.

As we have already noted, one of the goals of assessing thought disorder at two points in time, the acute phase and followup, is to determine whether particular thought disorder courses are associated with other aspects of functioning or psychosis at followup. The current research is among the first to explore subtypes of psychopathology based on longitudinal assessment of thought disorder. Of particular interest were comparisons of life adjustment among patients demonstrating (1) no thought disorder at either hospitalization or the first followup, (2) episodic patterns of cognitive disturbance between index and followup assessment (patients showing thought disorder *either* at hospitalization or followup but not at both), and (3) evidence of thought disorder at both index and followup assessment.

The longitudinal assessment of cognitive symptoms for the *overall sample* was strongly associated with outcome. Three × 3 ANOVAs (diagnosis × course of thought disorder) of life adjustment at followup indicated a significant main effect for diagnosis. Most important, the ANOVAs also showed significant main effects for the course of cognitive disturbance on overall outcome ($F = 4.21$, $df = 2, 158$, $p < .02$); work functioning ($F = 6.57$, $df = 2, 158$, $p < .002$); delusions ($F = 11.28$, $df = 2, 158$, $p < .001$); and hallucinations ($F = 6.37$, $df = 2, 158$, $p < .002$). The course of thought disorder between the acute phase and 1.5 year after hospital discharge was not significantly related to social functioning at followup.

Table 7 presents data on adjustment and psychosis for the schizophrenic subgroups defined according to the course of their thought disorder. Within the schizophrenic sample, nonthought-disordered patients exhibited a significantly better overall adjustment than did either continuously thought-disordered schizophrenics or those showing episodic thought disorder patterns ($F = 4.09$, $df = 2, 46$, $p < .03$). Schizophrenics with episodic patterns of cognitive disturbance often showed outcomes that fell midway between the relatively better adjustment of schizophrenics who were not thought disordered and the poorer adjustment of the continuously thought-disordered group.

The outcome distributions of the schizophrenic patients who showed signs of thought disorder at both the acute phase and at followup indicate a higher frequency of dysfunction in a variety of adjustment areas compared with those of other schizophrenics. Schizophrenics with thought disorder at both phases of disorder showed more severe impairment in terms of a 70 percent rate of rehospitalization and a 79 percent rate of unemployment. In addition, 53 percent showed hallucinations and 63 percent showed delusions at followup.

Similarly, continuously thought-disordered, psychotic nonschizophrenic and nonpsychotic patients showed trends ($p < .15$) toward more impaired adjustment and continuing psychosis than the nonthought-disordered patients and patients who showed episodic thought disorder.

Discussion

Positive Thought Disorder and Schizophrenic Vulnerability. The current research examined the frequency and severity of positive thought disorder in schizophrenia between the acute and posthospital phases over the early course of patients' disorders. The course of positive thought pathology in schizophrenia was compared with that of other psychotic and nonpsychotic disorders to assess potential differences between these patient groups. Some older beliefs center on the view of positive thought disorder as a permanent, traitlike feature in most schizophrenics. More recent views have proposed that positive thought disorder and positive symptoms remit after the acute phase. In particular, Strauss, Carpenter, and Bartko (1974) and Crow (1982; Crow et al. 1982) have proposed that positive thought disorder is likely to be episodic or to resolve after the acute phase of schizophrenic disturbance.

The current data on the course of positive thought disorder after the acute phase bear directly on views about schizophrenic vulnerability. Zubin and colleagues and, more recently, Neuchterlein and Dawson and Strauss and colleagues have succeeded in alerting the field to, and drawing attention to, a major issue involving the nature of schizophrenic vulnerability and whether schizophrenics are vulnerable to discrete episodes or a continuous disorder (Zubin and Spring 1977; Zubin, Magaziner, and Steinhauer 1983; Neuchterlein and Dawson 1984; Strauss et al. 1985). Zubin has

Table 7. Outcome functioning at followup in schizophrenics categorized by the course of thought disorder

Course of thought disorder	Overall adjustment[1] in the past year		Employment[2]		Social adjustment[2]		Percent with psychosis at followup
	Mean	SD	Mean	SD	Mean	SD	
Schizophrenics with no thought disorder at either assessment ($n = 8$)	4.25	3.15	2.50	1.51	2.75	1.58	13%
Schizophrenics with thought disorder at one assessment only ($n = 20$)	5.85	1.63	1.45	1.61	3.30	1.08	35%
Schizophrenics with thought disorder at both assessments ($n = 20$)	6.53	1.43	.84	1.17	3.30	1.34	68%

[1] Lower score = better outcome (scale = 1 to 8) on scales used by Levenstein, Klein, and Pollack (1966).

[2] Higher score = better outcome (scale = 0 to 4) on scales used by Strauss and Carpenter (1972).

proposed that what is continuous in schizophrenia is the vulnerability to episodes, rather than the episodes themselves being permanent or sustained. Recent data of ours on psychotic symptoms (Harrow, Carone, and Westermeyer 1985) and other data of ours on the potential persistence of thought disorder in schizophrenia (Marengo and Harrow 1984) have led us to propose a modified view in which schizophrenics show a vulnerability to a disorder or illness that is a more severe or sustained one with a long period after the more acute phase of persistent, but less intense and less disruptive, positive symptoms. To help analyze this issue on schizophrenic vulnerability and to shed further light on the context in which we found thought disorder after the acute phase of schizophrenia, we also analyzed the presence of thought disorder during the followup period to determine whether it occurred as part of a new episode of illness or as part of a persistent, unremitting course of illness.

Overall, the current data present a mixed picture on views of schizophrenic vulnerability. On one hand the data indicate that most thought-disordered schizophrenics showed at least some remission of thought pathology as they moved from the acute phase to the followup period, although a number of schizophrenics still showed evidence of positive thought disorder but at a reduced level. This reduction confirms more recent evidence of ours indicating that *severe* positive thought disorder is not a permanent feature for all schizophrenics.

The evidence suggests, however, that during early phases of disorder the majority of schizophrenics fit a model involving vulnerability to a more sustained disorder. Thus, of most importance, the data in table 4 begin to suggest that the severe positive thought disorder that was found among a subgroup of schizophrenics at followup was associated with a continual illness involving poor posthospital functioning during the followup period, rather than part of a new episode, for all but a few of these thought-disordered patients. Even among the other schizophrenics who did not show severe positive thought disorder at followup, about 70 percent of these schizophrenics also showed evidence of continuous illness or continuous malfunctioning during this early followup period; a portion of them also showed evidence of some degree of psychotic activity at followup (Harrow, Carone, and Westermeyer 1985).

One can begin to build a composite picture of postacute adjustment, based on various aspects of the data presented in this report on thought disorder at followup, and the presence of a continuous disorder during the followup period for many schizophrenics, as well as other data of ours on psychosis, cognition, and outcome (Harrow et al. 1978; Marengo and Harrow 1979, 1984; Harrow, Carone, and Westermeyer 1985). They suggest an overall picture in which early young schizophrenics are vulnerable to a severe, acute psychotic disorder leading to hospitalization, with aspects of the disorder continuing at a reduced intensity after the most acute phase and into the posthospital period for a number of years. The aspects of the disorder that persist into the posthospital period include some positive symptoms (but at a reduced level) for many schizophrenics, limited deficits in cognitive and motor performance, and an overall reduced level of work functioning for most schizophrenics. At this first followup, only a minority of schizophrenics showed complete remission in every area. In addition, the schizophrenic continues to be vulnerable to future severe, acute psychotic breaks.

Does Severe Thought Disorder Persist in Schizophrenia? When we turn from the broad question of schizophrenic vulnerability and focus on specific issues concerning the persistence of severe thought disorder, we can note that the current data did provide some degree of support to the hypotheses that early young schizophrenics show a reduction in thought disorder after the acute phase. The evidence also suggests, however, that even *severe* positive thought disorder is a relatively persistent characteristic for a subgroup of early schizophrenics, since the data showed a subgroup of early schizophrenics who were initially thought disordered at the acute phase also evidencing severe positive thought disorder at followup.

Our previous research on a smaller group of patients, with a measure derived from one test only, had indicated that thought disorder exists at followup in a subgroup of *DSM-II* schizophrenics. The present results indicate that in this particular area of disturbed cognition, *DSM-III* schizophrenics show some similarity, with 20 to 35 percent of these patients displaying severe thought disorder at followup.

In the current sample of early young patients, positive thought disorder was significantly more frequent at followup among the schizophrenics than among nonschizophrenics. The significant schizophrenic–nonschizophrenic differences in extent of positive thought disorder found in the early posthospital period was in good part a function of a subgroup of schizophrenics with *severe* positive thought disorder at followup, rather than for all schizophrenics. This subgroup may be an important cohort of schizophrenics. The data indicated that this subgroup of schizophrenics with severe positive thought disorder showed poorer posthospital functioning and more psychosis at followup than other subgroups of schizophrenic and nonschizophrenic patients.

Is Positive Thought Disorder in Schizophrenia Related to the Course of the Patient's Illness and to a Poor Prognosis? As we have noted, the data also shed some light on the context in which thought disorder at followup occurs, in terms of thought-disordered schizophrenics' early clinical course and functioning in other areas. The results indicate that for the subgroup of young schizophrenics who were severely thought disordered at followup, their positive thought disorder was not a case of strange and eccentric thinking in individuals who could function adequately in other areas. Rather, the positive thought disorder that occurred in this subgroup was present along with an overall picture of persistent, and perhaps chronic, dysfunction during the entire followup year.

In most instances it would be difficult for a severely thought-disordered person to function well at work and in other areas, with the thought disorder interfering with functioning and possibly leading to poorer overall adjustment. The positive association between thought disorder and both psychosis and instrumental adjustment, however, suggests that the very poor functioning of almost all the thought-disordered schizophrenics was part of a larger clinical picture. We would propose that underlying features associated with schizophrenia contribute to both severe thought disorder and to psychosis and poorer adjustment in other areas during the course of the followup period.

Recently, the issue of positive versus negative symptoms in schizophrenia has been of theoretical interest. It has been proposed by some that negative symptoms in schizophrenia are associated with poor outcome, but that positive symptoms represent more benign types of symptoms and may not be as closely associated to poor outcome. The current data, and other results we have reported (Harrow, Silverstein, and Marengo 1983; Pogue-Geile and Harrow 1984, in press), could suggest that when positive thought disorder is found in the posthospital phases of schizophrenia, it may also indicate a poor prognostic picture. Thus, the subgroup of schizophrenics with severe positive thought disorder could be a subgroup of poor-outcome nuclear schizophrenics, and we are currently investigating this possibility.

Schizophrenics Versus Other Psychotic Patients: The Presence and Severity of Positive Thought Disorder. The results showing more severe positive thought disorder in schizophrenics than in other psychotic patients at the acute phase and at followup confirm other recent results of ours. These data suggest that thought disorder is not unique to schizophrenics but is more *severe* in them than in other types of psychotic patients except for manic patients, and that it may also be more *persistent* than in other types of psychotic patients.

Thought disorder is not just a function of psychosis, since in the current research and in other recent research of ours, schizophrenics showed more thought disorder than other types of psychotic patients (Marengo and Harrow 1985). Psychosis and factors associated with psychosis may have an influence on thought disorder. It is not the only one, however, since the other psychotic patients did not show the severity or persistence of thought disorder found among the schizophrenics. Although many of the other psychotic patients showed signs of thought disorder at either the acute phase or followup assessment, a significantly smaller percentage of them than of the schizophrenics showed signs of positive thought disorder at both assessments ($\chi^2 = 4.64$, $df = 1$, $p < .05$).

The course of thought disorder among the group of other psychotic patients was less uniform than among the schizophrenics. In the present research some of the other psychotic patients who were not initially thought disordered at the acute phase showed subsequent thought disorder at followup; other psychotic patients who were not initially thought disordered remained free of thought disorder at followup. A larger percentage of the initially nonthought-disordered other psychotic patients than of the initially nonthought-disordered schizophrenics showed an increase in thought disorder at followup.

These data could suggest that once a patient is psychotic, he or she shows the potential for subsequent thought disorder and for other types of positive symptoms, such as delusions and hallucinations. It is possible that the other psychotic patients have the potential for positive thought disorder, and their psychosis at the acute phase is one indication that they have the potential for thought disorder later. Other data of ours also suggest that the other psychotic patients may have a greater potential for posthospital delusions than do patients who are not psychotic at the acute phase (Kettering et al. 1982). We are presently engaged in further followups of these psychotic patients over a longer period of time to provide additional evidence on this issue.

In contrast, the course of positive thought disorder was more consistent for the schizophrenics. Thought disorder was more likely to be found in schizophrenics who also exhibited cognitive pathology at index hospitalization, and it showed a relationship to other aspects of functioning and to the course of illness during the followup period. In the current sample, about half the hospitalized schizophrenics who showed moderate or severe thought disorder at the acute phase of illness also showed some type of residual cognitive impairment in the early posthospital period, although their posthospital cognitive impairment was at times less severe than at the acute phase.

Factors That May Influence or Play a Role in Positive Thought Disorder. In previous research we have proposed a model in which a number of factors, rather than only one factor, are influential in severe positive thought disorder (Harrow and Quinlan 1985). We have proposed and produced evidence about two such factors. One is an *intermingling* of personal material from one's own past or recent experiences into one's verbalizations and thinking at inappropriate times (Harrow and Prosen 1979; Harrow et al. 1983). The other is *impaired perspective* about the social appropriateness of one's thinking and behavior, such that thought-disordered patients have difficulty in effectively using long-term knowledge about which behaviors are appropriate and resourceful for a given social situation (Harrow and Miller 1980; Harrow and Quinlan 1985). At present it is still not clear whether these factors are more important in the thought pathology found in schizophrenics than in that found in nonschizophrenics who are thought disordered.

In addition, in previous research we have suggested that a third factor associated with the *acute phase* of both schizophrenic and nonschizophrenic pathology can be a contributor to positive thought disorder (Harrow and Quinlan 1977, 1985), and the current data bear on this hypothesis. This third factor, involving the acute phase of disturbance for schizophrenics and for other types of patients, appears to be based on the anxiety, turmoil, acute upset, and disturbance present at the acute phase, which can lead to disorganization, cognitive arousal, and cognitive pathology.

The current results, indicating severe thought pathology for the schizophrenics at the acute phase and a significant decline in positive thought disorder at followup, would fit in with this formulation. In addition to the significant decline in positive thought disorder for the schizophrenics, the significant decline in positive thought disorder at followup for each of the other two groups of nonschizophrenic patients studied could be viewed as further evidence about these disruptive factors at the acute phase. Thus, for all types of patients, including both schizophrenics and nonschizophrenics, the acute upset and turmoil had diminished by followup; and at the followup assessment all patient groups showed significantly less thought disorder.

Considered from the preceding standpoint, anxiety, acute upset, turmoil, and severe general disturbance—all of which can increase patients' level of disorganization—could be viewed as factors that the three patient groups experienced at the acute phase. This constellation of factors could be viewed as contributing to the thought disorder found at the acute phase for at least some patients from each diagnostic group in the current research. This constellation would not, however, be seen as the only factor contributing to thought disorder for any of the three groups. For schizophrenics it appears unlikely to be the main factor associated with their thought disorder, since a number of patients in the current research also showed some degree of thought disorder at followup, which is a less acute phase. In addition, other evidence of ours has suggested that even during the acute phase, when most psychotic patients are experiencing anxiety, acute upset, turmoil, and severe general disturbance, schizophrenics and manic patients tend to show more positive thought disorder than other psychotic patients (Marengo and Harrow 1985). Nevertheless, the current data, with the significant reduction from the acute phase to followup, suggest that factors associated with the acute phase for both schizophrenic and nonschizophrenic patients are additional and important contributors to thought disorder.

References

Adler, D., and Harrow, M. Idiosyncratic thinking and personally over-involved thinking in schizophrenic patients during partial recovery. *Comprehensive Psychiatry,* 15:57–67, 1974.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed.

Washington, DC: The Association, 1980.

Andreasen, N.C. Thought, language and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1330, 1979.

Andreasen, N.C., and Powers, P. Creativity and psychosis: An examination of conceptual style. *Archives of General Psychiatry*, 32:70–73, 1975.

Arieti, S. *Interpretation of Schizophrenia*. New York: Basic Books, 1974.

Bleuler, M. *The Schizophrenic Disorders: Long-term Patient and Family Studies*. New Haven: Yale University Press, 1978.

Buss, A.H., and Lang, P.J. Psychological deficit in schizophrenia: I. Affect, reinforcement, and concept attainment. *Journal of Abnormal Psychology*, 70:2–24, 1965.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia*. New York: Appleton Century Crofts, 1973.

Crow, T.J. The biology of schizophrenia. *Experientia*, 38:1275–1282, 1982.

Crow, T.J.; Cross, A.J.; Johnstone, E.C.; and Owen, F. Two syndromes in schizophrenia and their pathogenesis. In: Henn, F.A., and Nasrallah, H.A., eds. *Schizophrenia as a Brain Disease*. New York: Oxford University Press, 1982. pp. 196–234.

Endicott, J., and Spitzer, R.L. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35:837–844, 1978.

Fish, F.J. *Schizophrenia*. Bristol, England: John Wright and Sons Ltd., 1962.

Goldstein, K. Methodological approach to the study of schizophrenic thought disorder. In: Kasanin, J.S., ed. *Language and Thought in Schizophrenia*. New York: W.W. Norton and Company, 1944. pp. 17–39.

Gorham, D.R. Proverbs test for clinical and experimental use. *Psychological Reports*, 1 (Suppl):1–12, 1956.

Grinker, R.R., Sr., and Holzman, P.S. Schizophrenic pathology of young adults: A clinical study. *Archives of General Psychiatry*, 28:168–175, 1973.

Harrow, M.; Carone, B.J.; and Westermeyer, J.F. The course of psychosis in early phases of schizophrenia. *American Journal of Psychiatry*, 142:702–707, 1985.

Harrow, M.; Grinker, R.R., Sr.; Silverstein, M.; and Holzman, P.S. Is modern-day schizophrenic outcome still negative? *American Journal of Psychiatry*, 135:1156–1162, 1978.

Harrow, M.; Grossman, L.; Silverstein, M.; and Meltzer, H.Y. Thought pathology in manic and schizophrenic patients: Its occurrence at hospital admission and seven weeks later. *Archives of General Psychiatry*, 39:665–671, 1982.

Harrow, M.; Harkavy, K.; Bromet, E.; and Tucker, G.J. A longitudinal study of schizophrenic thinking. *Archives of General Psychiatry*, 28:179–182, 1973.

Harrow, M.; Lanin-Kettering, I.; Prosen, M.; and Miller, J.G. Disordered thinking in schizophrenia: Intermingling and loss of set. *Schizophrenia Bulletin*, 9:354–367, 1983.

Harrow, M., and Miller, J.G. Schizophrenic thought disorders and impaired perspective. *Journal of Abnormal Psychology*, 89:717–727, 1980.

Harrow, M., and Prosen, M. Schizophrenic thought disorders: Bizarre associations and intermingling. *American Journal of Psychiatry*, 136:293–296, 1979.

Harrow, M., and Quinlan, D. Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry*, 34:15–21, 1977.

Harrow, M., and Quinlan, D. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, 1985.

Harrow, M., and Silverstein, M. Psychotic symptoms in schizophrenia after the acute phase. *Schizophrenia Bulletin*, 3:608–619, 1977.

Harrow, M., and Silverstein, M.L. Cognitive processes during the postacute phase of schizophrenia. In: Sells, S.B.; Crandall, R.; and Strauss, J.S., eds. *Human Functioning in Longitudinal Perspective*. Baltimore: Williams and Wilkins, 1980. pp. 9–18.

Harrow, M.; Silverstein, M.; and Marengo, J. Disordered thinking: Does it identify nuclear schizophrenia? *Archives of General Psychiatry*, 40:765–771, 1983.

Himmelhoch, J.; Harrow, M.; Tucker, F.J.; and Hersh, J. *Manual for Assessment of Selected Aspects of Thinking: Object Sorting Test* (ASIS/NAPS #02206, 1–25). New York: Microfiche Publications, 1973.

Hollingshead, A.B., and Redlich, F.C. *Social Class and Mental Illness*. New York: John Wiley & Sons, 1958.

Holzman, P. Cognitive impairment and cognitive stability: Towards a theory of thought disorder. In: Serban, G., ed. *Cognitive Defects in the Development of Mental Illness*. New York: Brunner/Mazel, 1978. pp. 361–376.

Johnston, M.H., and Holzman, P. *Assessing Schizophrenic Thinking*. San Francisco: Jossey-Bass, 1979.

Katz, M.M., and Lyerly, S.B. Methods for measuring adjustment and social behavior in the community: I. Rationale, description, discriminative validity, and scale development. *Psychological Reports Monograph*, 13:503–535, 1963.

Kettering, R.; Harrow, M.; Grossman, L.; Meltzer, H.; and Rice, E. "Psychosis: Is It Relevant in Depression?" Presented at the 135th Annual Meeting of the American Psychiatric Association, Toronto, Canada, May 15–21, 1982.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorder. *Schizophrenia Bulletin*, 11:1–7, 1985.

Levenstein, S.; Klein, D.F.; and Pollack, M. Followup study of formerly hospitalized voluntary patients: The first two years. *American Journal of Psychiatry*, 10:1102–1109, 1966.

Marengo, J., and Harrow, M. "Abstract and Concrete Thinking in Schizophrenia: A Longitudinal Approach." Presented to the 87th Annual Meeting of the American Psychological Association, Toronto, Canada, September 1–5, 1979.

Marengo, J., and Harrow, M. Schizophrenic thinking: Two and four year followup. *Scientific Proceedings of the 137th Annual Meeting of the American Psychiatric Association*. Washington, DC: The Association, 1984.

Marengo, J., and Harrow, M. Thought disorder: A function of schizophrenia, mania, or psychosis? *Journal of Nervous and Mental Disease*, 173:35–41, 1985.

Marengo, J.; Harrow, M.; Lanin-Kettering, I.B.; and Wilson, A. A manual for assessing aspects of bizarre-idiosyncratic thinking. In: Harrow, M., and Quinlan, D., eds. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, 1985. pp. 394–449.

Neuchterlein, K.H., and Dawson, M.E. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin*, 10:300–312, 1984.

Pogue-Geile, M.F., and Harrow, M. Negative and positive symptoms in schizophrenia and depression: A followup study. *Schizophrenia Bulletin*, 10:371–387, 1984.

Pogue-Geile, M.F., and Harrow, M. Negative symptoms in schizophrenia: Their longitudinal course and prognostic importance. *Schizophrenia Bulletin*, 11:427–439, 1985.

Resnick, H.S., and Oltmanns, T.F. Hesitation patterns in the speech of thought-disordered schizophrenic and manic patients. *Journal of Abnormal Psychology*, 93:80–86, 1984.

Shimkunas, A.M. Reciprocal shifts in schizophrenic thought processes. *Journal of Abnormal Psychology*, 76:423–426, 1970.

Spitzer, R.L.; Endicott, J.; and Robins, E. Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, 35:773–782, 1978.

Strauss, J.S., and Carpenter, W.T. The prediction of outcome in schizophrenia. *Archives of General Psychiatry*, 27:739–746, 1972.

Strauss, J.S.; Carpenter, W.T.; and Bartko, J.J. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 11):61–69, 1974.

Strauss, J.S.; Hafez, H.; Lieberman, P.; and Harding, C.M. The course of psychiatric disorder. III: Longitudinal principles. *American Journal of Psychiatry*, 142:289–296, 1985.

Wechsler, D. *Wechsler Adult Intelligence Scale*. New York: Psychological Corporation, 1955.

Zubin, J.; Magaziner, J.; and Steinhauer, S.R. The metamorphosis of schizophrenia: From chronicity to vulnerability. *Psychological Medicine*, 13 (Supplement No. 3):551–571, 1983.

Zubin, J., and Spring, B. Vulnerability—A new view of schizophrenia. *Journal of Abnormal Psychology*, 36:103–126, 1977.

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Prediction of Long-Term Outcome in Schizophrenia by Prognostic Scales

by Hans-Jürgen Möller, Wilhelm Schmid-Bode, and Detlev von Zerssen

Abstract

The predictive validity of four prognostic scales described in the literature (Gittelman-Klein Scale, Goldstein Scale, Phillips Scale, Strauss-Carpenter Scale) and of four newly developed prognostic scores was tested in two long-term followup studies on schizophrenic patients. Among the prognostic scales of the literature, the Strauss-Carpenter Scale showed the best results; it reached a level that was attained by only three of the newly developed scales. The three other scales of the literature could also prove their predictive validity. It may be concluded from the results that a scale has a comparatively high predictive value when certain dimensions and aspects are considered but that a certain maximum cannot, in general, be exceeded—even if more items are added to the prognostic scale. A higher number of items may be able to guarantee a better stability of predictive power in different samples.

Although there are many discrepancies between the results of followup studies on schizophrenic patients, some findings could be replicated in many cases. Of importance is the result that, in general, schizophrenia has a poor outcome in less than 50 percent of patients. This has been demonstrated by short-term as well as long-term followup studies (Schooler et al. 1967; Bleuler 1972; Astrachan et al. 1974; Hogarty et al. 1974; Strauss and Carpenter 1974; Astrup 1975; Hawk, Carpenter, and Strauss 1975; Affleck, Burns, and Forrest 1976; Strauss and Carpenter 1977; Huber, Gross, and Schüttler 1979; World Health Organization 1979; Achté 1980; Möller et al. 1982*b*; Watt, Katz, and Shepherd 1983). Thus, one of the principal questions of followup research is whether long-term outcome can be predicted on the basis of data available at the time of the first or another index manifestation.

There is a long tradition concerning the differentiation of schizophrenia with good outcome from that with poor outcome. In this context the diagnostic concepts of schizophreniform psychoses, reactive psychoses, atypical psychoses, and schizoaffective psychoses—all describing psychoses with a comparatively good outcome—have to be mentioned (Kant 1941; Langfeldt 1956; Eitinger, Laane, and Langfeldt 1958; Welner and Strömgren 1958; Vaillant 1962, 1964; Phillips 1966; Achté 1967; Stephens 1970; Croughan, Welner, and Robins 1974; Tsuang, Woolson, and Fleming 1979). Besides psychopathological and anamnestic characteristics defining these subtypes, numerous other psychopathological, sociodemographic, and anamnestic data have been described as relevant concerning prognosis (Renton et al. 1965; Strauss and Carpenter 1974; Hawk, Carpenter, and Strauss 1975; Pokorny et al. 1976; Goldberg et al. 1977; Strauss and Carpenter 1977; Wittenborn, McDonald, and Maurer 1977; Bland, Parker, and Orn 1978; Huber, Gross, and Schüttler 1979; World Health Organization 1979; Achté 1980; Möller et al. 1982*b*). Only some of these predictors could be replicated by other research groups, e.g., lower age at onset, insidious onset, no precipitating factors, affective blunting,

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disturbances of premorbid personality or adjustment, and duration of hospitalization before index admission are known as predictors of poor outcome. Besides this substantial agreement on certain prognostic criteria, some findings remain contradictory, e.g., the prognostic implications of certain psychopathological symptoms.

In general the percentage of outcome variance explained by each of these predictors separately is rather low, so their practical value is weak. Therefore in recent studies an effort was made to increase the percentage of explained outcome variance by multivariate analyses (Strauss and Carpenter 1977; World Health Organization 1979; Möller et al. 1982*b*). In fact, the prediction could be remarkably improved. Such findings of multivariate analyses must be considered critically, however, because the multivariate method optimizes the result for the sample in question. Therefore, the practical relevance must be proven by cross validations, a demand usually not fulfilled.

Another approach to improving the prediction of outcome consists of combining relevant predictors, either according to the literature or as identified by their own univariate or multivariate analyses. Such combination scores should prove their predictive power in cross-validation studies, too. This attempt, however, if performed at all, was successful in only a few cases (Strauss and Carpenter 1972, 1974; Kokes, Strauss, and Klorman 1977).

The total score of such a prognostic scale based on univariate or multivariate analyses should guarantee a better prediction than each single predictor item, a presumption that seems false for some of these scales (Möller, Scharl, and von Zerssen 1984*a*).

Several prognostic scales for schizophrenic patients have been described in the literature. Some of them, such as the Phillips Scale (Phillips 1966), consist of data only on premorbid adjustment; some focus more on psychopathological data; and some, such as the Vaillant Scale (Vaillant 1962) or the Stephens Scale (Stephens 1970), on anamnestic items. The broadest approach is demonstrated by the Strauss-Carpenter Prognostic Scale, a multidimensional approach combining aspects of psychopathology, social adjustment, and history of illness (Strauss and Carpenter 1974). The predictive validity of these prognostic scales has been shown in some Anglo-American studies (Kokes, Strauss, and Klorman 1977), but there is a lack of comparative research concerning the predictive power of different prognostic scales in the same sample.

The aim of the following study was

- to test the predictive validity of four prognostic scales, described in the Anglo-American literature, when used by German-speaking psychiatrists;
- to compare the prognostic value of these scales in the same sample of schizophrenic patients; and
- to compare the scales' predictive value with self-constructed prognosis scores.

Methods

One hundred and three patients (first sample, original sample) who were diagnosed as suffering from a schizophrenic or paranoid psychosis and admitted to the Psychiatric Department of the Max Planck Institute of Psychiatry (Munich) during 1972 to 1974 were followed up 5 to 6 years after discharge. Seventy-seven percent of the patients met the International Classification of Diseases (ICD) descriptions for schizophrenia (ICD 295[1]), and 23 percent met the ICD descriptions for other types of paranoid psychoses (ICD 297, 298.2, 298.3, 298.9). At followup, seven patients had died, five by suicide. These patients were representative of the original sample in terms of sex, age, diagnostic subgroup, marital status, and global psychopathological state at discharge. Complete followup data could be obtained from 78 patients; extensive information about 3 other patients was obtained from relatives. These 81 patients were described in detail in terms of some relevant characteristics in an earlier report in the *Schizophrenia Bulletin* (Möller et al. 1982*b*). Sixty percent of the patients had not previously been admitted to a psychiatric hospital before their admission to the Max Planck Institute (index admission). The sample was almost equally divided between men (49 percent) and women (51 percent); 66 patients (82 percent) were 40 years old or younger, and only 1 patient was over age 60. Only 22 percent were married. Treatment consisted of neuroleptics (mostly haloperidol, in individualized but not high doses) and sociotherapy. Hospitalization was not longer than 3 months for 92 percent of the patients, with only 8 percent staying more than 4 months. Usually 1 to 2 weeks before discharge, the oral neuroleptic medication was changed to an intramuscular depot neuroleptic. The sample seems to be comparable to the schizophrenic clientele of university clinics and probably also to the International Pilot Study of Schizophrenia (IPSS) sample, which excluded chronic patients, but not to the schizophrenic patients of public hospitals where chronic patients are overrepresented.

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[1] Patients with schizoaffective psychoses (ICD 295.7) were excluded from the study because recent research suggests that they should be classified as having a special type or subtype of affective psychoses.

The data were assessed using standardized rating procedures and multiple outcome criteria (see Möller et al. 1982*b*). Among other instruments, the Inpatient Multidimensional Psychiatric Scale (IMPS) (Lorr 1974) and the Global Assessment Scale (GAS) (Spitzer, Endicott, and Fleiss 1976) were used. For the statistical analysis the following outcome criteria were selected:

- Impairment of level of functioning (GAS score)
- Global psychopathological state (self-constructed global rating, scored from 0 to 4; see appendix)
- Personality change in the sense of minus symptoms (self-constructed global rating, scored from 0 to 4; see appendix)
- Impairment of work ability (global rating suggested by Huber, Gross, and Schüttler (1979), scored from 0 to 4; see appendix)
- Paranoid-hallucinatory syndrome (sum of the IMPS scores for paranoid projection and perceptual distortion)
- Depressive-apathetic syndrome (sum of the IMPS scores for anxious depression, retardation, and apathy and impaired functioning)
- Duration of occupational disintegration in the followup period (inability to work, unemployment)
- Duration of psychiatric hospitalization in the followup period

The following four prognostic scales, described in the literature, were applied:

- The Gittelman-Klein Scale on premorbid adjustment (Gittelman and Klein 1969)
- The Phillips Scale on premorbid adjustment (Phillips 1966) in the short version of Harris (Harris 1975)
- The Goldstein Scale of premorbid adjustment (Rodnick and Goldstein 1974)
- The Strauss-Carpenter Prognostic Scale (Strauss and Carpenter 1974)

All these scales were used in the versions published in the appendix of the review by Kokes, Strauss, and Klorman (1977) except that we used an inverse scoring for the Strauss-Carpenter Scale. The data for these scales had to be gathered from the records, so that in this respect the study is complicated by problems typical of retrospective approaches. Fortunately, the records of the Psychiatric Department of the Max Planck Institute of Psychiatry are rather well structured and informative, so in most cases the necessary data were available. In this context, it must be mentioned that some of these scales, such as the Phillips Scale, were developed with the special aim of making it possible to create an evaluation on the basis of data from case records. To avoid a potential bias, the records were evaluated by a psychiatrist who was not informed about the patients' outcome data.

Based on the results of univariate and multivariate analyses of predictors concerning the first sample of patients (Möller et al. 1981; Möller et al. 1982*a*, 1982*b*), four different prognostic scores (Möller, Schmid-Bode, and von Zerssen 1984) were created with combinations of five to eight unweighted predictors (table 1). The first one contains seven variables, the predictive significance of which was proved through univariate analyses. They correlated comparatively high ($r > 0.35$) with the GAS score, and/or they correlated significantly with several other outcome criteria. The second score consists of five variables, which had been found to be the best predictor combination for the GAS score through stepwise multiple regression analyses. Score 4 is based on the results of stepwise multiple regression analyses, as well. It contains all those predictor variables that emerged several times among the combination of the five best predictors concerning the different outcome criteria. The third score is based on the stepwise multiple regression analysis of the 12 IMPS syndrome scores at admission and discharge. It was constructed by combining the five best predictors concerning the GAS score: total score of "disorientation" and "obsessional phobic" at admission and total score of "retardation and apathy," "paranoid projection," and "motor disturbances" at discharge. This is the only scale that consists of psychopathological data alone; the other scales contain psychopathological data as well as sociodemographic data, data on social adjustment, and illness-related information. The predictive values of those newly developed scales were compared with one another in the original sample and with the four prognostic scales described in tne literature.

In a second step, the predictive validity of these prognostic scores was tested in a cross-validation study on 61 patients (second sample, replication sample) with the same diagnosis and characteristics who were inpatients at the Psychiatric Department of the Max Planck Institute of Psychiatry. Of this group, 46 could be followed up to 5 to 8 years after discharge, using the same assessment methods mentioned earlier (Schmid-Bode et al., in press). The 46 patients were representative of the entire group in terms of sex, age, marital status, diagnostic subgroup, and global psychopathological state at discharge. Before their treatment at the Max Planck Institute (index admission), 32 percent of the patients had never been admitted to a psychiatric hospital. Fifty-nine percent of the patients were male and 41 percent female; 83 percent were 40 years old or younger; and none was older than 46. Only 24 percent were married. In this second sample the predictive power of the new scores was evaluated in comparison with the four prognostic scales from the literature.

Table 1. Items of the self-constructed prognostic scores

Score items	Score 1	Score 2	Score 3	Score 4
Premorbid working dysfunction				X
No precipitating factors before first manifestation	X	X		X
Younger age at first hospitalization		X		
No signs of manic depressive disorder at first manifestation				X
Duration of occupational disintegration (5 years before index admission)	X	X		X
Duration of psychiatric hospitalization (5 years before index admission)	X			
No lasting partnership at index admission	X			
Impairment of working performance (during the year after index admission)	X	X		X
Residual syndrome (personality change) before index admission	X			
Poor psychopathological state at discharge	X	X		X
IMPS superfactors				
Organic syndrome at discharge[1]				X
Depressive, phobic-compulsive syndrome[2] at discharge[3]				X
IMPS factors				
Disorientation at admission[3]			X	
Obsessional-phobic at admission[3]			X	
Retardation and apathy at discharge			X	
Paranoid projection at discharge			X	
Motor disturbances at discharge			X	

[1] IMPS factors disorientation + retardation and apathy.

[2] IMPS factors anxious depression + obsessional phobic.

[3] The value (10 percent of the theoretical score = 1, 20 percent = 2, etc.) must be subtracted from the total score.

To analyze the relationship between predictor and outcome variables, product moment correlations were calculated between the total scores of the different prognostic scales and eight outcome criteria. The GAS score was chosen as the main outcome criterion, because it reflects the other outcome criteria best (Moller et al. 1982*b*).

Results

Although the study had to be based on record data, only minor problems arose in collecting the data necessary to fill out the four prognostic scales selected from the literature. The percentage of missing data is rather low. With respect to the percentage of missing data, the Phillips Scale, in the short version described by Harris (1975), is the most practical one for such evaluations based on records (table 2).

Considering the main outcome criterion, the GAS score and the Strauss-Carpenter Scale obtained the best results ($r = 0.43$) in the first sample. The predictive value of the Gittelman-Klein Scale and the Phillips Scale is somewhat lower (about $r = 0.30$), and the Goldstein Scale failed to prove a significant correlation with the global level of functioning.

The Strauss-Carpenter Scale also has the best predictive power in terms of the magnitude and number of significant correlations with other outcome criteria. The scale reached prognostic significance for all eight outcome criteria at a correlation level between $r = 0.32$ and $r = 0.54$. Also the highest correlation with an outcome criterion was achieved by the Strauss-Carpenter Scale: the correlation with personality change is

$r = 0.54$. In comparison with this scale, the Gittelman-Klein Scale and the Phillips Scale had significant correlations with only five and six outcome criteria, respectively, and the Goldstein Scale, with only three outcome criteria.

The best correlations were found for the outcome criterion "personality change." Apart from the Goldstein Scale, the correlation between the prognostic scales and this outcome criterion was higher than 0.40. Also, the prediction of the depressive apathetic syndrome, the impairment of work ability, and the duration of psychiatric hospitalization is rather efficient. In contrast, the paranoid-hallucinatory syndrome and, consequently, the global psychopathological state—which includes productive symptoms as well as minus symptoms—cannot be predicted by three of the four scales. Only the Strauss-Carpenter Scale, which consists of data not only on premorbid social adjustment, but also on psychopathology and other illness-related characteristics, is predictive for these outcome criteria.

The newly developed scores showed better results (see table 3). There was a significant and rather close correlation with nearly all outcome criteria. Concerning the global level of functioning, score 4 obtained the best results ($r = 0.62$), closely followed by scores 1 and 2. Score 3, which consists of only psychopathological data, was less significant. Considering the other outcome criteria, scores 1, 2, and 4 also seem to be rather equivalent in their potential predictive power. They show rather close correlations to nearly all outcome criteria. Only score 3 has a lower, but mostly significant, relationship to the other outcome criteria.

The predictive validity of the prognostic scales and of the newly constructed scores was also tested for the subsample of patients with first manifestations. Concerning the four prognostic scales described in the literature, similar results were obtained. One interesting difference, however, should be mentioned: the Strauss-Carpenter Scale could not prove its superiority to the other three scales, and the Goldstein Scale was not so inferior to the three other scales. Again, the outcome criteria

Table 2. Product-moment correlations of different prognostic scales and outcome criteria, sample I (n = 55–77)

Prognostic scales	Impairment of level of functioning (GAS)	Global psychopathology	Personality change	Impairment of work ability	Paranoid-hallucinatory syndrome (IMPS)	Depressive-apathetic syndrome (IMPS)	Duration of occupational disintegration	Duration of psychiatric hospitalization
Gittelman-Klein Scale (n = 55–70)	0.28[1]	0.20	0.50[3]	0.30[1]	0.21	0.36[2]	0.19	0.34[1]
Goldstein Scale (n = 55–61)	−0.19	−0.07	−0.38[2]	−0.31	−0.14	−0.38[2]	−0.22	−0.31[1]
Phillips Scale (n = 75–78)	0.29[1]	0.22	0.43[3]	0.36[2]	0.19	0.38[3]	0.28[1]	0.46[3]
Strauss-Carpenter Scale (n = 67–77)	0.43[3]	0.42[3]	0.54[3]	0.37[2]	0.38[2]	0.39[3]	0.32[2]	0.49[3]

[1] $p < .05$
[2] $p < .01$
[3] $p < .001$

Table 3. Product-moment correlations of prognostic scores and outcome criteria, sample I (n = 66–76)

Prognostic scores	Impairment of level of functioning (GAS)	Global psycho-pathology	Personality change	Impair-ment of work ability	Paranoid-hallu-cinatory syndrome (IMPS)	Depressive-apathetic syndrome (IMPS)	Duration of occu-pational disinte-gration	Duration of psychiatric hospitali-zation
Score 1	0.53^{3}	0.51^{3}	0.55^{3}	0.45^{2}	0.36^{1}	0.28	0.32^{1}	0.36^{1}
Score 2	0.56^{3}	0.54^{3}	0.64^{3}	0.54^{3}	0.37^{2}	0.29^{1}	0.38^{2}	0.52^{3}
Score 3	0.32^{2}	0.27^{1}	0.35^{2}	0.39^{3}	0.33^{2}	0.27^{1}	0.24^{1}	0.27^{1}
Score 4	0.62^{3}	0.55^{3}	0.72^{3}	0.76^{3}	0.39^{2}	0.38^{1}	0.53^{3}	0.51^{3}

$^{1}p < .05$
$^{2}p < .01$
$^{3}p < .001$

"personality change," "depressive-apathetic syndrome," and "duration of psychiatric hospitalization" could be predicted best, whereas the "paranoid-hallucinatory syndrome" and the "global psychopathological" state were unpredictable by most of the scales except for the Strauss-Carpenter Scale.

Again the newly developed prognostic scores obtained better results in general than the prognostic scales described in the literature. They showed significant and close correlations to nearly all outcome criteria. Only score 3 gave unsatisfying results: it was significantly correlated only to the paranoid-hallucinatory syndrome. As for the whole sample, the predictive power of scores 1, 2, and 4 was nearly equal for all outcome criteria except the paranoid-hallucinatory syndrome, to which the correlations were lower.

It could be expected that, in the original sample of patients in which they have been developed, the newly developed scales show better results than the known prognostic scales. To test this predictive value more realistically, the data of the cross-validation study on the replication sample must be considered (table 4). Scores 1, 2, and 4 reached substantial correlations to all outcome criteria at a high level of significance. Only the paranoid-hallucinatory syndrome could not be predicted by these three scores. Score 3 failed widely to prove predictive significance. There were significant correlations only between score 3 and the outcome criteria "global psychopathological state" and "duration of psychiatric hospitalization." Scores 1, 2, and 4 were of better predictive value, considering the magnitude and frequency of significant correlations with the outcome criteria, than the best single

Table 4. Product-moment correlations of prognostic scores and outcome criteria, sample II (replication sample) (n = 39–46)

Prognostic scores	Impairment of level of functioning (GAS)	Global psycho-pathology	Personality change	Impair-ment of work ability	Paranoid-hallu-cinatory syndrome (IMPS)	Depressive-apathetic syndrome (IMPS)	Duration of occu-pational disinte-gration	Duration of psychiatric hospitali-zation
Score 1	0.44^{2}	0.50^{2}	0.60^{3}	0.49^{2}	0.08	0.45^{2}	0.47^{2}	0.49^{2}
Score 2	0.43^{2}	0.49^{2}	0.56^{3}	0.43^{2}	0.07	0.43^{2}	0.39^{1}	0.42^{2}
Score 3	0.21	0.30^{1}	0.26	0.22	−0.08	0.14	0.23	0.36^{1}
Score 4	0.43^{2}	0.57^{3}	0.59^{3}	0.54^{3}	0.09	0.46^{2}	0.49^{2}	0.47^{2}

$^{1}p < .05$
$^{2}p < .01$
$^{3}p < .001$

predictors. The superiority of score 4, which had been found in the original sample, was not replicable.

In comparison with the newly developed scores, the prognostic scales from the literature obtained slightly less favorable results (table 5) in the replication sample. In general, however, they were on the same predictive level as in the former sample. The Strauss-Carpenter Scale again proved the highest prognostic value, followed closely by the Phillips Scale.

Discussion

Compared with prediction based on single variables, the four prognostic scales described in the literature gave generally better results. The correlations to different outcome criteria were mostly comparable. This is especially true for the Strauss-Carpenter Scale, which could be used to explain, on average, 20 percent of the variance of the different outcome criteria. From this result it can be concluded that a multidimensional approach—taking many different aspects into account, from social functioning to illness-related variables—achieves a better prognostic value than scales regarding less complex concepts, such as premorbid adjustment. Nevertheless these three scales (the Phillips, Gittelman-Klein, and Goldstein Scales) also obtained rather good results—especially the Phillips Scale—showing that poor premorbid functioning is an important predictor for poor outcome.

The newly developed scales produced rather satisfactory results. This is especially true for the three scales that consisted of different aspects and that are, so far, comparable to the Strauss-Carpenter Scale. The scale that consisted of only psychopathological data concerning the index manifestation, however, gave poorer results; this was also evident in the results of the multivariate analyses on which the development of the scale was based. The fact that the newly developed scales also proved their predictive value in the cross-validation study on a second sample of schizophrenics gives confidence to their predictive validity. That is true for the three multidimensional scales, whereas the scale that consisted only of psychopathological data failed to prove its predictive significance in the cross-validation study. The unsatisfying results of the latter prognostic scale are reminiscent of the relative failure of cross-sectional diagnostic and symptomatic criteria associated with outcome (Hawk, Carpenter, and Strauss 1975; Carpenter, McGlashan, and Strauss 1977; Kendell, Brockington, and Leff 1979).

It is not surprising that the newly constructed prognostic scores show better results in the original sample of patients, on which their development was based, than in the scales from the literature. The newly constructed scores also show a slight advantage in the replication sample,

Table 5. Product-moment correlations of different prognostic scales and outcome criteria, sample II (replication sample) (n = 35–46)

Prognostic scales	Impairment of level of functioning (GAS)	Global psychopathology	Personality change	Impairment of work ability	Paranoid-hallucinatory syndrome (IMPS)	Depressive-apathetic syndrome (IMPS)	Duration of occupational disintegration	Duration of psychiatric hospitalization
Gittelman-Kline Scale	0.28	0.28	0.35^{1}	0.13	0.15	0.37^{1}	0.26	0.34^{1}
Goldstein Scale	−0.24	−0.33	-0.39^{1}	−0.12	0.19	−0.35	−0.29	−0.31
Phillips Scale	0.46^{2}	0.48^{2}	0.48^{2}	0.22	0.12	0.48^{3}	0.34^{1}	0.36^{1}
Strauss-Carpenter Scale	0.41^{2}	0.44^{2}	0.52^{3}	0.40^{2}	0.21	0.36^{1}	0.40^{2}	0.46^{2}

$^{1}p < .05$
$^{2}p < .01$
$^{3}p < .001$

which might be explained primarily by the fact that the items of the new scales are most adequate to the structure of our records. Another explanation might be that the new prognostic scores used data not only from patient's history and admission, but also from the discharge period (psychopathological state at discharge). Thus, the prognostic measures were projected further into the actual followup period than the items from traditional prognostic scales. The prognostic value of the psychopathological state at discharge has been emphasized by several authors (Affleck, Burns, and Forrest 1976; Renton et al. 1965; Wittenborn, McDonald, and Maurer 1977).

This result is interesting, especially since the Strauss-Carpenter Scale consists of more items than the newly developed scales. It was also shown by Strauss and Carpenter, however, that a reduction of the long scale on the three items "duration of hospitalization," "poor social relations," and "unemployment" has rather the same predictive value as the whole scale (Strauss and Carpenter 1977). The breakdown of this scale in our own study (Möller, Scharl, and von Zerssen, 1984*b*), however, did not confirm that these items are the most important. Therefore it did not seem meaningful for us to test the predictive value of the short version of the Strauss-Carpenter Scale.

It may be concluded from the results of the comparison of all these prognostic scales that such a scale has a comparatively high predictive value when certain dimensions and aspects are considered, but that a certain maximum cannot, in general, be exceeded, not even by adding more and more items to the prognostic scale. Perhaps a higher number of items can guarantee a better stability of predictive power in different samples.

The Strauss-Carpenter Scale has been criticized for making its prediction on the basis of items that reflect a chronicity of the illness (Bland, Parker, and Orn 1978), so that its results are more a conclusion from the former course of the illness than a real prediction. This argument may also be crucial for the three scales on premorbid adjustment and the self-constructed scores. Therefore it was necessary to study the predictive value of these scales in a sample of first manifestations, to test the real predictive validity, and to avoid the complication mentioned earlier. In the subsample of first manifestations of the original sample, the four scales from the literature and the self-constructed scales also proved their predictive power. It is interesting, however, that the Strauss-Carpenter Scale was not so superior to the other scales in this sample of first manifestations. This may indicate that the advantage of the Strauss-Carpenter Scale in the total sample is, in fact, induced by items reflecting the chronicity of illness. Nevertheless, this scale also achieved results at least as good as the three scales on premorbid adjustment in a sample of first manifestations.

Conclusion

It can be concluded that the four prognostic scales from the literature as well as three of the four self-constructed prognostic scores, which proved their predictive validity in a cross validation, are useful instruments if a prediction of many different outcome areas is intended. If an optimal prognosis of one special outcome area is the goal, however, the best predictor may be the same or a similar variable before the index evaluation, according to the hypothesis of an open-linked system between predictor and outcome variable (Strauss and Carpenter 1974, 1977).

References

Achté, K.A. On prognosis and rehabilitation in schizophrenic and paranoid psychoses. *Acta Psychiatrica Scandinavica*, Suppl. 196, 1967.

Achté, K.A. Verlauf und Prognose schizophrener Psychosen in Helsinki. In: Schimmelpenning, G.W., ed. *Psychiatrische Verlaufsforschung: Methoden und Ergebnisse*. Bern, Stuttgart, Wien: Huber, 1980. pp. 144–158.

Affleck, J.W.; Burns, J.; and Forrest, A.D. Long-term follow-up of schizophrenic patients in Edinburgh. *Acta Psychiatrica Scandinavica*, 53:227–237, 1976.

Astrachan, B.M.; Brauer, L.; Harrow, M.; and Schwartz, C. Symptomatic outcome in schizophrenia. *Archives of General Psychiatry*, 31:37–42, 1974.

Astrup, C. Long-term prognosis of the functional psychoses. In: Mitsuda, H., and Fukuda, T., eds. *Biological Mechanism of Schizophrenia and Schizophrenia-like Psychoses*. Stuttgart: Georg Thieme, 1975. pp. 120–125.

Bland, R.C.; Parker, J.H.; and Orn, H. Prognostic predictors and outcome. *Archives of General Psychiatry*, 35:72–77, 1978.

Bleuler, M. *Die schizophrenen Geistesstörungen im Lichte langjähriger Kranken- und Familiengeschichten*. Stuttgart: Georg Thieme, 1972.

Carpenter, W.T., Jr.; McGlashan, T.H.; and Strauss, J.S. The treatment of acute schizophrenia without drugs: An investigation of some current assumptions. *American Journal of Psychiatry*, 1:14–20, 1977.

Croughan, J.L.; Welner, A.; and Robins, E. The group of schizoaffective and related psychoses—Critique, record, follow-up and family studies. II. Record studies. *Archives of General Psychiatry*, 37:632–637, 1974.

Eitinger, L.; Laane, C.L.; and Langfeldt, G. The prognostic value of the clinical picture and the therapeutic value of physical treatment in schizophrenia and the schizophreniform states. *Acta Psychiatrica Scandinavica*, 33:35–58, 1958.

Gittelman-Klein, R., and Klein, D.F. Premorbid asocial adjustment and prognosis in schizophrenia. *Journal of Psychiatric Research*, 7:35–53, 1969.

Goldberg, S.C.; Schooler, N.R.; Hogarty, G.E.; and Roper, M. Prediction of relapse in schizophrenic out-patients treated by drug and sociotherapy. *Archives of General Psychiatry*, 34:171–184, 1977.

Harris, J.G. An abbreviated form of the Phillips rating scale of premorbid adjustment in schizophrenia. *Journal of Abnormal Psychology*, 84.129–137, 1975.

Hawk, A.B.; Carpenter, W.T., Jr.; and Strauss, J.S. Diagnostic criteria and five year outcome in schizophrenia: A report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry*, 32:343–347, 1975.

Hogarty, G.E.; Goldberg, S.C.; Schooler, N.R.; and Ullrich, R.F. Drug and sociotherapy in the aftercare of schizophrenic patients. II. Two-year relapse rates. *Archives of General Psychiatry*, 31:603–607, 1974.

Huber, G.; Gross, G.; and Schüttler, R. *Schizophrenie. Eine Verlaufs- und psychiatrische Langzeitstudie*. Berlin, Heidelberg, New York: Springer, 1979.

Kant, O. A comparative study of recovered and deteriorated schizophrenic patients. *Journal of Nervous and Mental Disease*, 93:616–624, 1941.

Kendell, R.; Brockington, I.F.; and Leff, J.P. Prognostic implications of six alternative definitions of schizophrenia. *Archives of General Psychiatry*, 36:25–31, 1979.

Kokes, R.F.; Strauss, J.S.; and Klorman, R. Premorbid adjustment in schizophrenia: Measuring premorbid adjustment: The instruments and their development. *Schizophrenia Bulletin*, 3:186–213, 1977.

Langfeldt, G.T. The prognosis of schizophrenia. *Acta Psychiatrica Scandinavica*, Suppl. 110:156, 1956.

Lorr, M. Assessing psychotic behavior by the IMPS. In: Pichot, P., and Olivier-Martin, R., eds. *Psychological Measurements in Psychopharmacology: Modern Problems in Pharmacopsychiatry*. Vol. 7. Basel: Karger, 1974. pp. 50–63.

Möller, H.J.; Scharl, W.; and Zerssen, D.v. Störungen der prämorbiden sozialen Adaptation als Prädiktor für die Fünfjahresprognose schizophrener Psychosen. *Nervenarzt*, 55:358–364, 1984*a*.

Möller, H.J.; Scharl, W.; and Zerssen, D.v. Strauss-Carpenter-Skala: Öberprufung ihres prognostischen Wertes für das 5-Jahres-"Outcome" Schizophrener Patienten. *Archiv für Psychiatrie und Nervenkrankheiten*, 234:112–117, 1984*b*.

Möller, H.J.; Schmid-Bode, W.; and Zerssen, D.v. Entwicklung neuer Prognose-Indizes für Patienten mit schizophrenen Erkrankungen und Oberprüfung ihrer Prädiktiven Validtität. *Fortschritte Der Neurologie-Psychiatrie und Ihrer Grenzgebiete*, 52:421–427, 1984.

Möller, H.J.; Werner-Eilert, K.; Wüschner-Stockheim, M.; and Zerssen, D.v. Relevante Merkmale für die 5-Jahres-Prognose von Patienten mit schizophrenen und verwandten Psychosen. *Archiv für Psychiatrie und Nervenkrankheiten*, 231:305–322, 1982*a*.

Möller, H.J.; Zerssen, D.v.; Werner-Eilert, K.; and Wüschner-Stockheim, M. Outcome in schizophrenic and similar paranoid psychoses. *Schizophrenia Bulletin*, 8:99–108, 1982*b*.

Möller, H.J.; Zerssen, D.v.; Wüschner-Stockheim, M.; and Werner-Eilert, K. Die prognostische Bedeutung psychopathometrischer Aufnahme- und Entlassungsbefunddaten schizophrener Patienten. *Archiv für Psychiatrie und Nervenkrankheiten*, 231:13–34, 1981.

Phillips, L. Social competence, the process-reactive distinction, and the nature of mental disorder. In: Hoch, P.H., and Zubin, J., eds. *Psychopathology of Schizophrenia*. New York: Grune & Stratton, 1966. pp. 471–484.

Pokorny, A.D.; Thornby, J.; Kaplan, H.W.; and Ball, D. Prediction of chronicity in psychiatric patients. *Archives of General Psychiatry*, 33:932–937, 1976.

Renton, C.A.; Affleck, J.W.; Carstairs, G.M.; and Forrest, A.D. A follow-up of schizophrenic patients in Edinburgh. *Acta Psychiatrica Scandinavica*, 39:548–581, 1965.

Rodnick, E.H., and Goldstein, M.J. Premorbid adjustment and the recovery of mothering function in acute schizophrenic women. *Journal of Abnormal Psychology*, 83:623–628, 1974.

Schmid-Bode, W.; Möller, H.J.; Wittchen, U.; and Zerssen, D.v. Social outcome in schizophrenia versus affective and schizoaffective psychoses: Results from a 5 to 8 year follow-up. *British Journal of Psychiatry*, in press.

Schooler, N.R.; Goldberg, S.C.; Boothe, H.; and Cole, J.O. One year after discharge: Community adjustment of schizophrenic patients. *American Journal of Psychiatry*, 123:986–995, 1967.

Spitzer, J.; Endicott, R.L.; and Fleiss, L. The Global Assessment Scale: A procedure for measuring overall severity of psychiatric disturbances. *Archives of General Psychiatry*, 33:766–771, 1976.

Stephens, J.H. Long-term course and prognosis in schizophrenia. *Seminars in Psychiatry*, 2:464–485, 1970.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia. I. Characteristics of outcome. *Archives of General Psychiatry*, 27:739–746, 1972.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia. II. Relationship between predictor and outcome variables. *Archives of General Psychiatry*, 31:37–42, 1974.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia. III. 5-year outcome and its predictors. *Archives of General Psychiatry*, 34:159–163, 1977.

Tsuang, M.T.; Woolson, R.F.; Fleming, J.A. Long-term outcome of major psychoses. I. Schizophrenia and affective disorders compared with psychiatrically symptom-free surgical conditions. *Archives of General Psychiatry*, 36:1295–1307, 1979.

Vaillant, G. The prediction of recovery in schizophrenia. *Journal of Nervous and Mental Disease*, 135:534–543, 1962.

Vaillant, G. Prospective prediction of schizophrenic remission. *Archives of General Psychiatry*, 11:509–518, 1964.

Watt, D.C.; Katz, K.; and Shepherd, M. The natural history of schizophrenia: A 5-year prospective follow-up of a representative sample of schizophrenics by means of a standardized clinical and social assessment. *Psychological Medicine*, 13:663–670, 1983.

Welner, J., and Strömgren, E. Clinical and genetic studies on benign schizophreniform psychoses based on follow-up. *Acta Psychiatrica Scandinavica*, 33:377–399, 1958.

Wittenborn, J.R.; McDonald, D.C.; and Maurer, H.S. Persisting symptoms in schizophrenia predicted by background factors. *Archives of General Psychiatry*, 34:1059–1061, 1977.

World Health Organization. *Schizophrenia: An International Follow-up Study*. Chichester, New York, Brisbane, Toronto: John Wiley, 1979.

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Appendix

Self-constructed outcome ratings:

1. Productive and/or minus symptoms
   - 0 = no symptoms
   - 1 = light symptoms: treatment indicated
   - 2 = marked symptoms: treatment necessary
   - 3 = severe symptoms: treatment necessary
   - 4 = extreme symptoms: patient must be treated, even if he or she refuses.
2. Signs of personality change (minus symptoms) in the past year: patient is less dynamic, less emotional, less resistant against stress, less interested in social contacts, etc.
   - 0 = no minus symptoms
   - 1 = light minus symptoms: only the patient recognizes minus symptoms
   - 2 = marked minus symptoms: minus symptoms are recognized by others
   - 3 = severe minus symptoms: the patient is severely incapacitated in social and professional functioning
   - 4 = extreme minus symptoms: the patient needs help for own basic needs.
3. Ability to work in past year (included as job: paid work, student, housewife)
   - 0 = no restriction
   - 1 = ability to work continuously but light reduction of the premorbid level
   - 2 = marked reduction concerning level of professional functioning and/or time spent working
   - 3 = severe reduction concerning level of professional functioning and/or time spent working
   - 4 = continuous or predominant inability to work

Announcement

Art Therapy Italiana will hold a third summer intensive training institute in creative arts therapy and an international painting and drawing program in Piemonte, Italy, from August 3-17, 1986.

A series of courses and workshops will be offered to professionals and students who wish to develop their therapeutic and expressive skills and to be a part of an ongoing international community in a charming and inspiring environment. The institute offers a complete curriculum in Art and Dance Therapy. A qualified American and European staff have designed the courses in order to integrate the learning process with personal experience.

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The Patient Rejection Scale: Cross-Cultural Consistency

by Hans Watzl, Fred Rist, and Rudolf Cohen

Abstract

The Patient Rejection Scale, which was developed to assess rejecting feelings of family members toward mental patients, was administered to a sample of 80 relatives living with schizophrenic patients in the Federal Republic of Germany. In spite of the cross-cultural differences involved, the response distributions of the German sample and a New York City sample of families were almost identical. The findings are discussed in the context of "expressed emotions" research.

Among the various attempts to relate family factors, e.g., interactions, emotions, and attitudes, to the developmental course of schizophrenia, the construct "expressed emotions" (EE) and its assessment schedule, the Camberwell Family Interview (Brown and Rutter 1966; Brown, Birley, and Wing 1972), have been singularly successful in identifying prognostically unfavorable cases. The earlier findings of higher relapse rates in patients living with critical, hostile, and/or emotionally overinvolved ("high EE") relatives were confirmed in England by Vaughn and Leff (1976) and in California by Vaughn et al. (1982).

The prevalence of high EE families was different in the two samples, however: families rated as high EE were more common in California (67 percent) than in England (57 percent). Two further studies suggest cultural differences in family attitudes toward schizophrenic members. Among Mexican families living in California, only 45 percent were rated high EE (Liberman 1983). In a Federal Republic of Germany report, the respective proportion was 56 percent (Köttgen et al., in press), practically duplicating the British figure.

Considering the almost unlimited number of variables that might determine patient selection for a study, discrepancies among reports should be the rule. As long as systematic, cross-cultural research on family reactions to schizophrenia is lacking, the identification of relevant variables may be facilitated by documenting consistencies among different studies. The present study reports such a remarkable consistency found between New York and the Federal Republic of Germany samples with the Patient Rejection Scale (PRS).

The PRS was developed by Kreisman, Simmens, and Joy (1979) to assess the rejecting feelings of relatives toward mental patients who return to live with their families. The authors report correlations of the PRS with rehospitalization and various indices of adjustment, symptomatology, and family burden. The construct validity of the PRS was supported by correlations with patients' reports of the way their families treat them. Recently, Kreisman mentioned an unpublished comparison with "expressed emotion" components that showed no relation between PRS score and "emotional overinvolvement" but did show correlations with "criticism" and "hostility" (personal communication, May 1984). Consisting of short, affective statements, the PRS could easily be translated into the German language.

We have compiled the self-reports of 80 relatives (33 mothers, 21 fathers, 11 husbands, and 15 other relatives) of 53 schizophrenic inpatients (35 male, 18 female; median age = 16) diagnosed

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Table 1. Patient Rejection Scale: Responses of a New York sample and a German sample

Item	Response distributions (percent)			Corrected item-total correlation NY/FRG
	Often NY/FRG	Sometimes NY/FRG	Never NY/FRG	
It gets easier to understand him/her.[1]	43/27	44/44	13/29	.45/.12
He/she is an important part of my life.[1]	88/71	9/22	3/7	.32/.34
I don't expect much from him/her anymore.	15/23	36/37	47/40	.38/.36
I'm tired of having to organize my life around him/her.	14/18	34/31	52/51	.56/.54
I enjoy being with him/her.[1]	45/50	47/37	8/13	.63/.61
I just don't care what happens to him/her anymore.	0/1	5/7	95/92	.30/.31
I get more and more irritated with him/her as time goes on.	13/20	37/43	50/37	.59/.52
If he/she leaves me alone, I leave him/her alone.	36/45	41/32	24/23	.42/.41
I don't mind doing things for him/her.[1]	61/72	26/15	13/13	.09/.10
I feel that I can help him/her get better.[1]	48/57	31/31	21/12	.59/.39
I wish he/she had never been born.	4/4	12/12	84/84	.50/.46

[1] Reverse scoring items (disagreement instead of agreement with the statement is scored as rejection).

according to *DSM-III* (American Psychiatric Association 1980). The item statistics of this German form are almost identical with Kreisman's original data of (single) family members of 133 discharged schizophrenic patients in New York City. Response distributions and item-total correlations of both samples are listed in table 1. Spearman rank correlations between New York and German response frequencies, calculated across the 11 items, separately for each scale point are rho = .97, rho = .81, and rho = .95. The agreement is so convincing that the statistical tests could have been omitted. As an estimate of scale reliability, coefficient alpha for the American version is .78; for our German translation it is .72.

The similarity in these distributions may appear surprising in view of the studies mentioned earlier showing cross cultural, differing rates of expressed emotions assessed by the Camberwell Family Interview. This discrepancy may be due to defensive verbal stereotypes induced by the questionnaire's intrusive statements, such as, "I wish she had never been born." Even if this objection is correct, however, the conformity in the use of defensive responses in New York City and the Federal Republic of Germany appears to be remarkable.

Furthermore, the Camberwell Family Interview results in a score combining critical comments, hostility, and emotional overinvolvement. This last component is not represented in the PRS. Thus, the cultural or ethnic differences between EE studies may be induced by differences in emotional overinvolvement, while the correspondence of the PRS responses could reflect similar critical attitudes in New York City, England, and the Federal Republic of Germany.

Finally, because of the lack of detailed descriptions of the families studied, we cannot exclude a third possibility. Factors such as family size, living space, and social networks (Lukoff et al. 1984) may generally determine attitudes toward the schizophrenic patient more than cultural, ethnic, or religious factors that could be confounded with the first ones.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Brown, G.W.; Birley, J.L.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry,* 121:241-258, 1972.

Brown, G.W., and Rutter, M.L. The measurement of family activities and relationships. *Human Relations,* 19:241-263, 1966.

Köttgen, C.; Sonnichsen, I.; Mollenhauer, K.; and Jurth, R. The family relations of young schizophrenic patients: I. Results of the Hamburg Camberwell Family Interview study.

International Journal of Family Psychiatry, in press.

Kreisman, D.E.; Simmens, S.J.; and Joy, V.D. Rejecting the patient: Preliminary validation of a self-report scale. *Schizophrenia Bulletin,* 5:220-222, 1979.

Liberman, R.P. "Mental Health Clinical Research Center for the Study of Schizophrenia: Summary 1983." Unpublished manuscript, 1983.

Lukoff, D.; Snyder, K.; Ventura, J.; and Nuechterlein, K.H. Life events, familial stress, and coping in the developmental course of schizophrenia. *Schizophrenia Bulletin,* 10:258-292, 1984.

Vaughn, C.E.; and Leff, J.P. The influence of family and social factors in the course of psychiatric illness. *British Journal of Psychiatry,* 129:125-137, 1976.

Vaughn, C.E.; Snyder, K.S.; Freeman, W.; Jones, S.; Falloon, I.R.H.; and Liberman, R.P. Family factors in schizophrenic relapse: A replication. *Schizophrenia Bulletin,* 8:425-426, 1982.

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Family Therapy in Schizophrenia

Family Therapy in Schizophrenia, edited by William R. McFarlane, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). Although family therapy originated nearly three decades ago through early efforts to understand the etiology of schizophrenia, it was all but abandoned in later years, as biological or constitutional factors were shown to contribute more to the occurrence of schizophrenia than family psychopathology. Though the etiology of the disorder is still understood in constitutional terms, recent findings suggest that the family—the key social unit in the patient's life—may have a substantial impact on treatment.

Family Therapy in Schizophrenia focuses on approaches developed since 1975. These approaches, brought together here for the first time, differ from earlier ones, according to McFarlane, in two important ways: "They seem to have major therapeutic effects on the schizophrenic process, beyond those achievable with drug therapy; and they all—with the exception of the systemic variety—start from a major expansion of family systems theory that includes extrafamily factors."

This volume presents practical strategies—developed by leading family therapists and researchers—for involving families of schizophrenics in the therapeutic process. The book is addressed to family clinicians, psychiatrists, rehabilitation counselors, psychiatric nurses and social workers, hospital and clinic administrators, and students in training for years to come.

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Language Changes in Schizophrenia: A Limited Replication

by Rodney Morice and Don McNicol

Abstract

Similar changes in the spoken language of schizophrenic patients were demonstrated in two separate studies. Schizophrenic patients used less depth of clausal embedding and fewer reduced relative clauses; they uttered more semantically deviant sentences and were more dysfluent than either manic patients or control subjects. They appeared to demonstrate a language impairment characterized especially by reduced syntactic complexity. Four linguistic variables in a discriminant analysis produced an overall diagnostic confidence for schizophrenia in "grouped" subjects of 87 percent, replicating at 83 percent for "ungrouped" subjects from a separate study. These results demonstrate the stability of language changes in schizophrenia, together with acceptable levels of diagnostic sensitivity and specificity. Currently, the possible diagnostic utility of language analysis is constrained by its time-consuming nature. The issue of whether the language changes represent a specific (linguistic) or general cognitive impairment is being addressed in a second phase of the current study.

Language changes in schizophrenia have been described frequently, most notably by Kraepelin (1919) and Bleuler (1950). Indeed, they have always played a salient role in the diagnosis of the disorder, whether they were regarded as evidence of "thought disorder" (following a Bleulerian model) or "speech disorder" (following a Kraepelinian model).

Despite this salience, there have been few attempts to characterize in linguistic detail the changes that do occur. At the clinical level, recent progress has been made with the development of Andreasen's (1979*a*, 1979*b*) Scale for the Assessment of Thought, Language, and Communication. Rochester and Martin (1979) have described changes in the cohesion between sentences in the discourse of some thought-disordered schizophrenic patients, making their speech hard to follow; those findings have been replicated by Wykes and Leff (1982). Gottschalk (1978) has examined the content of spoken language but not from a primarily linguistic or semantic point of view. As noted in various reviews of language and schizophrenia (Pavy 1968; Maher 1972; Schwartz 1978; Rieber 1980; Darby 1981), little attention has been paid to a detailed examination of syntax and fluency.

Morice and Ingram (1982) recently reported changes in the syntax, semantics, and fluency of speech in schizophrenic patients, changes sufficient to discriminate the schizophrenics from manic patients and nonpatient control subjects. In particular, the speech of schizophrenic patients was found to be grammatically less complex and more dysfluent and to contain more syntactic and semantic errors than the speech of manic patients and control subjects. It was suggested that language analysis could possibly be developed into a diagnostic aid for schizophrenia.

The study reported in this article sought to rectify some methodological and statistical problems present in the earlier study, so it was not merely an attempt at replication. It developed into a "reverse replication"; data from the earlier South Australian study were submitted to

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statistical procedures derived from the current Tasmanian study. Results from the earlier study were not permitted to influence the selection of variables used in the current study.

Method

Subjects. Patients were selected from consecutive admissions to two psychiatric facilities in southern Tasmania. The patients selected had received a provisional diagnosis of schizophrenia or mania on admission. The Diagnostic Interview Schedule (DIS) (Robins et al. 1981) was administered to all patients by one of two trained psychiatrists, and only those patients who received a *DSM-III* (American Psychiatric Association 1980) diagnosis of schizophrenia or mania were included in the study. All subjects with evidence of mental retardation, head injury or brain damage, or drug or alcohol abuse were excluded. Additionally, subjects had to have spoken English as their first language or used it as their first language before puberty. The same inclusion criteria were applied to nonpsychiatric control subjects, who were also screened on the DIS. Control subjects were selected from a range of hospital staff volunteers and were matched as closely as possible to the patients for age, sex, education, and social class. They were unaware of the nature of the study except that it had something to do with "the way people communicate." Patients received the same explanation.

Nineteen patients either did not receive a *DSM-III* diagnosis of schizophrenia or mania ($n = 17$) or refused the DIS ($n = 2$). Three manic patients were excluded because of alcohol and/or drug dependence, and one schizophrenic patient and one manic patient were excluded because of probable mental retardation. Four control subjects were excluded after receiving *DSM-III* diagnoses of substance abuse disorders (three alcohol, one drug). From the original 83 subjects tested, 55 remained in the study: 17 *DSM-III* schizophrenic patients, 19 *DSM-III* manic patients, and 19 nonpsychiatric, nonpatient controls.

Sociodemographic characteristics of the three diagnostic groups are listed in table 1. There were few statistically significant differences between the groups. Manic patients were older than schizophrenic patients ($t = 2.22$, $df = 34$, $p < .05$). Schizophrenic patients tended to be single, control subjects married, and manic patients divorced ($\chi^2 = 13.9$, $df = 4$, $p < .01$). Control subjects were of higher social class than schizophrenic patients ($t = 2.68$, $df = 34$, $p < .05$) and manic patients ($t = 2.30$, $df = 36$, $p < .05$). There were no significant group differences for social class, however, when parents of the three diagnostic groups were compared, suggesting that both schizophrenic and manic patients had experienced some social class "drift."

Control subjects had had more years of education than schizophrenic patients ($t = 3.38$, $df = 34$, $p < .01$) and manic patients ($t = 3.09$, $df = 36$, $p < .01$); because many were nurse trainees with their training counted in "years of education," however, these differences were probably of no functional significance.

All subjects except one member of the schizophrenic group completed the Ravens Progressive Matrices. The intention was to make more objective assessments of intelligence than reference to prior education would allow. It is unlikely, however, that Ravens scores for the patients provided accurate assessments of intelligence, because distractibility and partial cooperation often seemed to produce poorer results than might otherwise have been expected. Nevertheless, using quotients derived from the Ravens scores, the manic patients were slightly below schizophrenic patients (85.2 vs. 94.4; $t = 1.99$, $df = 33$, $p < .05$), while controls (at 107.5) were significantly above both schizophrenic patients ($t = 3.11$, $df = 33$, $p < .01$) and manic patients ($t = 5.99$, $df = 36$, $p < .001$). When the testing situations, as well as the education records were taken into account, however, all subjects included in the study were regarded as having at least average ability.

Some clinical characteristics of the two patient groups are listed in table 2. As might be expected on clinical grounds, the schizophrenic patients were significantly younger than the manic patients at their first admission for a psychotic illness ($t = 2.67$, $df = 34$, $p = .01$). Both groups had an equivalent mean duration of illness, although schizophrenic patients had been admitted to the hospital slightly more frequently. The premorbid functioning of the schizophrenic patients had been significantly poorer than that of the manic patients, as measured by the Premorbid Assessment Schedule (PAS) (Cannon-Spoor et al. 1982) ($t = 3.44$, $df = 33$, $p = .002$).

At the time of speech sample collection, both groups were receiving equivalent amounts of antipsychotic medication (measured in chlorpromazine equivalents), and they had been receiving medication for an equivalent length of time (in the estimation of duration of medication only, lithium carbonate was included with antipsychotic medication). Ratings on five selected items (tension, emotional withdrawal, mannerisms and posturing, motor retardation, and uncooperativeness) of the Brief Psychiatric

Table 1. Sociodemographic characteristics of the three diagnostic groups of Tasmanian subjects

Sociodemographic characteristics	Schizophrenic patients (*n* = 17)	Manic patients (*n* = 19)	Control subjects (*n* = 19)
Age: Mean (SD)	30.3 (8.4)	37.8 (11.4)	31.5 (11.9)
Sex:			
Male	11	6	7
Female	6	13	12
Marital status:			
Single	11	5	7
Married	1	8	11
Divorced	5	6	1
Social class of parents: 1-5 scale, mean (SD)	3.1 (0.8)	3.2 (0.9)	3.3 (0.7)
Social class of subjects: 1-5 scale, mean (SD)	3.7 (1.2)	3.5 (1.0)	2.9 (0.4)
Years of education: mean (SD)	11.5 (1.7)	11.2 (2.7)	13.5 (1.7)
IQ scores derived from Ravens: mean (SD)	94.4 (14.7)	85.2 (12.7)	107.5 (10.0)

Rating Scale (BPRS) (Overall and Gorham 1962) at the time of speech sample collection revealed the schizophrenic patients to be slightly, but not significantly, more disturbed than the manic patients.

These 55 Tasmanian subjects, having been diagnosed according to the more stringent criteria of *DSM-III*, were selected as the "grouped" cases for the discriminant analysis. Subjects from an earlier study conducted in South Australia were selected as "ungrouped" cases for the analysis. This method of "reverse replication" was chosen because the subjects in the initial study had been diagnosed by the Present State Examination (PSE)/CATEGO system (Wing, Cooper, and Sartorius 1974). That system—largely because of its reliance on first-rank symptoms—might now be regarded by many as less appropriate than other diagnostic systems including *DSM-III* (Bland and Orn 1979; Kendell, Brockington, and Leff 1979; Helzer, Brockington, and Kendell 1981; Silverstein and Harrow 1981; Kendell 1982; Lewine et al. 1982).

The characteristics of the 63 South Australian subjects have been described elsewhere (Morice and Ingram 1982); in summary, the differences and similarities between the three diagnostic groups were equivalent to those between the three groups of this current Tasmanian study.

To bring the diagnoses of the South Australian subjects closer to those of the Tasmanian subjects, the PSE protocols of the former were reexamined and diagnoses assigned according to the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1975). Insufficient data had been collected in the previous study to allow for accurate *DSM-III* diagnoses. As a result of this procedure, 15 RDC schizophrenic and 14 RDC manic patients emerged from the previous 34 PSE/CATEGO schizophrenic and 11 PSE/CATEGO manic patients; 3 PSE/CATEGO schizophrenic patients were reclassified as manic according to RDC. The number of controls remained unchanged at 18.

Subject numbers used in data analysis were as follows: DIS/*DSM-III* "grouped" subjects (Tasmanian study)—17 schizophrenic, 19 manic, 19 control; and PSE/RDC "ungrouped" subjects (South Australian study)—15 schizophrenic, 14 manic, 18 control.

Language Analysis. Free speech samples of 1,000 words were collected from all subjects, who were asked to talk about anything they wished. Collector comments were kept to a minimum. In particular, two-way dialog was avoided. Most samples comprised narrative accounts of the subjects' life histories. When necessary, standard prompts were used such as, "Perhaps you could talk about a holiday," or "Tell me about your family." All speech samples were collected at one sitting.

As soon after collection as possible, the collector transcribed the speech samples from tape without punctuation. One of two trained grammatical coders then checked the transcription for accuracy and inserted sentence boundaries, which were determined by syntax, intonation, and meaning (in that order of priority). A sentence consisted of one independent or main clause, together with any attached dependent or subordinate clauses and coordinated clauses. The second coder then checked the sentence boundaries and proceeded with manual grammatical analysis of the

Table 2. Clinical features of schizophrenic and manic patient groups: means (SD)

Clinical features	Schizophrenic patients (n = 17)	Manic patients (n = 19)
Age at first admission[1]	22.4 yr (5.5)	30.7 yr (12.2)
Duration of illness (from first admission)	8.9 yr (7.1)	8.1 yr (5.8)
Number of previous admissions	8.1 (7.5)	5.8 (4.4)
Premorbid Assessment Schedule (PAS) total score[1]	0.42 (0.15)	0.24 (0.16)
Modified (selected items) Brief Psychiatric Rating Scale (BPRS) score at time of speech sample	4.2 (3.7)	3.0 (2.4)
Daily dose of antipsychotic medication (chlorpromazine equivalent) at time of speech sample	784 mg (788)	731 mg (801)
Duration of antipsychotic and/or antimanic medication at time of speech sample	200 days (326)	194 days (368)

[1] $p < .01$.

sample. Coders were unaware of the group to which speech samples belonged. While they may have been able at times to assume patient status on the basis of a speech sample's bizarre nature, they remained blind to the actual diagnosis.

Analysis was guided by a coding manual (Ingram and Morice 1979) based on a standard reference on English grammar (Quirk et al. 1972). Conventional surface grammar was used rather than transformational grammar (Chomsky 1965), because of the latter's continuing controversial status (Bresnan 1978). Analysis was performed as a series of "scans" over each sentence.

The first scan tagged sentences as well-formed major, syntactically or semantically deviant, minor, or unanalyzable sentences. The second scan located and named dysfluencies (pause fillers, repeat words, retraced false starts) and lexical errors (neologisms, derivational and inflectional morphological errors, phonological errors).

The most important scan was the construction of a syntax tree diagram for each analyzable sentence. The tree diagrams (see figure 1) captured the surface grammatical structure of each sentence. Each clausal element was identified as a marked node in the tree, with phrase level information making up the tree's base. Detailed structural indices, verb phrase markings, and dependent clause types were subsequently identified and counted during a computer scan of the syntax trees. The structural indices included variables such as mean length of sentences, percentage of complex sentences in the sample, mean number of dependent clauses per sentence, and mean maximum depth to which dependent clauses were embedded.

Syntactic errors were tagged and categorized in the final manual scan. In the initial study (Morice and Ingram 1982), 22 categories of syntactic error were used, but subsequent interrater reliability studies revealed inadequate agreement among raters, probably because of the large number of categories creating an overlap of boundaries. For this study, syntactic errors were merely categorized as sentence level errors (indeterminate clause structure or telescoped sentences) or as errors of omission or commission. To date, no attempt has been made to distinguish between trivial and serious syntactic errors—a nontrivial exercise.

All linguistic data, including the tree diagrams, were disk-stored for processing by microcomputer. A specially written program package, PSYCHLAN (Heatley 1981), enabled data entry and subsequent generation of a detailed linguistic profile for each subject. Each profile consisted of 64 variables, computed mainly as percentages or mean values and measuring various aspects of syntactic structural complexity, verbal fluency, dependent clause type, marked sentence type, verb phrase markings, and semantic and syntactic integrity.

Not all variables can be described here; full descriptions are provided in the study manuals (Ingram and Morice 1979; Heatley 1981). Preempting the "Results" section, however, brief descriptions of the four variables selected for use in the discriminant analyses are given here.

Mean maximum depth of clausal embedding was computed after the deepest level of embedding in each sentence was recorded. Generally

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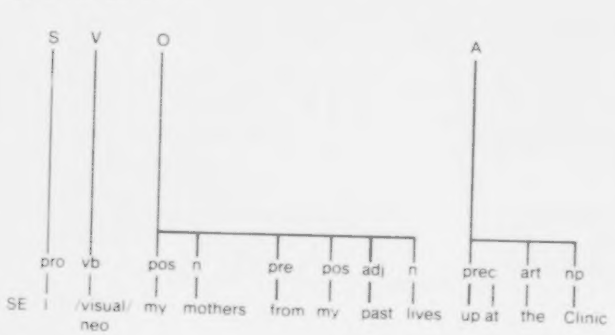
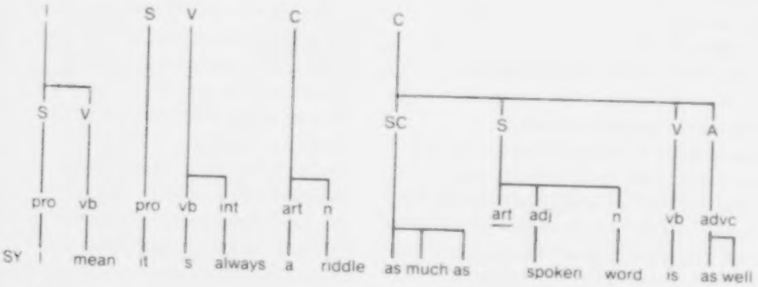
Figure 1. Examples of syntax tree diagrams, the third and most important "scan" of manual grammatical analysis

Subject 5, Sentence 33



Subject 33, Sentence 16



Note.—S = subject, V = verb clause, O = object, C = complement, A = adverbial, CN = coordinator, SC = compound subordinator, I = introducer, pro = pronoun, vb = verb, adj = adjective, n = noun, prec = compound preposition, np = proper noun, pre = preposition, int = intensifier, art = article, advc = compound adverb, neo = neologism; SE = semantically deviant sentence, SY = syntactically deviant sentence

speaking, the deeper the level of clausal embedding, the more complex the sentence.

Clausal embedding allows the expression in a single sentence of more complex relationships between objects. For example, a sentence such as

> The boy, who tripped over a stone, which was hidden in the grass, cried,

which contains two levels of embedding, combines the three sentences:

> The boy tripped over a stone. The stone was hidden in the grass. The boy cried.

More than just providing for economy of expression (by reducing the number of words), the complex sentence specifies the relationship between the events: the boy cried *because* he had tripped over the stone. With the use of the simple sentences, the sequence alone *suggests* the causal relationship; it is not clearly specified.

Stated simply, relative clauses are dependent (or subordinate) clauses that modify, or add information to, noun phrases. *Full* relative clauses are preceded by a relative pronoun (who, which, or that) that "stands in" for the head noun, as in

> The boy [*who* tripped over] cried.

The relative pronoun is omitted (elided) in *reduced* relative clauses, as in

> The boy [I saw] tripped over.

In addition, present and past participle clauses may be regarded as reduced relative clauses (Quirk et al. 1972), as in

> The boy [tripping over] cried.

Semantically deviant sentences were difficult to define, probably accounting for the reduced interrater reliability reported later. To be coded as such, a sentence had to be superficially grammatical but unclear in meaning or had to have a distinctly bizarre meaning. Examples of semantically deviant sentences recorded were

> Feelings that feel nice are countered by sort of intrachivalry interesting things that are black and not so nice

and

> I am also commander of the timeprism series that came on stream during the Iranian hostage crisis under my commander-in-chief President Jimmy Carter.

Repeat words and part words need no defining. Each occurrence apart from the target word was counted, and the total for each sample was expressed as a percentage of the total number of words or tokens.

Data Analysis. One-way analyses of variance (ANOVA) across the three diagnostic groups in the Tasmanian study were performed on all 64 variables comprising the linguistic profiles. The variables were grouped into "families" representing coherent linguistic dimensions. The overall level of significance (α) was set at .05, and the Bonferroni correction applied to each variable family (Grove and Andreasen 1982). The variable families with their corresponding levels of significance necessary to meet the overall α of .05 are listed in table 3. Only variables meeting these stringent criteria were selected for entry in a discriminant analysis.

Because of the different diagnostic criteria used in the current study (DIS/*DSM-III*), the 55 Tasmanian subjects were entered in the discriminant analysis as "grouped" cases, meaning that their language variables were used to form the discriminant functions. South Australian subjects from the previous study, with diagnoses reassigned according to RDC criteria, were entered as "ungrouped" cases and the discriminant function scores they received were computed from the functions formed from the Tasmanian subjects. The use of these scores to allocate the South Australian subjects to diagnostic groups thus constituted a methodologically sound, albeit limited, reverse replication.

Table 3. Significance levels for variable families after application of Bonferroni correction, with alpha = .05

Variable family	*n* of variables	Significance
Structural complexity	6	.008
Lexical integrity	5	.01
Fluency	4	.01
Dependent clause type	14	.004
Locus of maximum depth of embedding	5	.01
Coordination	6	.008
Verb phrase markings	12	.004
Syntactic errors	10	.005
Semantic errors	3	.02

Results

After application of the Bonferroni correction to the ANOVA results, four linguistic variables demonstrated significant *F* values. These, together with the mean values for the Tasmanian diagnostic groups, are listed in table 4.

An interrater reliability study conducted during the current study, using two raters and 16 subjects drawn randomly from the three diagnostic groups, revealed acceptable to excellent results for the four variables. Expressed as intraclass correlation coefficients with raters' anchor points removed (i.e., as Cronbach's alpha), these were

Depth of clausal embedding	0.93
Percent repeat words	0.98
Reduced relative clauses	0.66
Semantic deviancy	0.72

Results for these four variables were entered in a discriminant analysis with the Tasmanian (*DSM-III*) subjects as "grouped" cases, and the direct method was used. Values for the discriminant functions formed from variables for the Tasmanian subjects are listed in table 5. Classification results for the "grouped" Tasmanian subjects are listed in table 6.

These classification results revealed only moderately good predictive power with respect to the three diagnostic groups. Closer examination, however, revealed that the poorest discrimination was between manic patients and control subjects.

Sensitivity and specificity figures for schizophrenia can be derived from these classification results. Sensitivity for schizophrenia was 70.6 percent; in other words, the combined language analysis/discriminant analysis correctly identified 12 of the 17 *DSM-III* schizophrenic patients in the study. With respect to schizophrenia, specificity refers to the number of cases without schizophrenia correctly identified by the procedure. Specificity for schizophrenia was 94.7 percent. Combining sensitivity and specificity, the overall diagnostic confidence for schizophrenia was 87.3 percent. Five schizophrenic patients were reclassified as manic, two manic patients were reclassified as schizophrenic, and no control subjects were reclassified into the schizophrenic group.

Replication of these results from the Tasmanian study was attempted by entering in the discriminant analysis as "ungrouped" subjects the South Australian subjects from the earlier study—those who had been "rediagnosed" according to Research

Table 4. Linguistic variables demonstrating significance after application of Bonferroni correction—ANOVA results for Tasmanian subjects

	Mean (SD)		
Linguistic variables	**Schizophrenic patients ($n = 17$)**	**Manic patients ($n = 19$)**	**Control subjects ($n = 19$)**
1. Structural complexity Mean maximum depth of clausal embedding[1]	1.29 (0.13)	1.38 (0.11)	1.45 (0.14)
2. Dependent clause type Percent reduced relative clauses[2]	8.86 (5.44)	11.15 (5.18)	15.63 (5.88)
3. Semantic error Percent semantically deviant sentences[3]	3.26 (5.85)	1.04 (1.37)	0.00
4. Fluency Percent repeat words and part words[4]	1.97 (1.14)	1.18 (0.81)	0.98 (0.53)

[1] Depth of clausal embedding: $F(2,52) = 6.88$, p (corrected) $< .01$
Scheffé: schizophrenics $<$ controls, $p < .01$

[2] Reduced relative clauses: $F(2,52) = 7.10$, p (corrected) $< .05$
Scheffé: schizophrenics $<$ controls, $p < .05$

[3] Semantic deviancy: $F(2,52) = 4.40$, p (corrected) $< .05$
Scheffé: schizophrenics $>$ controls, $p < .05$

[4] Percent repeat words: $F(2,52) = 6.76$, p (corrected) $< .01$
Scheffé: schizophrenics $>$ controls, $p < .01$
schizophrenics $>$ manics, $p < .05$

Diagnostic Criteria. Classification results are listed in table 7.

Sensitivity for schizophrenia for these "ungrouped" subjects was increased to 80.0 percent, and specificity was reduced to 84.4 percent; overall diagnostic confidence was 83.0 percent. Three schizophrenic patients were reclassified into the manic group, two manic patients were reclassified as schizophrenic, and three South Australian controls were reclassified as schizophrenic. Again, the functions displayed poor discriminating power for manic patients vis-à-vis control subjects. These results constituted a very satisfactory reverse replication of the results from the current Tasmanian study.

Having demonstrated replication, a discriminant analysis using the same variables was then run using the combined Tasmanian and South Australian subjects as "grouped" cases (i.e., combining *DSM-III* and RDC schizophrenic patients into one group, *DSM-III* and RDC manic patients into a second group, and both control groups into a third group). Classification results for these combined groups demonstrated a sensitivity for schizophrenia of 75.0 percent and a specificity for schizophrenia of 92.9 percent (table 8). Correlation matrices for the four variables for the combined diagnostic groups are listed in table 9.

Discussion

The spoken language of *DSM-III* schizophrenic patients as a group was characterized by reduced syntactic complexity, as measured by the mean maximum depth of clausal embedding; by a lower percentage of reduced relative clauses; by semantically deviant utterances; and by relative verbal dysfluency, as measured by the percentage of repeated words and part words. In contrast, the speech of matched control subjects was significantly more complex and contained more reduced relative clauses; it was free of semantically deviant utterances and was more verbally fluent. *DSM-III* manic patients, as a group, exhibited a syntactic complexity and a use of reduced relatives midway between those of the schizophrenic patients and the controls; they uttered some semantically deviant sentences. They were more verbally fluent than the schizophrenic patients and only slightly less dysfluent than the controls.

Table 5. Discriminant functions formed for Tasmanian subjects

Function	Eigen value	Percent variance	Canonical correlation	Wilks lambda	Chi-squared	*DF*	Significance
1	1.387	97.61	.762	.405	45.62	8	.0000
2	.034	.239	.181	.967	1.69	3	.6398

Standardized function coefficients

	Function 1	Function 2
Semantic deviancy	.64561	.21916
Percent repeat words	.72163	.50546
Depth of clausal embedding	–.60046	–.09095
Reduced relative clauses	–.49302	.78099

Rotated standardized function coefficients

	Function 1	Function 2
Percent variance	71.85	28.15
Percent repeat words	.87924*	.05638
Semantic deviancy	.66540*	–.14861
Depth of clausal embedding	–.56015*	.23463
Reduced relative clauses	–.01488	.92347*

Group centroids

	Function 1	Function 2
Schizophrenic patients	1.39473	–.72316
Manic patients	–.25337	–.13327
Control subjects	–.99455	.78031

These spoken language characteristics mirrored those for RDC schizophrenic patients, RDC manic patients, and controls demonstrated in a separate study conducted in a different Australian State 4 years earlier. While subject numbers were small in both studies (an expected consequence of the adoption of more stringent diagnostic criteria), the original South Australian results constitute a satisfactory reverse replication of the subsequent Tasmanian findings and confirm the presence of measurable language changes in schizophrenia, especially changes of a syntactic nature, something not widely regarded in the past as characteristic of the disorder.

Structural, or syntactic, complexity can be measured in a variety of ways. Six main indices were contained in the linguistic profiles generated by PSYCHLAN. The majority of schizophrenic patients in both studies recorded low values for all of them—mean length of sentence, percentage of complex (as opposed to simple) sentences in the sample, mean number of dependent clauses per sentence, mean number of coordinated clauses at top level, mean number of embedded coordinated clauses, and mean maximum depth of clausal embedding. There was a statistically significant difference between the groups only with respect to the latter variable, which is also arguably the single most sensitive measure of structural complexity. This has been demonstrated experimentally by Wang (1970). Additionally, complex sentences (containing dependent clauses) are acquired later than simple sentences and, with increasing age, dependent clauses can be embedded more deeply (Crystal, Fletcher, and Garman 1976).

While excessive clausal embedding, either in number of clauses or depth of embedding, can produce comprehension problems for the listener (and perhaps for the speaker as well), excessive *lack* of embedding can make spoken language relatively vague and empty. There is thus an optimal range of structural complexity to provide, on one hand, enough information and specification

Table 6. Classification results of discriminant analysis with Tasmanian *(DSM-III)* subjects as "grouped" cases

		Predicted group membership, *n* (%)		
Actual group	No. of cases	Schizophrenic patients	Manic patients	Control subjects
Schizophrenic patients	17	12 (70.6)	5 (29.4)	0 (0)
Manic patients	19	2 (10.5)	12 (63.2)	5 (26.3)
Control subjects	19	0 (0)	6 (31.6)	13 (68.4)

Note.—"Grouped" cases correctly classified: 67.3%
Sensitivity for schizophrenia: 70.6%
Specificity for schizophrenia: 94.7%
Overall diagnostic confidence for schizophrenia: 87.3%

Table 7. Classification results of discriminant analysis for South Australian (RDC) subjects as "ungrouped" subjects—reverse replication

		Predicted group membership, *n* (%)		
Actual group	No. of cases	Schizophrenic patients	Manic patients	Control subjects
Schizophrenic patients	15	12 (80.0)	3 (20.0)	0 (0)
Manic patients	14	2 (14.2)	11 (78.6)	1 (7.1)
Control subjects	18	3 (16.7)	11 (61.1)	4 (22.2)

Note.—"Ungrouped" cases correctly classified: 57.4%
Sensitivity for schizophrenia: 80.0%
Specificity for schizophrenia: 84.4%
Overall diagnostic confidence for schizophrenia: 83.0%

Table 8. Classification results for discriminant analysis run on combined diagnostic groups from both studies, using same four variables

		Predicted group membership, *n* (%)		
Actual group	No. of cases	Schizophrenic patients	Manic patients	Control subjects
Schizophrenic patients	32	24 (75.0)	8 (25.0)	0 (0)
Manic patients	33	5 (15.2)	19 (57.6)	9 (27.3)
Control subjects	37	0 (0)	13 (35.1)	24 (64.9)

Note.—"Grouped" cases correctly classified: 65.7%
Sensitivity for schizophrenia: 75.0%
Specificity for schizophrenia: 92.9%
Overall diagnostic confidence for schizophrenia: 87.3%

of relationships and, on the other, to avoid breaching the constraints that operate on syntactic processing abilities and short-term memory. In this context, it was interesting to note that 91 percent of schizophrenic patients from both studies recorded values for mean maximum depth of clausal embedding below the mean for the combined control groups. Only three control subjects (8 percent) recorded values below the mean for the combined schizophrenic group.

The general use of relative clauses is acquired after the syntactic processes of conjunction and complementation in the developmental sequence (Bloom et al. 1980), suggesting that it is a more complex syntactic process than the other two. There is also some evidence to suggest that reduced relative clauses are acquired later than full relatives (Slobin and Welch 1973), although Limber (1973) presents some conflicting evidence. Both studies exhibited the characteristic constraints of many child language studies in that they were based on very few subjects. In a detailed review of relative clause acquisition, Hamburger and Crain (1982) only refer to the presence or absence of the relative pronoun in a discussion of very early proto-relative stages of acquisition. They do not distinguish between full and reduced relative clauses at the later stages (ages 4 and 5) of acquisition.

It seems likely that both variables, mean maximum depth of clausal embedding and percentage of reduced relative clauses, provide indices of some aspects of syntactic complexity. Their significantly lower mean values as recorded by schizophrenic patients suggest the presence of some constraints on syntactic complexity in schizophrenia.

In the absence of test-retest and/or

Table 9. Correlation matrices (Pearson's *r*) for discriminating linguistic variables for combined diagnostic groups from both studies

Diagnostic groups	Depth of embedding	Reduced relatives	Semantic deviancy	Repeat words
Schizophrenic patients ($n = 32$)				
Depth of embedding	1.00	.30[1]	.55[2]	.23
Reduced relatives		1.00	.16	.20
Semantic deviancy			1.00	.05
Repeat words				1.00
Manic patients ($n = 33$)				
Depth of embedding	1.00	– .17	– .10	.07
Reduced relatives		1.00	.04	.03
Semantic deviancy			1.00	– .32[1]
Repeat words				1.00
Control subjects ($n = 37$)				
Depth of embedding	1.00	– .18	.16	.19
Reduced relatives		1.00	– .17	.11
Semantic deviancy			1.00	– .20
Repeat words				1.00

[1] $p < .05$.

[2] $p < .001$.

longitudinal studies, it is not possible to attribute these low values to a state or trait phenomenon in schizophrenia. All subjects tested were hospital inpatients, indicating in most cases some degree of psychotic decompensation. This might suggest a state phenomenon. The clinical psychotic status of the subjects was quite variable, however, suggesting a trait phenomenon because language complexity did not seem related to symptomatology.

If reduced syntactic complexity of spoken language represents a trait phenomenon, it is interesting to speculate on its temporal relationship to the schizophrenic syndrome. In other words, does spoken language impairment occur in response to the onset of schizophrenia, or does it preexist the clinical syndrome and provide a possible index of vulnerability to its development? This question could be subjected to empirical investigation.

The most popular view of language impairment in schizophrenia has been articulated clearly by Maher (1972) in his review article. This hypothesis assumes a disorder of thinking as the primary event, with expressed language merely "a mirror of the [disordered] thought," and could perhaps be called the "Bleulerian position." Research findings demonstrating consistent impairment to conceptual representations in schizophrenia would add support to such a position.

The "Kraepelinian position," by way of contrast, seems to assume a greater saliency for linguistic impairment in the causal chain. Certainly constraints on the syntactic complexity of language, especially if they were constraints on language competence in a Chomskyan sense (Chomsky 1976), might reasonably be expected to produce constraints on thinking. This is not a restatement of the controversial Sapir-Whorf hypothesis (Whorf 1956) that semantic categories derive from different linguistic conventions in different societies. Rather, it is suggested that because syntax is a process for expressing relationships between semantic categories and—in the wider realm—between concepts and ideas, constrained syntactic processing may result in a poverty of form and, through it, of content, of thought. This "poverty" would become more clearly manifest in spoken language beyond a certain developmental threshold, as demands for increased conceptual thought were placed on the growing individual. Such a model could provide some explanatory power for the characteristic onset of schizophrenia during the mid- to late adolescence. Results from the initial study did in fact demonstrate a statistically significant correlation between syntactic complexity and age of onset of schizophrenia (Morice and Ingram 1983). In this current study with Tasmanian subjects, there was a positive partial correlation of 0.23 for depth of clausal embedding with age at first admission after controlling for chronological age, but this did not reach statistical significance ($p = .19$).

Such a decompensation into manifest poverty of form and content of speech could perhaps assume another expression, and in this context it is interesting to examine the production of semantically deviant utterances. They were produced in quantity (more than 4 percent of utterances) by 11 schizophrenic patients and by only 1 manic patient. It should be noted, however, that several patients with grossly incoherent speech were not included in the study as the presence of marked formal thought disorder precluded formal diagnosis by either the DIS or the PSE.

There appeared to be a possible relationship in schizophrenia between increased syntactic complexity and semantic deviancy. Nine of the 11 schizophrenic patients with the high rate of semantic deviancy received a depth of embedding (complexity) score equal to or above their overall diagnostic group means. Four of them received scores equal to or above the group means for the controls. These findings were reflected in the correlation matrices (see table 9) in which significant positive correlations between syntactic complexity and semantic deviancy occurred only for the schizophrenic patients. In other words, for these patients only, as syntactic complexity increased, so did the frequency of semantically deviant utterances.

These schizophrenic patients who used syntactically more complex speech also made substantially more syntactic errors, as well as semantically deviant utterances. Often syntactic and semantic errors were present in the one sentence; indeed distinguishing one type of error from the other was often difficult. These associations suggested a possible tradeoff between structural complexity and syntactic and semantic integrity. While these subjects were able to produce more complex speech, it was more error ridden. They still demonstrated a language impairment, though at first glance not one marked by constraints on structural complexity.

Verbal dysfluency, as measured by the relative frequency of repeat words and part words, also characterized both groups of schizophrenic patients. Dysfluencies could indicate problems in syntactic performance, in that they might occur with the attempted production of complex sentences in the presence of complexity constraints. There was a trend for dysfluencies to increase as structural complexity increased for the schizophrenic and control groups but not for the manic group; the association was strongest for the combined schizophrenic group.

In a sense, then, all four variables could represent an impairment of syntactic processing in schizophrenic patients, although semantic deviancy only very indirectly so. The issue of whether the impairment is one of language competence or performance could not be addressed by these studies. In addition to the need for empirical investigation of the state or trait nature of these demonstrated and replicated language changes in schizophrenia, the relation of language changes in particular to cognitive changes in general should be studied. In other words, is schizophrenia characterized by global impairment of cognitive processes or by more selective cognitive deficits (such as language performance or competence), which Bleuler seemed to imply by his coining of the term *schizophrenia*? A second phase of the current study will seek to address this issue by comparing language performance with performance on tasks of visual rotation in imagery across the three diagnostic groups.

A possible clinical diagnostic utility for the combined language analysis/discriminant analysis was suggested by the replication results. The method of grammatical analysis currently used, however, is time-consuming and employs specially trained raters. These constraints make it unlikely that a practical diagnostic aid could be developed in the near future. This situation could change with the development of effective computerized parsers of natural language and of speech-to-text machines.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Andreasen, N.C. Thought, language and communication disorders: I. Clinical assessment, definition of terms, and evaluation of their reliability. *Archives of General Psychiatry,* 36:1315–1321, 1979*a*.

Andreasen, N.C. Thought, language and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry,* 36:1325–1330, 1979*b*.

Bland, R.C., and Orn, H. Schizophrenia: Diagnostic criteria and outcome. *British Journal of Psychiatry,* 134:34–38, 1979.

Bleuler, E. *Dementia Praecox, or the Group of Schizophrenias.* (1911) Translated by J. Zinken. New York: International Universities Press, 1950.

Bloom, L.; Lahey, M.; Hood, L.; Lifter, K.; and Fiess, K. Complex sentences: Acquisition of syntactic connectives and the semantic relations they encode. *Journal of Child Language,* 7:235–261, 1980.

Bresnan, J. A realistic transformational grammar. In: Halle, M.; Bresnan, J.; and Miller, G.A., eds. *Linguistic Theory and Psychological Reality.* Cambridge, MA: MIT Press, 1978.

Cannon-Spoor, H.E.; Potkin, S.G.; and Wyatt, R.J. Measurement of premorbid adjustment in chronic schizophrenia. *Schizophrenia Bulletin,* 8(3):470–484, 1982.

Chomsky, N. *Aspects of the Theory of Syntax.* Cambridge, MA: MIT Press, 1965.

Chomsky, N. *Reflections on Language.* Glasgow: Fontana, 1976.

Crystal, D.; Fletcher, P.; and Garman, M. *The Grammatical Analysis of Language Disability.* London: Edward Arnold, 1976.

Darby , J.K., Jr. *Speech Evaluation in Psychiatry.* New York: Grune & Stratton, 1981.

Gottschalk, L.A. Cognitive defect in the schizophrenic syndrome as assessed by speech patterns. In: Serban, G., ed. *Cognitive Defects in the Development of Mental Illness.* New York: Brunner/Mazel, 1978.

Grove, W.M., and Andreasen, N.C. Simultaneous tests of many hypotheses in exploratory research. *Journal of Nervous and Mental Disease,* 170(1):3–8, 1982.

Hamburger, H., and Crain, S. Relative acquisition. In: Kuczaj, S.A. II, ed. *Language Development, Volume I. Syntax and Semantics.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1982.

Heatley, D. *PSYCHLAN, A Language Analysis Package: Users Manual.* Hobart, Tasmania, Australia: Mental Health Services Commission, 1981.

Helzer, J.E.; Brockington, I.F.; and Kendell, R.E. Predictive validity of *DSM-III* and Feighner definitions of schizophrenia. *Archives of General Psychiatry,* 38:791–797, 1981.

Ingram, J.C.L., and Morice, R.D. *The Grammatical Analysis of Free Speech Samples.* Adelaide, Australia: Flinders University School of Medicine, 1979.

Kendell, R.E. The choice of diagnostic criteria for biological research. *Archives of General Psychiatry,* 39:1334–1339, 1982.

Kendell, R.E.; Brockington, I.F.; and Leff, J.P. Prognostic implications of six alternative definitions of schizophrenia. *Archives of General Psychiatry,* 36:25–31, 1979.

Kraepelin, E. *Dementia Praecox and Paraphrenia.* Translated by R.M. Barclay. Edinburgh: E. & S. Livingstone, 1919.

Lewine, R.; Renders, R.; Kirchhofer, M.; Monsour, A.; and Watt, N. The empirical heterogeneity of first rank symptoms in schizophrenia. *British Journal of Psychiatry,* 140:498–502, 1982.

Limber, J. The genesis of complex sentences. In: Moore, T.E., ed. *Cognitive Development and the Acquisition of Language.* New York: Academic Press, 1973.

Maher, B. The language of schizophrenia: A review and interpretation. *British Journal of Psychiatry,* 120:3–17, 1972.

Morice, R.D., and Ingram, J.C.L. Language analysis in schizophrenia: Diagnostic implications. *Australian and New Zealand Journal of Psychiatry,* 16:11–21, 1982.

Morice, R.D., and Ingram, J.C.L. Language complexity and age of onset of schizophrenia. *Psychiatry Research,* 9:233–242, 1983.

Overall, J.E., and Gorham, D.R. The Brief Psychiatric Rating Scale. *Psychological Reports,* 10:799–812, 1962.

Pavy, D. Verbal behavior in schizophrenia: A review of recent studies. *Psychological Bulletin,* 70(3): 164–178, 1968.

Quirk, R.; Greenbaum, S.; Leech, G.; and Svartvik, J. *A Grammar of Contemporary English.* London: Longman, 1972.

Rieber, R.W. *Applied Psycholinguistics and Mental Health.* New York: Plenum Press, 1980.

Robins, L.N.; Helzer, J.E.; Croughan, J.; and Ratcliff, K.S. National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry,* 38:381–389, 1981.

Rochester, S.R., and Martin, J.R. *Crazy Talk: A Study of the Discourse of Schizophrenic Speakers.* New York: Plenum Press, 1979.

Schwartz, S., ed. *Language and Cognition in Schizophrenia.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1978.

Silverstein, M.L., and Harrow, M. Schneiderian first-rank symptoms in schizophrenia. *Archives of General Psychiatry,* 38:288–293, 1981.

Slobin, D.I., and Welch, C.A. Elicited imitations as a research tool in developmental psycholinguistics. In: Ferguson, C.A., and Slobin, D.I., eds. *Studies of Child Language Development.* New York: Holt, Rinehart & Winston, 1973.

Spitzer, R.L.; Endicott, J.; and Robins, E. *Research Diagnostic Criteria.* New York: Biometrics Research Division, New York State Psychiatric Institute, 1975.

Wang, M. The role of syntactic complexity as a determiner of comprehensibility. *Journal of Verbal Learning and Verbal Behavior,* 9:398–404, 1970.

Whorf, B.L. *Language, Thought and Reality.* Cambridge, MA: MIT Press, 1956.

Wing, J.K.; Cooper, J.E.; and Sartorius, N. *Measurement and Classification of Psychiatric Symptoms.* London: Cambridge University Press, 1974.

Wykes, T., and Leff, J. Disordered speech: Differences between manics and schizophrenics. *Brain and Language,* 15:117–124, 1982.

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Encoding, Memory, and Thought Disorder in Schizophrenia and Mania

by Philip D. Harvey, Elizabeth A. Earle-Boyer, Mark S. Wielgus, and Joyce C. Levinson

Abstract

Schizophrenic ($n = 24$) and manic ($n = 20$) inpatients were compared with a normal comparison sample ($n = 10$) on memory and encoding performance for both self- and other-generated speech. It was found that the level of encoding, as indexed by the level of organization present in the recalled speech, predicted memory performance for both schizophrenic and normal samples. Schizophrenics were less effective at encoding than normals, although the relationship between level of encoding and memory performance was similar for both samples. For the manic patients, however, clinically rated thought disorder predicted memory performance better than encoding performance. It appeared that thought disorder specifically disrupted recall performance, with less of an effect on encoding. These relationships, as well as the lack of a significant relationship between thought disorder and task performance in the schizophrenics, are discussed in terms of their implications for later research in the area of information processing in psychotic patients.

Much recent research has focused on the issue of information processing in psychosis. Specifically, it has been suggested that schizophrenics have difficulties in controlled information processing, especially the active encoding of information (Koh, Kayton, and Berry 1973; Koh, Kayton, and Peterson 1976). For example, it has been demonstrated that schizophrenics, relative to nonschizophrenic psychiatric patients and normals, ineffectively use embedded semantic characteristics in word lists to facilitate recall performance (Koh, Kayton, and Berry 1973). Other data indicate that this deficit is not due to some sort of complete inability to use encoding devices but rather to some other factors. Successful encoding can be induced in schizophrenics (Koh, Kayton, and Peterson 1976), and when this is done, schizophrenic recall performance has been found to improve significantly. The induction of appropriate incidental encoding does take more trials to reach criterion for schizophrenics than for normals. These data also cannot answer whether schizophrenics' deficits are in the recognition of situations that are appropriate for the application of strategies or if consciously applied strategies are simply ineffective in a single trial format. The data from the studies by Koh and colleagues do indicate, however, that schizophrenics have no permanent or irreversible inability to use encoding strategies.

There are several shortcomings in the preceding studies. First, the investigators assessed nonpsychotic schizophrenics identified with diagnostic criteria that are not consistent with current *DSM-III* criteria (American Psychiatric Association 1980). Second, those investigators assessed memory for word lists only and not for speech. As noted by Rochester and Martin (1979), the further the laboratory analog of real-world performance deviates from the criterion of interest (i.e., clinical state variables), the less suitable the data are for making statements about those variables. Finally, those earlier investigators did not correlate behavioral referents of schizophrenia with the results of laboratory testing. As indicated elsewhere (e.g., Harvey and Neale 1983; Neale, Oltmanns,

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and Harvey 1985), few statements can be made about the relationships of laboratory tasks to "real world" clinical phenomena without using a design that correlates the variables.

In this report, we present an investigation that was oriented toward replicating and improving upon the studies by Koh and colleagues. In our investigation, we used samples of currently psychotic manic and schizophrenic inpatients diagnosed with *DSM-III* criteria, along with a normal comparison sample. All subjects were tested on memory for self-generated and other-generated speech samples. Analogous to the Koh, Kayton, and Berry (1973) study, we presented the subjects with two other-generated passages: one consisted of random sentences, and the other was highly integrated. As a result, one passage could be encoded easily if the subject attended to the organization of the passage, but the other could not. Analogous to the Koh, Kayton, and Peterson (1976) study, we also ensured that all subjects encoded certain samples of speech by having them generate their own descriptive passages. Subjects were then asked to recall the self- and other-generated passages, and their memory for both the content of the passages and their level of organization was examined at recall. The Waters and Lomenick (1983) scale for organization of descriptive passages was used to complete the organization ratings. Finally, we completed a clinical assessment of thought disorder for all our patients using Andreasen's (1979*a*) Scale for the Assessment of Thought, Language, and Communication (TLC). To assess the relationship between clinical symptoms and our laboratory measures, we then related those clinical ratings of thought disorder to our indices of memory performance and discourse organization.

To summarize our research, memory and the level of organization present at recall for self- and other-generated speech were examined across the levels of organization of the presented passages or that level of organization extant in the self-generated passage. These memory and organization variables were compared across subject samples and were then correlated with clinical measures of thought disorder. As a result, we believe that this investigation provides some content valid information about encoding and memory performance for the speech of schizophrenic and manic patients, as well as offering the possibility of gaining some insight into the relationship of these variables to clinical thought disorder.

Methods

Subjects. The psychiatric subjects were 24 schizophrenic patients and 20 manic patients selected from consecutive admissions to an acute care unit of a State psychiatric center. Ten normal control subjects were also assessed. Diagnostic information for the patient subjects was collected with a structured psychiatric interview, the Schedule for Affective Disorders and Schizophrenia (SADS) (Spitzer, Endicott, and Robins 1978), and diagnoses used current *DSM-III* criteria (American Psychiatric Association 1980). Patients with a hospital diagnosis of alcoholism or disorders of the central nervous system were excluded. Normal control subjects were selected from the patient care, clerical, and maintenance staffs at the psychiatric center and the State University of New York at Binghamton.

All schizophrenic patients were receiving neuroleptics (either haloperidol or chlorpromazine), and all manic patients were receiving either lithium carbonate or neuroleptics. Diagnoses were generated independently by a doctoral level clinical psychologist (Philip D. Harvey) and by a group of graduate students (Elizabeth A. Earle-Boyer, Mark S. Wielgus, and Joyce C. Levinson). Interrater agreement on the diagnoses was quite good (Cohen's [1960] kappa = .82), and consensus diagnoses were reached in cases of diagnostic disagreement. Demographic data on all subjects are presented in table 1.

Task Description. In the laboratory testing session, subjects received two trials in each of the following three conditions, presented in a random order: listen-random passage, listen-organized passage, and self-generated passage. In the two listen conditions, subjects were asked to listen to a passage with the sequential presentation of 11 concepts describing a commonplace theme (e.g., shopping, a park). In the random passage condition, the 11 concepts were each the subject of a clause; the clauses were unrelated to each other, but all described some aspect of the general theme. The integrated passages, in contrast, were highly organized and were created to contain a level of organization rated as the highest possible according to the Waters and Lomenick (1983) rating system. In the self-generated condition, subjects were presented with an 11-word prompt set consisting of nouns related to the theme, which was written on the top; they were then asked to generate a passage to describe the theme using all the words (concepts) in their presented order. All subjects were told before the first trial that they would later be asked to recall the passages verbatim.

There were eight different 11-word

prompt sets, three from Waters and Lomenick (1983) and five others generated with the procedures specified by Waters and Lomenick. Each set was written on a sheet of paper with all 11 words related to the theme of the set, which was written at the top. From each prompt set, we generated two passages in the format just described; one was highly integrated, and the other consisted of 11 random sentences. Each prompt word was used as the subject of a clause, and the passages in random and organized forms were matched for length. The prompt sets for the two random, two organized, and two self-generated passages were randomly assigned to each subject, and no subject was exposed to two different forms of the same 11-word set.

Table 1. Demographic information on all subjects

	Group					
	Schizophrenic		Manic		Normal	
Variable	Mean	SD	Mean	SD	Mean	SD
N	24	—	20	—	10	—
Age	33.86	9.95	34.28	6.69	27.00	8.60
N female	4	—	8	—	6	—
Years of education	11.83	1.74	12.88	2.11	12.30	1.42
Occupational status[1]	4.4	1.1	3.6	2.0	4.5	1.8
Global thought disorder score[2]	1.90	0.85	1.61	0.92[3]	—	—

[1] From the Current and Past Psychopathology Scales (CAPPS) (Spitzer and Endicott 1968); 7 = unskilled worker; 1 = major professional.

[2] From the Scale for Assessment of Thought, Language, and Communication (TLC) (Andreasen 1979*a*); 0 = absent; 4 = severe.

[3] Since we planned to use the thought disorder ratings for correlational analyses, we examined the distributions of thought disorder scores across the manic and schizophrenic samples. We found that the range of scores was identical (0 to 3) and that the variances did not differ ($F(23, 19) = .85$).

Procedure. Patients were approached within 10 days of their admission to acute treatment units at the State psychiatric center, and their participation was solicited. After informed consent was obtained, each subject was interviewed by a doctoral level psychologist or a trained graduate student. The interviewer first talked with the subject about any topic of interest to them for about 15 minutes, explicitly avoiding any discussion of symptoms or the reason for admission. This interview was tape-recorded and later used to assess clinical thought disorder. After completion of the open-topic interview, the interviewer completed the SADS. Nonpatient subjects were not interviewed with the SADS, but no one with a personal or family history of psychiatric care or hospitalization was tested.

Patient subjects were tested on the memory task by undergraduate assistants at a subsequent hospital visit, within a 3-day period. The normal control subjects were tested in a laboratory at either the psychiatric center or at the State University of New York at Binghamton.

In the memory testing procedure, subjects were told that the investigators were interested in the subjects' ability to generate and recall story material. They were informed that they either would be asked to generate a series of passages describing certain everyday topics or would have such a passage read to them, after which they would be asked to recall the passage. In the two listen conditions, subjects were told, "We are going to read you a story about [title], and afterwards we will ask you to remember it." They were instructed to recall the passage verbatim, with all information in the order presented. They were further told that if they could not recall the passage, they should recall the main points. A 2-minute interval was allowed to elapse between the subjects' hearing the story and recall; that period was filled with unrelated conversation prompted by the tester. Subjects were allowed as long as they required to recall the passage, and their recall was tape-recorded.

In the self-generated condition, subjects were shown a list of 11 prompt words, with a title at the top of the sheet; they were instructed to generate a passage describing the titled theme using all 11 words in the order presented. The passages were tape-recorded for later examination. Recall instructions and procedures were identical to those for the other-generated passages.

Organization Scale. The scale used to generate organization scores was developed by Waters and Lomenick (1983). Those researchers wanted to define a hierarchical scale to measure the level of organization in descriptive passages. Ratings on the scale range from 1 (random collection of sentences) to 7 (completely organized) and are assigned on the basis of the presence and frequency of several elements of organization. These elements are, in order of their organizational importance, groupings of informational items, topic headings for the groupings, and interconnection of the topics with themes or related devices. Across several studies with diverse child and adult subject populations, Waters and Lomenick found that their scale met Guttman scale criteria for a consistently hierarchical scale. Furthermore, they reported that the reliability of the ratings on the 7-point scale was quite good, ranging from .82 to .95 across the three studies. Waters and Lomenick also reported that they believed that encoding performance, analogous to the subjective organization measures used to measure encoding for word list material, could be measured with the scale, since the normal adult subjects who produced the most organized passages produced the best recall. In our report we used the organization scores at recall as an index of how well subjects had encoded the material to be remembered, in the same fashion that Koh, Kayton, and Berry (1973) used subjective organization of the word lists to index how well subjects had encoded the lists.

Coding, Scoring, and Clinical Thought Disorder Ratings. All subjects' self-generated passages, as well as their recall performance, were transcribed from the tape recordings by undergraduate research assistants who were blind to the identities and diagnoses of the subjects. Two other undergraduate assistants generated the organization scores for all passages. These raters read the passages while they were blind to the identities of the subjects and all the experimental hypotheses. The order of the ratings of the passages was randomized between and within subjects. The interrater reliability was quite good, with agreement found to be 84 percent for the generated passages and 87 percent for the recall performance. Averaged ratings were compiled and used in the data analysis. At a later time, the same raters evaluated memory performance, which was scored as the number of the 11 presented concepts—each corresponding to a single prompt word—recalled in the same fashion as they were heard or generated (i.e., no credit was given for words recalled as homonyms or other parts of speech). Interrater reliability was 96 percent for ratings of memory performance.

Clinical thought disorder ratings were completed by two trained raters, a doctoral level psychologist and a graduate student who had no part in conducting the SADS interviews. The ratings were generated from listening to the 15-minute, open-topic interview collected before the SADS, with appropriate corrections as contained in the TLC's rating manual for the interview length. Each rater assigned a global thought disorder score from the TLC's 5-point (0 to 4) scale. Interrater reliability (kappa) was .77, and all cases of disagreement were resolved in favor of the more experienced rater.

Results

The individual data analyses for each aspect of the laboratory task performance are presented in the following sequence: recall performance; organization of self-generated passages; organization scores for recalled passages; and interrelationships of organization, memory, and thought disorder scores. Dependent variables for the analyses are presented in each section.

The dependent variables for the recall performance were the number of concepts, out of a possible 11, that were recalled by the subjects in each input condition. Means and standard deviations of these dependent variables are presented in table 2. The means and standard deviations for organization of the self-generated passages at generation and for the recall of all the passages are presented in table 3.

Since the task was designed to measure processes that were expected to be correlated with each other, and since there were three conditions that contained two of these ostensibly correlated dependent variables, the

Table 2. Memory performance for all subjects in three input conditions

	Group					
	Schizophrenic		Manic		Normal	
Condition	Mean	SD	Mean	SD	Mean	SD
Self-generated	5.6	3.1	6.6	2.0	10.1	0.7
Other-generated: Integrated	2.6	2.0	3.5	1.8	7.3	1.5
Other-generated: Random	2.5	2.2	2.9	2.1	6.2	1.3

Table 3. Organization scores for all subjects at generation and at recall

	Group					
	Schizophrenic		Manic		Normal	
Condition	Mean	SD	Mean	SD	Mean	SD
Self-generated: Generation	3.1	1.2	3.2	1.1	5.8	1.3
Self-generated: Recall	2.3	1.3	2.6	1.1	5.7	1.2
Other-generated: Integrated	2.1	1.4	2.8	1.3	5.4	1.2
Other-generated: Random	1.7	1.4	2.1	1.4	3.8	1.5

first step in the data analysis was the computation of a 3 (diagnosis) × 3 (condition) repeated measures multivariant analysis of variance (MANOVA), with the final factor repeated. The results from both organization scores and memory performance were examined for each condition. The MANOVA found a highly significant overall effect (Wilks λ = .32, approx. F (14,90) = 5.01, $p < .001$). At that point, the decision was made to examine the memory and organization scores separately across the three diagnostic groups and conditions.

For the memory scores, a 3 (schizophrenic, manic, normal) × 3 (self-generated passage, listen-random passage, listen-organized passage) repeated measures analysis of variance (ANOVA), with the final factor repeated, was performed on the data. Significant main effects of diagnosis (F (2,51) = 22.85, $p < .001$) and condition (F (2,102) = 59.17, $p < .001$) were detected. Newman-Keuls followup tests were used to examine the results more closely. It was found that normal subjects performed better overall in their recall of the passages, while manics and schizophrenics performed more poorly than normals, but did not differ from each other. In addition, it was found that all subjects performed better in the generate condition than in the other two, which did not differ from each other. No interactions of the variables reached significance.

A 3 (diagnosis) × 3 (condition) repeated measures ANOVA, with the final factor repeated, was performed on the organization scores for the recalled passages. A significant two-way interaction of diagnosis × condition was detected (F (2,102) = 2.68, $p < .05$). A strategy of simple effects tests (Winer 1962) was used to interpret the interaction. To examine the differential performance of the three diagnostic groups across conditions, three one-way, simple effects tests were computed. Significant effects of diagnosis were found in all three conditions: self-generated passages (F (2,102) = 48.36, $p < .001$), other-generated integrated passages (F (2,102) = 44.50, $p < .001$), and other-generated random passages (F (2,102) = 17.58, $p < .001$). Newman-Keuls followup tests indicated that the same pattern of results was found in all conditions: normals performed better than manics and schizophrenics, who did not differ from each other.[1]

In a further attempt to examine the interaction of diagnosis and condition, three one-way, simple effects tests were used to assess the level of recall organization by condition within each diagnostic group. For both the manic (F (2,102) = 3.58, $p < .05$), and normal control subjects (F (2,102 = 11.71, $p < .01$), significant effects of condition were found, although no significant effects of condition were found for the schizophrenic subjects (F (2,102 = 1.78, ns), indicating that level of organization at recall was unrelated to input condition. Newman-Keuls followup tests indicated that, for normal subjects, recall organization was significantly higher ($p < .05$) in both the self-generated and other-generated, integrated passage conditions than in the random passage condition. For the manics, recall organization of the integrated passages was significantly higher ($p < .05$) than that of the random passages, while the level of recall organization in the self-generated passages did not differ from either of the other two conditions.

Correlational Analyses. In this final analysis, all the memory and organization variables were interrelated and, for the two patient groups, correlated with the clinical thought disorder ratings. These intercorrelations were computed separately for each diagnostic group. The correlation matrix for the normals is presented in table 4, and the matrix

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[1] A one-way ANOVA, with diagnosis as the factor, was used to compare the level of organization present in the subjects' self-generated passages. A significant effect of diagnosis was detected (F (2,51) = 20.72, $p < .001$). Newman-Keuls followup tests indicated that normals produced more highly integrated passages than manics and schizophrenics, who did not differ.

Table 4. Intercorrelations of memory and organization variables for the normal control sample

Variables	1	2	3	4	5	6	7
1. Self-generated: Generation organization	1.0	.98[1]	.60	.22	.37	.44	.38
2. Self-generated: Recall organization		1.0	.67[1]	.17	.46	.57	.39
3. Self-generated: Memory performance			1.0	−.16	.63[1]	.40	.10
4. Other-generated: Integrated-recall organization				1.0	.18	.43	.37
5. Other-generated Random-recall organization					1.0	.48	.71[1]
6. Other-generated: Integrated-memory						1.0	.47
7. Other-generated: Random-memory							1.0

[1] $p < .05$.

for the manics and schizophrenics is presented in table 5.

For the normal subjects, the level of organization present in the self-generated passages was significantly related both to organization present at recall and the memory performance of the subjects. The level of organization present at recall also was positively related to memory performance. Finally, the organization level at recall in the random story condition significantly predicted memory performance. In the schizophrenic sample, the level of organization present at generation of the stories was significantly related to organization levels at, and memory performance in, that same condition. It was also significantly related to the level of organization at recall in the integrated story condition. The level of organization present at recall in the self-generated story condition was significantly related to memory performance in that condition, as well as to the level of organization present at recall in the integrated story condition. Memory performance in the self-generated condition was related to organization and memory performance in both the other-generated story conditions. Organization at recall in the integrated story condition significantly predicted recall in that condition. No correlations between global TLC ratings of thought disorder and any laboratory variables were significant in the schizophrenic sample.

In the manic sample, organization present at generation in the self-generated story condition was significantly related to organization at recall but not to memory performance. The level of organization at output, however, was significantly related to organization at memory in both the other-generated story conditions and to recall performance in the integrated story condition. Recall organization scores in the self-generated story condition were significantly related to memory performance in that condition and to organization scores at recall in the random story condition. Global scores on thought disorder were significantly related to memory scores in the self-generated story condition. To see whether this relationship was stronger than that between thought disorder and organization scores, partial correlations were computed between thought disorder and recall scores and thought disorder and recall organization scores, with each partial removing the effect of the alternative relationship. We found that the partial correlation between thought disorder and memory performance was significant when recall organization was removed (r (20) = −.50, $p < .05$), but that the relationship between thought disorder and recall organization remained nonsignificant with the relationship between thought disorder and memory removed (r (20) = .17, $p < .10$). Finally, we computed the partial correlation between recall organization and memory in the self-generated condition, partialing the relationship between thought disorder and memory performance. The partial correlation was larger than the nonpartialed correlation and became significant (r (20) = .57, $p < .05$).

Discussion

In evaluating the results of the present investigation, it becomes evident that the normal subjects performed consistently better than both patient groups, generating more highly integrated stories, recalling more information in all conditions, and producing more highly organized stories at recall. The best predictor of the amount of information recalled by the normal subjects in both the

Table 5. Intercorrelations of memory and organization variables for the manic and schizophrenic groups[1]

Variables	1	2	3	4	5	6	7	8
1. Self-generated: Generation organization	1.00	.79[2]	.23	.50[2]	.54[2]	.50[2]	.09	−.38
2. Self-generated: Recall organization	.76[2]	1.00	.48[2]	.43	.56[2]	.27	.10	−.41
3. Self-generated: Memory performance	.65[2]	.81[2]	1.00	.20	.01	.28	.24	−.61[2]
4. Other-generated: Integrated-recall organization	.71[2]	.81[2]	.75[2]	1.00	.21	.13	−.03	−.11
5. Other-generated: Random-recall organization	.33	.27	.43	.39	1.00	.06	.26	−.33
6. Other-generated: Integrated-memory	.14	.34	.66[2]	.57[2]	.37	1.00	−.02	−.14
7. Other-generated: Random-memory	.30	.21	.51[2]	.19	.65[2]	.34	1.00	−.20
8. Global thought disorder rating	−.05	−.15	−.06	.02	.10	.09	.20	1.00

[1] The data for the manics are above the diagonal; the data for the schizophrenics are below.

[2] $p < .05$.

self-generated and other-generated random story conditions was the level of organization of the recalled passage, as evidenced by the high correlations between recall organization and memory performance. From these results, we would infer that the level of encoding of the passage, as measured by the level of organization present at recall, predicted success in recall. In the other-generated, integrated condition, these correlations were nonsignificant, indicating perhaps a ceiling effect in recall, limiting the range of the correlations. An interesting aspect of the normals' information-processing performance was that they appeared to impose organization on the random stories, as evidenced by the higher level of organization at recall ($m = 3.80$) than at presentation (level = 1). This increase in the level of story organization from external presentation to recall implies that normal subjects are likely to impose integrative structure on random sentences, in effect adding to the organization inherent in random information.

One final note on the normals' performance is related to the issue of convergent and divergent validity of the various conditions. We believe that the high correlations we consistently found between recall organization and memory performance within condition provide some evidence of convergent validity of the measures of encoding. The lack of cross-condition correlations between organization scores and recall performance (i.e., recall organization–other-generated random, memory performance–self-generated) provides some evidence of divergent validity across conditions in that there does not appear to be some general overriding ability that determines performance on all dependent variables in all conditions.

Several cautions should be mentioned before presenting our interpretation of the data from our patient groups. First, the three task conditions were not equalized *a priori* for discriminating power. As a consequence, that alternative explanation for our findings cannot be completely discounted. Second, the generalized lack of differences between our manic and schizophrenic patient samples could possibly be interpreted as reflecting some general "psychotic" deficits in performance. We believe this interpretation may be in some way reasonable but wish to point out that the nonpsychotic schizophrenic subjects in the studies by Koh and colleagues manifested similar deficits in both encoding and memory performance.

In evaluating the performance of the schizophrenics in the present investigation, it does not appear that there are qualitative differences in their performance relative to the normal sample. The schizophrenics performed consistently more poorly than the normal subjects, although the pattern of correlations (i.e., organization at recall predicting memory performance) was similar to that of the normals. A possible interpretation of these data, therefore, is that the deficit in recall performance manifested by the schizophrenic patients was caused by less effective encoding on the part of these patients. This finding of a deficit in controlled aspects of information processing (i.e., encoding) is consistent with results from earlier investigations (e.g., Koh, Kayton, and Berry 1973; Koh, Kayton, and Peterson 1976; Oltmanns 1978; Harvey, in press) of schizophrenics' information processing.

There appear to be two components of encoding deficit manifested by the schizophrenics. The first is in the area of voluntary elaboration of information presented to them. For example, normals apparently imposed additional organization on random stories, while the schizophrenics failed to do so. When schizophrenics were forced to encode the organization in a story, such as when they generated a passage themselves in the present investigation or when they were required to perform an encoding task to criterion (Koh, Kayton, and Peterson 1976), the relationship between successful encoding and memory performance was qualitatively similar to that in normals.

The second area of encoding deficit appears to be the extent to which incidental encoding tasks normalize schizophrenics' performance. In the present study, enforced encoding via self-generation of discourse passages failed to normalize recall performance. In the Koh, Kayton, and Peterson (1976) study, however, the semantic sorting task performed by the schizophrenic subjects resulted in normal-range recall of word lists. This difference in findings may be due to several different factors. First, in our investigation, there was no external criterion for successful encoding, and the extent of possible recall organization of the speech samples was self-determined in part by the extent of organization present at the generation of the passages. Second, our measure of organization was oriented toward the inherent organizational properties of descriptive passages in speech and may not exactly parallel measures of subjective organization in word lists, which were used to determine the level of encoding in the study by Koh, Kayton, and Peterson (1976). Third, our subjects had only a single generation trial in which to encode the information in the passages, and the performance of Koh's subjects probably would not have been normalized after a single forced encoding trial. Finally, the differences between Koh's and our patient samples were relatively great; our older, lower-socioeconomic status, psychotic patients might have responded differently in the paradigm used by Koh, Kayton, and Peterson (1976).

Finally, it is important to contrast the performance of the manic and schizophrenic samples. In general, the performance of the two groups did not differ on many of the discourse organization and memory dependent variables. The only appreciable performance difference was that the schizophrenic patients did not have an increase in effective encoding in the integrated story condition relative to random stories, probably reflecting a differential inability to use external encoding cues. The manics, in contrast, had significantly higher recall organization scores in the integrated story than in the random story conditions, although they did not differ from the schizophrenics on a between-group basis.

Conclusion

We believe that the correlational analyses provided interesting insights into how clinical thought disorder may be differentially related to performance in the two patient samples. In interpreting these analyses, however, it is important to acknowledge the possibility of artifactual correlations and to regard our conclusions as rather speculative. First of all, organization at recall in the self-generated condition was appreciably less related to memory performance for the manics than for the schizophrenics, suggesting that either different memory processes were operating in the manic patients or that some factor intervened between encoding and recall in those patients to impair performance. We suggest that the correlation between thought disorder and recall may explain these data. Even when the relationship between thought disorder and encoding was partialed out, the correlation between thought disorder and recall deficits remained significant. We believe that clinical thought disorder in manics may interfere with accurate recall at a postencoding level, even though it did not appear to interfere with encoding itself. Further evidence for this interpretation is contained in the fact that when the influence of clinical thought disorder was partialed, recall organization significantly predicted memory performance.

If this is indeed correct, we could interpret the data as indicating that we have found that clinical symptoms were related differentially to memory performance in manic and schizophrenic patients. The implication is that thought disorder in schizophrenia is unrelated to both encoding and memory performance, while thought disorder in mania may interfere with memory performance at a postencoding level. There has been a discrepancy in findings to date in the area of the relationship of clinical thought disorder and memory and information-processing performance. For example, some earlier investigators have reported that certain deficits in information processing were unrelated to clinical state in schizophrenia (Asarnow and MacCrimmon 1981; Harvey, et al. 1981; Frame and Oltmanns 1982; Neale, Oltmanns, and Harvey 1985), while some other data suggest a relationship between clinical thought disorder and other types of information-processing deficits (Oltmanns, Ohayon, and Neale 1979; Manschreck, Maher, and Rucklos 1981; Harvey, in press).

Another interpretation of the differential correlation between global thought disorder ratings and psychological deficits in our two patient samples is that thought disorder in schizophrenia is more heterogeneous than in mania. For example, Pogue-Geile and Harrow (1984) reported that there are several orthogonal symptoms within negative thought disorder (Andreasen 1979*b*) alone, with negative thought disorder being only a component of global thought disorder. In contrast, our own research (Harvey, Earle-Boyer, and Wielgus 1984; Walker and Harvey, in preparation) has indicated that manics are more likely than schizophrenics to manifest only positive thought disorder and that positive thought disorder is most highly correlated with information-processing deficits. The implication is that a more specific assessment of thought disorder in our subjects may have revealed differential relationships between positive and negative thought disorders and performance on our measures. We believe that later researchers would do well to go beyond correlation of deficits and diagnoses and to correlate laboratory performance and clinical symptomatology. In the case of thought disorder, it also appears that a correlation between specific thought disorders and laboratory deficits would be even more informative and useful.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd. ed. Washington, DC: The Association, 1980.

Andreasen, N.C. Thought, language, and communication disorders: I. Clinical assessment, definition of terms, and assessment of their reliability. *Archives of General Psychiatry*, 36:1315–1321, 1979*a*.

Andreasen, N.C. Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1330, 1979*b*.

Asarnow, R.F., and MacCrimmon, D.J. Span of apprehension deficits during the postpsychotic stages of schizophrenia. *Archives of General Psychiatry*, 38:1006–1011, 1981.

Cohen, J. A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, 20:37–46, 1960.

Frame, C., and Oltmanns, T.F. Serial recall by schizophrenic and affective patients during and after psychotic episodes. *Journal of Abnormal Psychology*, 91:311–318, 1982.

Harvey, P.D. Laboratory research: Its relevance to symptom variables. In: Harvey, P.D., and Walker, E., eds. *Positive and Negative Symptoms in Psychosis: Description, Research and Future Directions*. Hillsdale, NJ: Erlbaum, in press.

Harvey, P.D.; Earle-Boyer, E.; and Wielgus, M. The consistency of thought disorder in mania and schizophrenia: An assessment of acute psychotics. *Journal of Nervous and Mental Disease*, 171:458–463, 1984.

Harvey, P.D., and Neale, J.M. The specificity of thought disorder to schizophrenia: Research methods in their historical perspective. In: Maher, B.A., ed. *Progress in Experimental Personality Research*. Vol. 12. New York: Academic Press, 1983. pp. 153–180.

Harvey, P.; Winters, K.; Weintraub, S.; and Neale, J.M. Distractibility in children vulnerable to psychopathology. *Journal of Abnormal Psychology*, 90:296–304, 1981.

Koh, S.D.; Kayton, L.; and Berry, R. Mnemonic organization in young nonpsychotic schizophrenics. *Journal of Abnormal Psychology*, 81:299–310, 1973.

Koh, S.D.; Kayton, L.; and Peterson, R. Affective encoding and consequent remembering in schizophrenic young adults. *Journal of Abnormal Psychology*, 85:156–166, 1976.

Manschreck, T.C.; Maher, B.A.; and Rucklos, M. Formal thought disorder, the type-token ratio, and disturbed voluntary motor movement in schizophrenia. *British Journal of Psychiatry*, 139:7–15, 1981.

Neale, J.M.; Oltmanns, T.F.; and Harvey, P.D. The need to relate cognitive deficits to specific behavioral referents of schizophrenia. *Schizophrenia Bulletin*, 11:286–291, 1985.

Oltmanns, T.F. Selective attention in schizophrenic and manic psychoses: The effect of distraction on information processing. *Journal of*

Abnormal Psychology, 87:212-225, 1978.

Oltmanns, T.F.; Ohayon, J.; and Neale, J.M. The effect of medication and diagnostic criteria on distractibility in schizophrenia. *Journal of Psychiatric Research*, 14:81-91, 1979.

Pogue-Geile, M.F., and Harrow, M. Negative and positive symptoms in schizophrenia and depression: A followup. *Schizophrenia Bulletin*, 10:371-387, 1984.

Rochester, S.R., and Martin, J.R. *Crazy Talk: A Study of the Discourse of Schizophrenic Speakers*. New York: Plenum, 1979.

Spitzer, R.L., and Endicott, J. *Current and Past Psychopathology Scales*. New York: Biometrics Research, 1968.

Spitzer, R.L.; Endicott, J.; and Robins, L. *The Schedule for Affective Disorders and Schizophrenia*. New York: Biometrics Research, 1978.

Walker, E., and Harvey, P.D. "Positive and Negative Symptoms in Psychosis: Internal Consistency and Attentional Correlates." In preparation.

Waters, H.S., and Lomenick, T. Levels of organization in descriptive passages: Production, comprehension, and recall. *Journal of Experimental Child Psychology*, 35:391-408, 1983.

Winer, B.J. *Statistical Principles in Experimental Design*. New York: McGraw-Hill, 1962.

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A Social Network Approach and the Revolving Door Patient

by Robert C. Morin and Edward Seidman

Abstract

Traditional psychiatric treatment approaches have not been very successful with the "revolving door patient." A variety of findings suggest that the social network, as opposed to the individual patient, may be a more viable locus for intervention. A conceptual review and analysis of the literature reveals two constructs salient to understanding social networks: flexibility and stability. The networks of revolving door patients are frequently characterized as inflexible and/or unstable. Assessment methods as well as strategies for balancing flexibility and stability are described. Enlarging the network, increasing multiplexity, and/or reducing the negative effects of attitudinal inflexibility encompass the strategies for increasing flexibility, while developing connections between individuals, generating spans between clusters of people in the network, and increasing multiplexity are recommended for increasing stability. The assets and liabilities of each of these strategies are discussed.

Since the advent of deinstitutionalization, a group of patients alternately referred to as new chronics (Caton 1981), young adult chronics (Pepper, Kirshner, and Ryglewicz 1981), or revolving door patients has emerged. Typically, these revolving door patients demonstrate a pattern of short hospitalization and discharge followed by repeated admissions. The most important characteristic of these patients is that they have rarely been treated successfully. Caton (1981) reports a rehospitalization rate of almost 60 percent and almost 30 percent multiple rehospitalizations in the first year after discharge. Most alternative demonstration programs attempting to facilitate community adjustment are characterized by low rehospitalization rates, although 12 to 18 months after these programs are terminated, patients begin to relapse (Test and Stein 1978; Fenton, Tessier, and Struening 1979; Dellario and Anthony 1981; Test 1981; Salem, Seidman, and Rappaport 1984). Of course, the ongoing instrumental and social support that had been created by these experimental programs had effectively disappeared by that point.

A second important characteristic of revolving door patients is that they are notoriously difficult to work with. Though they are characterized by varying diagnoses (primarily schizophrenia and borderline personality disorder) and symptom profiles, they have similar difficulties in social functioning related to "their acute vulnerability to stress, their difficulty in making stable and supportive relationships, their inability to get and keep something in their lives and their repeated failures of judgment" (Pepper, Kirshner, and Ryglewicz 1981, p. 464). Under stress, their reality testing may be severely impaired, their affect extremely labile, and their impulse control almost nonexistent (Greenwood 1981). Furthermore, the pattern of relatively short periods of acute stress, followed by rapid stabilization, often make it difficult for these patients to perceive their need for treatment. As their symptoms abate, they typically lack insight into the circumstances that led to their hospitalization and perceive their problems as being external and resolved. Immediate release from the hospital is demanded. Upon release, they often sever contacts with mental health professionals, both from the

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hospital and the community (Harris and Bergman 1981).

Understanding the importance of social networks is of particular relevance in working with the revolving door patient. The networks of these individuals before their first admission are smaller and more conflictual than those of healthy individuals, and the continued loss of network members after multiple admissions reduces even further the resources available to aid the patient returning to the community (Lipton et al. 1981). Early intervention in this process may help patients retain more of their network's original strengths to build on, rather than having to create a totally new network. While network intervention strategies are not new, they have not been addressed for this population nor, in particular, from a coherent conceptual framework. It is hoped that such intervention will break and/or curtail the revolving door pattern.

Before presenting various techniques of network intervention for working with the revolving door patient, it is vital to gain an understanding of a network's critical components and their relationship to the experience of the revolving door patient. First, network variables that appear relevant to working with the revolving door population are examined. Next, it is argued that these variables are interrelated and combine to determine two critical network constructs: flexibility and stability. Finally, the concepts of flexibility and stability are used to guide the clinician in planning network interventions.

Key Network Characteristics

The major structural characteristics of a network are size and the pattern of connections among network members (density and clusters). The salient relational characteristics in terms of the focal person are multiplexity and attitudes held by network members toward the patient.

Structural Characteristics

Size. The size of one's network has been found to be negatively correlated to degree of pathology (Pattison et al. 1975; Cohen and Sokolovsky 1978; Hammer 1980). Patients with higher levels of social contact before hospitalization demonstrate better outcome (Strauss and Carpenter 1977); this, too, suggests the importance of network size. After reviewing a variety of community maintenance studies, Budson and Jolley (1978) concluded that the presence of a strong psychosocial kinship system comprised of friends and neighbors, as well as family, was the crucial factor in determining program success. Smaller network size may be both a predictor of rehospitalization (Cohen and Sokolovsky 1978) and a result of hospitalization (Lipton et al. 1981).

Larger networks may provide a patient with access to a greater number of resources. If one member is unable or unwilling to supply the type of support desired, the patient can simply turn to another member. It is not true, however, that more is always better and that merely increasing network size is always therapeutic. There are many cases of psychiatric patients pulling their lives together only after the loss of a significant network member (e.g., an overinvolved mother) or group of members (Beels 1981). Clinical experience suggests that network size is not consistently related to positive outcome. Many of the larger networks are so fragmented that patients feel pulled in several directions at once.

Density. Density refers to the degree of interconnectedness, usually defined as the ratio of actual links to potential ones. Results interrelating network density, resources, psychopathology, and rehospitalization rates are contradictory.

Two studies have found density to be positively related to pathology and rehospitalization among ex-patients (Hammer 1963-1964; Pattison et al. 1975). This finding is paralleled among nonpsychiatric populations (Hirsch 1979, 1980). Denser networks develop between individuals who share a common set of values and expectations. Because of this commonality, there is often little support or tolerance for behavior that does not conform to the group norms and expectations. For the psychiatric patient, this may mean that the network holds a single perception of what his or her role is and should be. Attempts to change this role, possibly by becoming more independent, may be met with resistance.

Tolsdorf (1976) failed to find a significant relationship between density and the availability of either tangible or intangible resources. He also failed to find significant differences in density between psychiatric and nonpsychiatric medical samples. Similarly, Cohen and Sokolovsky (1978) failed to find a relationship between density and the degree of pathology or rehospitalization rates. In fact, using an additional measure of interconnectedness, these authors concluded that schizophrenics form less intertwined networks.

Clusters. Hirsch (1980, 1981) has found that boundary density (the proportion of interconnections between the nuclear family network and the friend network) is related to greater symptomatology, poorer mood, and lower self-esteem and, inversely, to measures of social support in samples of college students, recent widows, and mature women returning to full-time undergraduate study. This suggests that a critical issue regarding connections between network members may be the separation between clusters of relationships.

A cluster can be defined as a group of people richly connected to one another. Clusters are usually composed of individuals who share a common role with each other, e.g., family members, lodge brothers, or coworkers. If these clusters are too interconnected, as in having numerous connections between friends and family, the network begins to look like one large cluster. Consequently, an individual cannot withdraw from one cluster during intracluster tension and obtain support from an alternative cluster. The alternative cluster is too enmeshed with the original cluster to be free of its tension or to hold different values and expectations. This is a particular problem for psychiatric patients because—unlike normal people's networks, which are characterized by five or six clusters—the networks of psychotic individuals contain few clusters (Hammer 1981).

Relational Characteristics

Multiplexity. A multiplex relationship is one in which a network member serves or fulfills more than one role or provides several types of exchange (e.g., advice and information, emotional support) that are important to the patient. Multiplex relationships have consistently been found to be beneficial. That is, they decrease with increasing pathology (Tolsdorf 1976); their absence in the patients' local living environment significantly predicts rehospitalization (Cohen and Sokolovsky 1978); and they relate to overall satisfaction with one's network and with better support and mental health in nonpsychiatric populations (Hirsch 1979, 1980). These findings hold despite the different ways in which authors operationalize the construct.

Multiplex relationships can be advantageous to the patient in two major ways. Each relationship can be satisfying because the patient can derive benefit from any one of several different content areas, and thus can frequently gain satisfaction even if one type of support is not available at a certain time from a network member. For example, a friend who is unable to socialize with the patient at a particular time may, nevertheless, lend the patient money for a movie. Second, there is less chance that any one type of support will be unavailable, since network members overlap in the functions they serve for the patient.

Attitudes Toward the Patient. While no studies have systematically examined the attitudes of all members of an individual's network, examination of familial attitudes suggests this is an important variable. For schizophrenic patients living with key relatives, the level of "expressed emotion" (EE) on the part of family members has been found to be the best predictor of symptomatic relapse (Brown, Birley, and Wing 1972; Vaughn and Leff 1976; Liberman et al. 1979). The EE rating is based on the number of critical or rejecting statements family members make about the patient, as well as any examples of emotional overinvolvement. The relationship between high EE families and high relapse rates is independent of the patient's level of behavioral disturbance or work impairment.

High EE families are more rigid and demanding than low EE families (Vaughn and Leff 1981). They are highly intrusive, making repeated attempts to establish contact and to dictate the behavior and lifestyle of the patient. They do not perceive the patient as legitimately ill and are intolerant of his or her symptoms. The patient often cannot deal with the overstimulation of this socially intrusive environment. If the patient is unable to withdraw from it, the underlying thought disorder may be manifested in florid symptoms resulting in rehospitalization. Clinical observation reveals that most revolving door patients' families fit this high EE pattern.

Just as with network size, density, cluster presence, and multiplexity, the attitudes of the family are not the sole determinants of success or failure in the community. Patients living with low EE families also relapse, and better than 40 percent of those from high EE families remained in the community (Brown et al. 1972). Further research is needed to determine the effect of nonfamily members holding the high EE attitudes.

In summarizing the network characteristics that are potentially important to the well-being of the revolving door patient, we are left with less than a clear picture. Nevertheless, in most cases, large network size, distinct clusters, multiplex relationships, and network members holding tolerant, nonintrusive attitudes toward the patient appear to be independently related to positive outcome.

Emerging Constructs

Flexibility

The difficulty in obtaining consistent findings for any one of the network factors may be a function of their interrelationships and the fact that they all contribute to network flexibility. One network variable providing flexible support may offset another variable that does not. For example, the negative effect of a high EE family can be reduced by having access to social contacts outside the family. Almost 70 percent of those patients whose network options centered largely on their families (i.e., they spent 35 hours a week in contact with their families) were rehospitalized. Those with outside contacts who spent less than 35 hours a week in family contact had a relapse rate of less than 30 percent (Vaughn and Leff 1976).

Watzlawick, Beavin, and Jackson (1967) have suggested that fixed, rigid patterns of interaction characterize families of patients, while flexible patterns characterize less psychopathological relationships. In a flexible network, the positive functions, e.g., acceptance and support, are available both when symptoms are being experienced and when they are not. A flexible network provides the patient with the opportunity to experiment with other social identities, such as friend, wage earner, club member, spouse. Conflict between the patient and network members, or between network members in general, does not result in the patient's losing all network resources. The patient can temporarily withdraw from an area of conflictual overstimulation and not suffer from social isolation because there are other network members to fall back on. If a key network member becomes unavailable, other network members are available to provide, at least partially, the lost support.

Flexibility is a function both of the number of alternative routes available for gratification and of the "flexible" attitudes of network members. More network members, more clusters, and more multiplex relationships all add to the number of social arenas in which the patient can experience different roles and obtain different types of support. The increased options decrease dependency on any one network segment, so that if one member or cluster has difficulty accepting the patient's symptoms, he or she can turn to another member or cluster. Similarly, multiplex relationships increase options by increasing the probability of overlap in the type of support the network supplies. Thus, if the person a patient usually turns to for tangible assistance is unavailable, another network member can take over that function.

The accepting, nonintrusive (low EE) attitude of network members provides the same type of flexibility within a specific cluster. Network members with these attitudes do not demand the patient play one role (either "normal" or "sick") but provide support regardless of the patient's current level of functioning. Also, since low EE members do not demand to be involved in all aspects of the patient's life, they are more supportive of the patient's taking advantage of the flexibility the network's structure offers.

The flexibility of a network can be operationalized as the sum of the number of network members, the number of clusters, the degree of multiplexity, and the level of attitudinal acceptance. However, network research beyond its current embryonic stage of development is required to determine how much weight to attach to each of these components.

Case Example. The concept of flexibility may be clarified by examining the network of a revolving door patient and observing how it is inflexible. Mr. S. was a 26-year-old patient experiencing his eighth psychiatric hospitalization in 6 years. Typically, he was returned to the hospital after having behaved in a threatening and assaultive fashion at home. Increased drug use often preceded readmission.

Mr. S.'s network consisted of three small clusters: a family cluster, a friend cluster, and a hospital cluster. The hospital cluster was unconnected to any members of the other two clusters. There was only minimal contact between the family cluster and the friend cluster. As with many revolving door patients, Mr. S. would typically stabilize within a month and want nothing to do with the hospital. Consequently, the hospital cluster would drop out of his network as soon as he was discharged.

Mr. S.'s family cluster consisted of his mother, with whom he lived, a younger brother with whom he seldom interacted, and an older sister whom he saw twice weekly. His family was clearly of the high EE variety. His mother was overinvolved, intrusive, and demanding; she was constantly concerned with trying to meet all his needs. She was also frustrated by his illness and interpreted his problems as being totally under his control. His sister was hypercritical of him. When Mr. S. returned home, he initially was rewarded for his dependency, but excessive demands were placed on him very quickly. Mr. S.'s friend cluster consisted of three young men in his mother's neighborhood. These men were unemployed, as was the patient. They did little with the patient but socialize, usually around drug use.

Mr. S.'s network was inflexible in that the number of routes he had through which to satisfy his needs were few. Upon discharge, he had only two small clusters within which to function. His mother was his major multiplex relationship and his primary access to instrumental support. This made him extremely dependent upon her. His friends provided a major source of emotional support. Problems with either of these clusters resulted in loss of the corresponding support. Because neither of these clusters provided access to job or housing information, Mr. S. had no one to turn to for aid in these critical life domains.

The attitudes of these two clusters also made it very difficult for Mr. S. to break out of his role of a revolving door patient. If he spent all his time with his family, their intrusiveness and criticisms would generate sufficient stress to result in decompensation. On the other hand, his friends encouraged his unemployed status and his drug use, which would also result in decompensation. The only roles for which Mr. S. received support were those of an "inadequate, sick son" and a "drug-abusing street person." The limited and rigid structure of these clusters, as well as the intrusiveness of the network actors were the antithesis of the network flexibility needed for improved community adjustment.

Stability

Closely related to the concept of flexibility is that of stability. In a sense, the network must have the flexibility or adaptability to remain the same, to retain the same membership over time, even if the patient is temporarily removed from it because of rehospitalization. Leaving the hospital becomes more difficult if the patient finds the social network decimated each time he or she returns home.

Hammer (1963-1964, 1980, 1981) has demonstrated that, for both psychiatric and normal populations, unsupported connections (network members who are connected only to the focal person and not to other network members) are most likely to be lost over time. It is reasonable to speculate that unsupported connections in the networks of revolving door patients may be lost for two reasons. First, relationships with these patients are often quite stormy and stressful. Unsupported connections cannot receive support from others who know of, and can empathize with, the difficulties of being involved with the patient. Without this support, the network member may become frustrated and burned out and may withdraw from the patient. Second, rehospitalization often means that the unsupported connection must look elsewhere to obtain the gratification that was received from the relationship with the patient. As his or her needs are met elsewhere, the probability decreases that the member will maintain the perseverance and initiative required to reestablish a relationship with the patient.

Just as connections between individual network members increase stability, it can be hypothesized that connections (spans) between clusters increase the likelihood that the clusters will maintain their involvement with the patient. Often the patient will temporarily withdraw from certain clusters, e.g., a work cluster. Such clusters may remain part of the patient's network by virtue of their spans to other clusters. Spans between clusters may also enable clusters to provide support for each other and prevent them from working at cross-purposes. Information not available to one cluster may be provided by another, which may result in a new perspective for dealing with the patient. Also, in times of crisis, spans may enable a network to activate all its resources quickly and to develop a unified approach to the problem.

The presence of multiplex relationships also contributes to the stability of a network. A relationship based on more than one form of interaction may signify a greater level of involvement for both the network member and the patient. This greater involvement may enable the relationship to weather stormy times or make it possible for interaction to continue even during rehospitalization.

While stability is certainly important, maximum stability would hinder flexibility. Flexibility requires that the network provide a variety of types of relationships. This is promoted by members not being overly interconnected and by the presence of relatively independent clusters. Stability is promoted by connections between members and by spans between clusters. Consequently, a balance must be reached such that members are sufficiently connected to prevent fragmentation and eventual network disintegration but not so connected that the patient is functionally interacting with only one cluster.

Combining the need for both flexibility and stability, a picture of an ideal network emerges. This network would be composed of several distinct clusters. While each cluster must be able to operate as a relatively independent unit, a small number of spans should connect the different clusters. In general, totally unconnected members or clusters not connected to any other clusters should be avoided. The network should be dominated by multiplex relationships rather than uniplex ones. Finally, the attitudes and behavior of network members should fit the model of the low EE family. Members should accept the fact that the patient has a legitimate problem, allow the patient personal space, and be supportive of his or her efforts to function as a "healthy" individual without demanding this be done at all times. In working with the revolving door patient, the clinician's task is to help transform the patient's inflexible, and frequently unstable, network into one having the preceding characteristics.

Network Intervention

A network meeting provides a critical locus for network intervention (see, for example, Speck and Attneave 1973; Garrison 1974, 1981; Rueveni 1979). Ideally, these meetings should be part of an ongoing treatment approach while having a crisis orientation. New network members are introduced, and all members are helped to discover new ways to interact with the patient and one another. Experience indicates that it is best to meet with 4 to 10 members of the patient's network at a time, rather than with the entire network. Attempting to assemble all members of a network on a regular basis has major logistical problems and may end up generating resistance and resentment among them. Also, frequent meetings of the whole network run the risk of generating too many connections between members and reducing flexibility. Network meetings with subsets of the patient's network reduce both these problems.

Increasing Flexibility

The strategy used to increase network flexibility will, in part, be determined by the ascertained source of inflexibility: the lack of sufficient supportive alternatives; the negative effect of critical, hostile, or overinvolved (high EE) members; or a combination of the two. The lack of sufficient supportive alternatives can be dealt with either by enlarging the network or increasing the degree of multiplexity in existing relationships. The negative effect of high EE members can be dealt with by reducing contact with those members or by modifying their attitudes (see table 1).

Increasing Network Size by Adding Clusters. Network size can be increased by adding individual members or clusters of members. Adding clusters is particularly useful because it provides the patient with numerous connections at one time; since these connections are also connected to one another, a certain level of stability preexists.

Clusters, as well as individual connections, can be classified as formal or informal (Beels 1981). Formal clusters exist for some reason other than the reciprocal exchange of services among members. They may have scheduled meetings and often have prescribed roles for each member. They tend to be enduring organizations. A vital characteristic is that these clusters will continue to exist with, or without, the input of a specific patient; thus they require little initiative or exchange by the patient in order to maintain membership. Formal clusters with whom the patient typically has contact include the family, the hospital, and human service agencies in the community.

Informal clusters, on the other hand, tend to lack the structure of formal clusters and depend more on the successful initiation, and receipt, of social exchange. With informal clusters, such as friends, the inability to initiate interaction, to interpret interactions adequately, or to reciprocate support may result in the dissolution of the cluster or the expulsion of that member who cannot participate in the exchange.

Given the difficulties patients have in interacting with others, it is advisable initially to attach them to preexisting formal clusters that will make few demands on them. Another advantage of formal clusters is that since they are not dependent on the patient's involvement, they will continue to exist even if the patient's membership is temporarily curtailed by rehospitalization. The cluster is still present for the patient to return to upon discharge and often welcomes the patient with open arms. Examples of such clusters, to which one might attempt to introduce a patient, are church groups, day and evening programs run by local mental health organizations, and community service organizations such as the Elks Lodge, Lions Club, YMCA, a citizens' group, or neighborhood coalition.

Introducing or reintroducing a patient to a cluster in the community requires a significant amount of work. In attempting to connect a patient to a church, it must be remembered that most pastors have had little contact with severely disturbed people. This is even more true of the congregation. The clinician must pave the way for the patient by meeting with the pastor and relevant church members and educating them about these patients' strengths, as well as their needs, on a regular basis. The church members with whom the patient has routine contact must be seen as part of the patient's network. For example, Mr. S. was referred to a weekly outpatient group that was established in a local church. Initially this group was run largely by hospital staff, but three involved church women

Table 1. Network intervention strategies and tactics

Goal	Source of problem	Strategy	Tactic
Increase flexibility	Lack of sufficient supportive alternatives	Increase size	Add: (1) Clusters (2) Individual members
		Increase multiplexity	Encourage/assist members to: (1) Provide additional types of assistance/aid (2) Take on new roles, re: patient
	Negative impact of high EE network members	Reduce contact with high EE[1] network member	(1) Enlarge patient network (2) Involve patient in daily activity (3) Post-hospitalization placement in a different environment (4) Enlarge network of high EE member
		Modify attitudes of high EE network members	(1) Educate network members re: mental illness (2) Combine high and low EE network members in group as modeling tactic
Increase stability	Members at high risk of being lost to network	Generating connections	(1) Between unconnected members (2) Between members with similar role/function
	Unconnected, vital clusters	Generating spans	(1) Between clusters with similar functions (2) Between clusters in conflict
	Low involvement of network members	Increase multiplexity	Encourage/assist members to: (1) Provide additional types of assistance/aid (2) Take on new roles, re: patient

[1] EE = expressed emotion

eventually took over the meeting. Connections between group members were encouraged by planning parties together, exchanging bus routes, and advising one another on how to deal with community life. Another patient whose network consisted primarily of his family and mental health professionals was encouraged to attend services at his former synagogue. Now he stays after services and is beginning to use this as a social outlet.

An additional method of adding a cluster to a patient's network is to move patients into the community in groups. Patients immediately have roommates or neighbors they know. This establishes a reference group that can provide peer support and set up a situation in which patients may take mutual responsibility for one another's welfare (Fairweather 1980).

Once a cluster of patients is placed in the community, the differences between formal clusters and this informal cluster may become evident. Informal contact, be it with neighbors or even roommates, may remain minimal because neither party feels comfortable generating interaction. Therefore, the clinician must, at least initially, monitor these relationships closely. This usually requires weekly or biweekly meetings with that cluster. To reduce dependency on the hospital further and to generate reintegration into the community, these meetings should take place in the homes of the patients.

Increasing Network Size by Adding Individual Members. Adding individual members to a patient's network is at times more difficult than adding a cluster. Cluster membership often requires maintaining only loose contact with several members of the cluster. Individual relationships tend to require more effort on the part of both parties since they do not have other connections supporting their relationship. Consequently, the clinician may have to monitor and intervene with new relationships even more than with new clusters.

A major source of new network members is past members who have disengaged from the patient's network. Contacting these ex-members and explaining how important they could be in helping the patient and the network, as well as offering the assistance of the treatment team in dealing with any problems that might arise, are often sufficient to get ex-members to come to an initial network meeting. The treatment team may need to do additional work to help the patient and the ex-member work out the problems that led to the disengagement.

Adding new members to a patient's network often requires that the clinician do some detective work to discover natural helpers in the community, e.g., a bartender who lends poor patrons money and helps regulate their drinking (Collins and Pancoast 1976). These natural helpers must be approached to determine if they might be willing to expand their aid to a specific patient.

Increasing Multiplexity. Another method of dealing with the inflexibility resulting from the lack of sufficient supportive alternatives is to increase the degree of multiplex relationships among current network members. Increasing multiplexity provides the patient with alternative sources of encouragement, advice, tangible assistance, and social companionship. It also tends to add to the total number of exchanges the patient might receive and reduces dependency on any one person for a specific social purpose.

The first step in increasing multiplexity is to determine whether a major deficit exists in a potential area of supportive exchange. The next step in transforming a uniplex relationship into a multiplex one is to encourage that network member to take on a new role during network meetings. For example, the friend who is solely a social companion is helped to provide advice and information by being asked to share how a job was located or a problem with a landlord solved. The friend is helped to translate those experiences into concrete steps the patient can follow. This initial work demonstrates, both to the network member and to the patient, that the member is capable of supplying various exchanges. Through "homework" assignments that call for the new roles to be practiced, these roles become generalized to interactions outside network meetings.

Initially, network members find it difficult to adjust to additions to their roles. Participants feel awkward in their new behavior and often do not experience the patient as sufficiently reinforcing. Therefore, it is vital that any attempts to fulfill homework assignments be discussed in subsequent meetings. On occasion, to make the transition easier, network members may be paired to learn from each other. A relative who generally offers support and encouragement may be paired with a friend to help the patient find housing or move into a new apartment. This way, network members may learn to work together and provide mutual support.

Reducing Negative Affect Toward the Patient. The greatest source of attitudinal inflexibility in the networks of most revolving door patients tends to be their families. In part, this may be a consequence of the fact that high EE, nonfamily network members can withdraw from the patient's network or the patient from them. It is far more difficult, however, for family members and patients to withdraw from one another.

Overinvolved foster sponsors (supervisors of community residential facilities) are often a major source of attitudinal inflexibility with this population. Most foster sponsors are experienced in working with more severely institutionalized chronic patients; they often control or are involved with almost all aspects of the patients' lives. They do most if not all the cooking, cleaning, and laundry and set rules on when patients go to bed and who they spend time with. Revolving door patients are often more active and independent than chronic patients and may resent foster sponsors' attempts to treat them like children who must be watched over constantly. Finally, neighbors, friends, coworkers, and even mental health workers may hold some of these attitudes toward the patient.

Reducing Contact. One method of dealing with the effects of attitudinal inflexibility is to reduce the patient's contact with inflexible members. This can be achieved by enlarging the patient's network. If the patient has other social spaces in which to interact, he or she will have less time and less need to interact with those who hold inflexible attitudes. Involving the patient with a daily activity, such as a day treatment program, volunteer work, or a job, will temporarily reduce contact with the family or foster sponsor. A work schedule may be established so that when the patient is asleep the parents are awake, and vice versa. If possible, when the patient is placed in the community after hospitalization, he or she should not be returned to the same critical or overinvolved environment that was home before hospitalization. For example, Mr. S.'s contact with his high EE family was reduced by helping him obtain a job, establishing a schedule for home visits, and reaching an agreement that he would not return to his mother's home to live.

Another tactic for reducing patient contact with a high EE family member is to enlarge that member's network. Parents with low rates of contact with friends and relatives, and single parents with no one else but the patient at home, are more likely to register as high EE (Leff 1976). This suggests that the networks of the families themselves are fairly impoverished, forcing them to interact only with one another to have their needs met. This may also be the case with other overinvolved network members. If the high EE member's network is made more adequate, that member may have less need to interact with the patient.

Enlarging a member's network is similar to enlarging the patient's network. Also, when selecting which former members of the network to target for reengagement, the clinician may decide to choose an individual who was also previously connected to the high EE member. Thus, both the patient's network and the member's may be enlarged at the same time.

Modifying Attitudes. Another method of dealing with inflexible attitudes is to attempt to alter them. Regardless of how often a patient has been hospitalized, most network members have little understanding of the illness, its etiology, the importance of medication, warning signs of decompensation, and those things of which the patient may or may not be capable. In early network meetings, time must be devoted to explaining the patient's "illness" and dispelling various myths about "mental illness" held by members. As network members learn more about the patient and his or her problems, they may become less intimidated by the symptoms they see. This may increase their ability to respond to the patient more appropriately.

Attitudes and/or resulting behavior may also be modified by placing high EE members in groups with low EE members. This usually involves composing groups from members of different networks. The most common group of this nature is the multiple family group, although the same principles should operate in foster sponsor groups, friends, or neighbors' groups. In all these groups, members can learn by example, seeing how others deal with problems similar to their own.

In practice, it is seldom advisable to deal with attitudinal inflexibility by either reducing contact or attempting attitude change alone. To obtain the maximum benefit, both approaches are often employed simultaneously. Similarly, since inflexibility seldom stems from only one factor, a variety of techniques for increasing network size, the number of clusters, and the degree of multiplexity—as well as for dealing with attitudinal inflexibility—are typically used at the same time. The specific components addressed should depend on appraisal of the major sources of inflexibility as well as on knowledge of the resources available. Flexibility is a cumulative process. An increase in flexibility of any of the components by definition increases overall flexibility. This, in turn, reduces the potency of any of the other inflexible components. Thus, if the clinician is aware of some strong natural helpers who could easily be added to the patient's network, this addition should probably be attempted first, even though the major sources of inflexibility may be a lack of independent clusters or attitudinal inflexibility.

Increasing Stability

Increasing the flexibility of a patient's network is often only part of the work required. There must be some certainty that the network's positive aspects will endure. The key components creating network stability are connections between individual members, spans between clusters, and a high percentage of multiplex relationships (see table 1). Since methods of increasing multiplexity have already been discussed, they are not reiterated here.

Generating Connections and Spans. The first task is to target which connections and spans should be generated. Since unconnected members (those connected only to the patient) are at high risk of being lost by the network, connecting them to others is a high priority. Such individuals may be integrated into the network by connecting them with another network member who serves a similar role with the patient. Connections between two friends or two coworkers are encouraged, because such people are more likely to have something in common than a friend and a coworker.

Similarly, spans should be generated between clusters that perform similar functions. Thus, spans should be generated between all mental health clusters so that services are not duplicated or undermined. Clusters that the patient may play off each other, e.g., the treatment staff and the family, should have a couple of spans to reduce this process. Any clusters that are in conflict, by virtue of the conflicting demands they place on the patient, should also be connected. It must be remembered, however, that the various clusters' independence must also be preserved to provide flexibility. For this reason, no more than two or three spans between clusters should purposely be generated.

The network meeting is the major forum for beginning to develop the desired connections and spans. Here individual members or representatives of different clusters may meet each other for the first time. The clinician can facilitate interaction by helping to resolve differences, encouraging future interaction, and helping participants discover common interests. For example, Mr. W.'s network consisted primarily of his foster sponsor, employees at a local topless club, and hospital staff. The foster sponsor disapproved of Mr. W.'s going to the club and attempted to limit his visits. This agitated the patient, and he would return to the hospital rather than face arguments at home. A network meeting was convened with the manager of the club, the foster sponsor, Mr. W., and the treatment team. During the meeting, specific times were mutually agreed upon for Mr. W. to attend the club, and both relationships were maintained. Later, when Mr. W. was in the new foster home, network meetings included both old and new foster sponsors.

To encourage further interaction, the clinician never takes full responsibility for arranging subsequent network meetings. Connections are stimulated by having several members contact other members or other clusters. During the network meeting, future contact is arranged by establishing that certain members will work together to help the patient deal with a specific problem. Connections are also encouraged by the treatment team's referring a network member or cluster back to other members or clusters. Early in the network process, network members are very dependent on the treatment staff. They call for assistance with any problem that arises. The treatment team facilitates connections by directing members to other members who may be of assistance. For example, to generate spans between the family and other clusters, a parent may be directed to the leader of the patient's church group and a representative of a community agency for help on enlisting the aid of these clusters in finding housing for the patient. As the connections and spans become stronger, individual members and different clusters will turn to one another more and more, and the treatment team can begin to minimize its involvement.

While increasing stability is often easier than increasing flexibility, it is no less important. Most revolving door patients, particularly early in the networking process, will be rehospitalized periodically. To have their networks decimated each time this occurs, or each time there is network conflict, results in needless suffering for both the patient and the network.

Summary

In the last 10 years it has become evident that the traditional modes of psychiatric treatment are not effective in dealing with revolving door patients. Rehospitalization rates remain high, and many patients who stay in the community continue to lead a marginal existence.

A variety of findings suggest that the focus of intervention should be on the patient's social network, particularly its flexibility and stability. It is important that a patient's network have the flexibility to provide support regardless of the patient's behavior or the social identity being attempted, and regardless of any conflict that may exist within the network. Furthermore, the network must endure over time, i.e., remain stable.

Networking, however, will not solve all the problems that exist for the revolving door patient. Patients may still require occasional short hospitalizations, as do Mr. S. and Mr. W., although the duration and frequency of these hospitalizations may be greatly reduced. In addition, treatment strategies for common deficiencies in social and work skills, as well as for substance abuse, must be specifically designed and incorporated into comprehensive treatment plans. Experience has demonstrated, however, that merely addressing these treatment issues is not sufficient to enable such patients to succeed in the community. The clinician's major focus must turn toward the patients' networks. Careful assessment of the networks and appropriate interventions that increase flexibility and stability promise to help slow and reduce the revolving door pattern.

References

Beels, C.C. Social support and schizophrenia. *Schizophrenia Bulletin*, 7:58-72, 1981.

Brown, G.W.; Birley, J.L.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, 121:241-268, 1972.

Budson, R., and Jolley, R. A crucial factor in community program success: The extended psychosocial kinship system. *Schizophrenia Bulletin*, 4:609-621, 1978.

Caton, C. The new chronic patient and the system of community care. *Hospital & Community Psychiatry*, 32:475-478, 1981.

Cohen, C., and Sokolovsky, J. Schizophrenia and social networks: Ex-patients in the inner city. *Schizophrenia Bulletin*, 4:546-560, 1978.

Collins, A., and Pancoast, D. *Natural Helping Networks: A Strategy for Prevention*. Washington, DC: National Association of Social Workers, 1976.

Dellario, D.J., and Anthony, W.A. On the relative effectiveness of institutional and alternative placement for the psychiatrically disabled. *Journal of Social Issues*, 37:21-33, 1981.

Fairweather, G. The development of group process in treatment with mental patients. In: Fairweather, G., ed. *The Fairweather Lodge: A Twenty-year Retrospective: New Directions for Mental Health Services*. San Francisco: Jossey-Bass, 1980. pp. 3-12.

Fenton, F.R.; Tessier, L.; and Struening, E.L. A comparative trial of home and hospital psychiatric care: One year follow-up. *Archives of General Psychiatry*, 36:1073-1079, 1979.

Garrison, J. Network techniques: Case studies in the screening-linking-planning conference method. *Family Process*, 13:337-353, 1974.

Garrison, J. Clinical construction of action social networks. *International Journal of Family Therapy*, 3:258-267, 1981.

Greenwood, V. "Cognitive Therapy With the Young Adult Chronic Patient." Paper presented at the Association for Advancement of Behavior Therapy annual convention, Toronto, November 1981.

Hammer, M. Influence of small social networks as factors on mental hospital admission. *Human Organization*, 22:243-251, 1963-1964.

Hammer, M. Predictability of social connections over time. *Social Networks*, 2:165-180, 1980.

Hammer, M. Social supports, social networks, and schizophrenia. *Schizophrenia Bulletin*, 1:45-57, 1981.

Harris, M., and Bergman, H. "Community Connections: Theoretical Rationale and Practical Implications." Unpublished manuscript, St. Elizabeths Hospital, Washington, DC, 1981.

Hirsch, B. Psychological dimensions of social networks: A multimethod analysis. *American Journal of Community Psychology*, 7:263-277, 1979.

Hirsch, B. Natural support systems and coping with major life changes. *American Journal of Community Psychology*, 8:159-172, 1980.

Hirsch, B. Coping and adaptation in high-risk populations: Toward an integrative model. *Schizophrenia Bulletin*, 7:164-172, 1981.

Leff, J. Schizophrenia and sensitivity to the family environment. *Schizophrenia Bulletin*, 4:566-574, 1976.

Liberman, R.; Wallace, C.; Vaughn, C.; and Snyder, K. "Social and Family Factors in the Course of Schizophrenia: Towards an Interpersonal Problem-solving Therapy for Schizophrenics and Their Families." Paper presented at the Conference on Psychotherapy of Schizophrenia, Yale University, New Haven, CT, April 1979.

Lipton, F.R.; Cohen, C.; Fischer, E.; and Katz, S. Schizophrenia: A network crisis. *Schizophrenia Bulletin*, 7:144-151, 1981.

Pattison, E.M.; deFrancisco, D.; Wood, P.; Frazier, H.; and Crowder, J. A psychosocial kinship model for family therapy. *American Journal of Psychiatry*, 132:1246-1251, 1975.

Pepper, B.; Kirshner, M.; and Ryglewicz, H. The young adult chronic patient: Overview of a population. *Hospital & Community Psychiatry*, 32:463-499, 1981.

Rueveni, U. *Networking Families in Crisis*. New York: Eastern Pennsylvania Psychiatric Institute, 1979.

Salem, D.; Seidman, E.; and Rappaport, J. "Deinstitutionalization of the Mentally Ill: The Promise of Mutual Help Organizations." Unpublished manuscript, 1984.

Speck, R.V., and Attneave, C.L. *Family Network*. New York: Pantheon Books, 1973.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: III. Five-year outcome and its predictors. *Archives of General Psychiatry*, 34:159-163, 1977.

Test, M.A. Effective community treatment of the chronically mentally ill: What is necessary? *Journal of Social Issues*, 37:71–86, 1981.

Test, M.A., and Stein, L.I. Community treatment of the chronic patient: Research overview. *Schizophrenia Bulletin*, 4:350–364, 1978.

Tolsdorf, C.C. Social networks, support and coping: An exploratory study. *Family Process*, 15:407–418, 1976.

Vaughn, C.E., and Leff, J.P. The influence of family and social factors on the course of psychiatric illness. A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129:125–137, 1976.

Vaughn, C.E., and Leff, J.P. Patterns of emotional response in relatives of schizophrenic patients. *Schizophrenia Bulletin*, 7:43–44, 1981.

Watzlawick, P.; Beavin, J.H.; and Jackson, D.D. *Pragmatics of Human Communication*. New York: W.W. Norton & Company, 1967.

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Announcement

The **Family Support Group of the Northeast Ohio Chapter of Alliance for the Mentally Ill** is compiling a resource list of all facilities that are available to and provide services for those afflicted with schizophrenia. All information, inquiries, and cooperation are solicited. Please contact:

M. Jaffe
2447 Edgerton Road
Cleveland, OH 44118

A Holistic Program for Chronic Schizophrenic Patients

by David Lukoff, Charles J. Wallace, Robert P. Liberman, and Karen Burke

Abstract

A 10-week, inhospital holistic health program for male schizophrenic patients was compared with an equally intense social skills training program. The holistic program included training in the stress reduction techniques of exercise and meditation as well as education in stress management. Patients were also encouraged to explore the growth potential of their psychotic experiences and to develop positive beliefs about the outcome of their illness. Both groups showed similar significant decreases in psychopathology from admission to discharge, but the use of medication and a token economy milieu by all patients confounds the interpretability of this finding. After the holistic patients were discharged into the community, there was no maintenance of any of the holistic techniques. The 2-year relapse rate did not differ significantly between the two treatments. Findings from various studies associating schizophrenic relapse with stressful life events and familial tension make further experimentation with stress reduction techniques for the treatment of schizophrenia worthwhile.

Stress reduction and other holistic techniques have received minimal trial or attention in the treatment of schizophrenia. Yet findings that the onset of schizophrenic episodes is associated with stressful life events (Brown and Birley 1968) and familial tension (Vaughn et al. 1984; see Spring and Coons 1982 and Lukoff et al. 1984 for reviews) suggest that stress reduction interventions may be effective in the treatment of schizophrenic patients.

The holistic health perspective views stress as a major, nonspecific causal factor in the genesis of all diseases. Holistic treatments emphasize stress reduction techniques, including meditation and physical exercise. There is suggestive evidence that stress reduction techniques are effective with psychiatric disorders. In studies with diagnostically mixed inpatients, physical exercise has been found to have a positive effect on mood and anxiety (Dodson and Mullens 1969). Griest et al. (1979) found that depressed outpatients treated with sessions of jogging showed reductions in depression equal to those of the medication-treated patients. Glueck and Stroebel (1975) reported that a diagnostically mixed group of psychiatric inpatients trained in meditation showed significantly greater global improvement ratings at discharge than a group of matched controls.

Unfortunately, the few studies of stress reduction techniques with schizophrenic patients have been characterized by diagnostic imprecision and poor specificity of procedures and outcomes. This is the first report of a controlled trial of jogging, meditation, visualization, and other holistic techniques with a sample of carefully diagnosed schizophrenic patients.

In addition to reducing stress, a key issue in holistic treatment is "mobilizing an individual's volition" (Pelletier 1977, p. 28). Visualization techniques, first developed for treating cancer patients (Simonton, Matthews-Simonton, and Creighton 1978), are used in holistic programs to increase positive expectations of recovery from illness. The treatment techniques developed at Diabysis (Perry 1974) and Soteria (Mosher and Menn 1979) projects for

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psychotic patients are also designed to increase positive attitudes toward illness. They used expressive art therapy techniques to help patients see the growth-promoting dimensions of their psychotic episodes.

The opportunity to develop a holistic program arose during a study being conducted at the UCLA-Camarillo Hospital Clinical Research Unit on the effectiveness of an inhospital social skills training and behavioral family therapy program for high-risk male schizophrenic patients. The senior author was given the responsibility for developing a treatment program based on holistic principles for the comparison group.

Methods

Subjects. Twenty-eight male patients who met CATEGO class S and *DSM-III* criteria (American Psychiatric Association 1980) for schizophrenia elicited by the Present State Examination (PSE) (Wing, Cooper, and Sartorius 1974) were recruited for participation in the study from inpatient units at UCLA, Camarillo State, and Ventura County hospitals. They were admitted in cohorts of six, and the program was repeated five times. During the first administration of the program, two patients dropped out of a nonmedication social skills training condition, which was dropped from the subsequent four programs.

Measures. Patients were evaluated pre- and posttreatment with a multilevel battery that included a patient self-report measure: Symptom Checklist-90 (SCL-90) (Derogatis et al. 1974); a psychiatric interviewer rating scale: Psychiatric Assessment Scale (PAS) (Krawiecka, Goldberg, and Vaughn 1977); and a nursing staff rating: Nurses Global Impressions Scale (NGI) (Guy 1976). The Tennessee Self-Concept Test (TSC) (Fitts 1972) was also administered pre- and posttreatment.

Once discharged into the community, patients were followed closely by the project's social worker to determine compliance with medication and to track any relapses. Relapses were operationally defined based on the PSE and the PAS. Agreement on blind ratings of relapse between two raters, both psychiatrists, was 100 percent.

The following measures, which assessed areas targeted for intervention by the holistic program, were administered only to the holistic patients: the Cooper (1977) 12-minute aerobic fitness test, which correlates .90 with maximum oxygen consumption, generally considered the best overall measure of aerobic fitness; blood pressure before and after a 20-minute meditation session, which assessed changes in the ability to induce a relaxed state; and six-item true/false quizzes, which tested the patient's comprehension of the stress management education sessions.

Procedures. Patients were randomly assigned to the social skills and holistic treatments, which were conducted simultaneously. Patients were housed on the Camarillo-UCLA Clinical Research Unit during the study. All patients also participated in the unit's token economy for the duration of the program. During the first week, baseline assessments were obtained. The treatment phase of the program lasted 9 weeks. To control for the amount of time spent in therapy, all treatment sessions were scheduled concurrently. To control for therapist personality effects, therapists rotated on a daily basis between both treatments except for the evening sessions, which were conducted by the same therapist throughout the 9-week program. Therapists included two doctoral level psychologists, two master's level psychologists, a recreation therapist, and predoctoral psychology interns.

Social Skills Training. In the daily morning sessions, the social skills patients participated in role-playing exercises that systematically covered 250 standardized interpersonal problem situations. In the twice-weekly afternoon generalization sessions, the same role plays rehearsed in the morning were replayed in a different setting and with a different partner. Once each week, independent living skills were covered. A second weekly group covered the principles of assertive behavior, anger control, and problem solving.

The social skills patients and their relatives met in weekly behavioral family therapy sessions that were designed to train the entire family in communication and problem-solving skills and to provide information regarding the role of medication in preventing relapses. Detailed descriptions of the behavioral family therapy program (Liberman, Falloon, and Aitchison 1984) and the social skills training program (Wallace 1982) have been published elsewhere.

Holistic Health Sessions. Each weekday morning, the patients participated in 30 minutes of exercise, which consisted of either a 1.8-mile walk/run around the hospital grounds or sports activities in the hospital gym. Each exercise session was preceded by 15 minutes and followed by 5 minutes of yoga. The yoga was included to prevent the occurrence of torn or sore muscles during the exercise.

The meditation sessions used

Carrington's (1978) clinically standardized meditation (CSM) technique, which is similar to transcendental meditation (TM). Each patient chose his mantra from a list of 16 Sanskrit mantras. Patients were gradually introduced into the meditation by having them close their eyes and listen to soothing music (*Spectrum Suite* by Stephen Halpern 1977). Starting in the third week, daily meditation sessions lasting 20 minutes were held each weekday morning.

The twice-weekly stress education sessions began with 10 to 15 minute audiotapes that presented the rationale for using stress reduction techniques. At each session, an accompanying handout, with simplified diagrams and graphs illustrating the key concepts of the day's topic, was used to stimulate discussion among the patients. Most sessions were followed by a six-item true/false quiz and an art therapy activity based on the topic.

In the weekly "Mobilizing Positive Beliefs" sessions, simplified and anthropomorphized diagrams were used to illustrate the state of harmony and healing in the brain (figure 1). The image of the right and left hemispheres cooperating and communicating with each other was meant to be equivalent to the image of white blood cells overpowering cancer cells used in the Simontons' program to influence their patients' beliefs in a positive direction. The holistic patients used the image in figure 1 and created similar ones for visualization and art therapy sessions. These sessions were intended to increase the patients' expectations of recovering and maintaining their well-being.

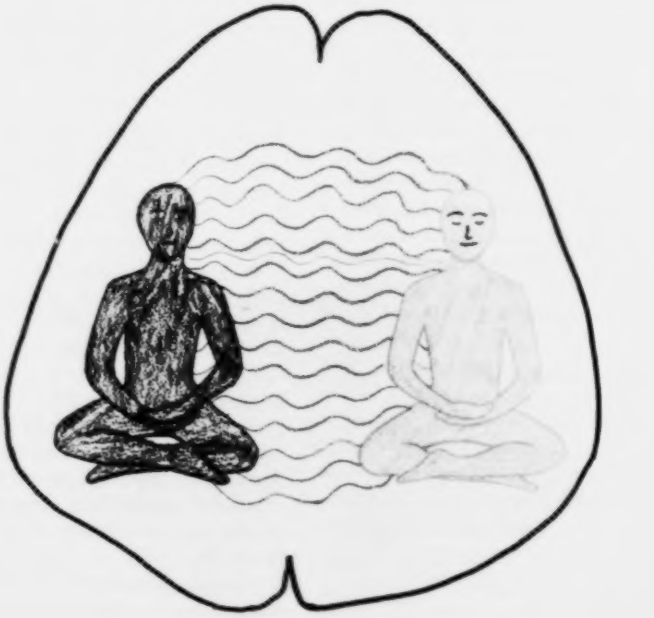
In weekly "Growth and Schizophrenia" sessions, patients were encouraged to look at the positive dimensions of their psychotic experiences. The sessions exposed them to accounts of people who made significant contributions to society, religion, and art after their psychotic episodes and presented the similarities between psychotic episodes and Native American vision quests, mystical experiences, and shamanic rituals (Lukoff and Everest 1985). Selections from popular rock music were played to promote sharing of similar experiences by the patients and to encourage acceptance of their psychotic experiences, e.g., "Acute Paranoia Schizophrenia Blues" by the group SuperTramp. Art therapy techniques as well as written exercises were used to structure the expression and reframing of the patients' psychotic experiences.

Exercises for the weekly "Learning to Love Yourself" sessions were drawn from books on building self-esteem among students (Wells and Canfield 1976). The sessions emphasized the importance of maintaining a positive self-concept in order to cope with stress.

The weekly family therapy sessions were dynamically oriented. The families were not introduced to the holistic health philosophy or trained in any stress reduction techniques.

Medication. All patients were provided optimal doses of neuroleptic medications, usually chlorpromazine and thioridazine, based on their individual histories with previous medications and their side effects. Doses varied from 200 to 600 mg per day chlorpromazine equivalent, with a mean dose of 350 mg, and did not differ between the two treatment groups.

Figure 1. Image used to illustrate harmony between the two sides of the brain.



Aftercare and Followup. Placement and aftercare were coordinated by the social worker involved in the study. She maintained regular contact with the patients starting at five times per week for the first 2 weeks but then fading to two times per week by the 8th week, and to twice a month from the 9th to the 24th month.

Results

Pre- and Posttreatment Effects. First, analysis of covariance was used to determine whether there were differential treatment effects between the holistic and social skills groups on the psychopathology measures. Second, correlated *t* tests were conducted to ascertain if there were any significant changes in either group from pre- to posttreatment.

Psychopathology. None of the summary scores or individual subscales showed significant pre- to posttreatment differences between the two groups. The pre-post change differences, however, show that the holistic program's delta scores were larger for 7 of the 8 PAS variables, 9 of the 12 SCL-90 variables, the NGI severity variable, and the self-concept total P score (table 1).

The results from the correlated *t* tests showed that there were significant reductions in overall psychopathology in both the holistic and social skills groups on the PAS Total, NGI Severity, and SCL-90 General Symptom Index scores.

Self-Concept. The holistic group's mean Total P score rose from the 6th to the 19th percentile, while that of the social skills group increased from the 28th to the 30th percentile. Because the self-concept data lacked homogeneity of regression, the Johnson-Neuyman technique (Kerlinger and Pedhazer 1973) was applied. The region of nonsignificance encompassed virtually the entire spectrum of possible scores on this measure, indicating no difference between the holistic and social skills treatment in self-concept (table 1). The correlated *t* test showed that neither the holistic group ($t = 1.42$, ns) nor the social skills group ($t = .16$, ns) improved significantly in self-concept.

Relapse. The chi-square test was used to determine whether there was a difference in the incidence of relapses between the two treatments. There were 11 relapses among the 14 holistic patients and 7 relapses among the 14 social skills patients during the 2-year followup. This was not a significant difference at either the .05 or .10 levels ($x^2 = 2.48$, $df = 1$). Number of weeks on medication during the 104-week followup period did not differ between the two groups: social skills: 80.1 weeks; holistic: 64.7 weeks ($t = .50$, ns).

Physical Fitness. At pretest on the Cooper (1977) 12-minute aerobics fitness test, the 11 patients for whom there are data averaged 1.04 miles. At posttest, the patients improved significantly to 1.21 miles, an increase of 16.7 percent ($t = 2.75$, $df = 10$, $p < .05$). Eight of the 11 patients improved their performance.

Meditation. The mean change in systolic blood pressure during the relaxation session at pretest was an increase of .4 mm Hg. At posttest, the 10 patients for whom there are data showed a .3 mm Hg decrease in systolic readings. Given that the systolic pressure averaged 111.4 mm Hg, this was a nonsignificant change clinically as well as statistically ($t = .63$, $df = 9$, ns). The change in diastolic pressure was also nonsignificant ($t = .09$).

Stress Management Education. To check whether the material was being comprehended by the patients, six-item true/false quizzes were administered following many of the sessions. The mean score on the quizzes was 5.36, which—based on the sign test—was significantly beyond chance ($p < .001$).

Stress Reduction in the Community. During the followup phase, the project's social worker inquired about the holistic patients' utilization of the stress reduction techniques. One patient in the first program and one in the second program continued exercising, but only for the first 2 to 3 months after discharge. Three patients reported using meditation occasionally during the 2-year followup.

Discussion

While there were no significant differences between the outcomes of the holistic and social skills patients, both groups showed substantial and significant reductions in psychopathology over the course of the program. The reductions in psychopathology, however, cannot be attributed solely to the social skills and holistic treatments, since all patients also received medication and participated in a behavior modification token economy during the study.

Despite the lack of significant differences on the psychopathology and self-concept measures, the pattern of differences across the measures seemed to favor the holistic program. Given that these two treatments overlapped considerably and

Table 1. Pre- and posttreatment evaluations with the Symptom Checklist-90, Psychiatric Assessment Scale, Nurses Global Impressions Scale, and the Tennessee Self-Concept Test

Scales	Group (*n*)	Pre mean (SD)	Post mean (SD)	△	Within-group correlated *t*-test	Between-group ANOVA adjusted	
						Post mean	*F*
Symptom Checklist-90 Derogatis et al. (1974)							
Somatization	Holistic (12)	.96 (.55)	.48 (.43)	.48	2.51^{2}	.47	< 1
	Social skills (14)	.76 (.55)	.41 (.39)	.35	4.41^{3}	.45	
Obsessive-compulsive	Holistic (12)	1.39 (.66)	.70 (.81)	.69	3.34^{3}	.58	1.77
	Social skills (14)	1.18 (.77)	.85 (.81)	.33	2.12^{1}	.92	
Interpersonal sensitivity	Holistic (12)	1.09 (.74)	.56 (.51)	.53	2.56^{2}	.53	3.31^{1}
	Social skills (14)	1.10 (.63)	1.01 (.88)	.09	.52	1.00	
Depression	Holistic (12)	1.38 (.86)	.79 (.73)	.59	2.06^{1}	.85	< 1
	Social skills (14)	1.53 (.69)	1.16 (.90)	.37	1.73	1.13	
Anxiety	Holistic (12)	1.08 (.54)	.62 (.49)	.46	3.19^{3}	.63	< 1
	Social skills (14)	1.19 (.81)	.66 (.63)	.53	3.46^{3}	.63	
Hostility	Holistic (12)	.65 (.57)	.47 (.50)	.18	1.13	.45	< 1
	Social skills (14)	.69 (.70)	.48 (.53)	.21	1.36	.47	
Phobic anxiety	Holistic (12)	.83 (.57)	.41 (.43)	.42	2.10^{1}	.41	1.33
	Social skills (14)	.84 (.76)	.68 (.81)	.16	.80	.68	
Paranoid ideation	Holistic (12)	.74 (.71)	.41 (.43)	.33	1.40	.81	< 1
	Social skills (14)	1.26 (.74)	.88 (.76)	.38	2.16^{2}	.85	
Psychoticism	Holistic (12)	1.01 (.69)	.56 (.68)	.45	1.58	.62	< 1
	Social skills (14)	1.24 (.78)	.82 (.82)	.43	1.80^{1}	.78	
Positive symptom total	Holistic (12)	51.92 (21.82)	36.08 (26.74)	15.84	2.11^{1}	34.11	< 1
	Social skills (14)	48.36 (23.65)	38.79 (24.75)	9.57	1.57	39.69	
General symptom index	Holistic (12)	1.08 (.54)	.61 (.51)	.47	2.62^{2}	.64	< 1
	Social skills (14)	1.21 (.71)	.81 (.66)	.40	2.52^{2}	.79	
Positive symptom distress index	Holistic (12)	1.82 (.46)	1.47 (.40)	.35	2.33^{2}	1.50	1.64
	Social skills (14)	1.85 (.36)	1.73 (.50)	.12	.98	1.72	
Psychiatric Assessment Scale Krawiecka, Goldberg, and Vaughn (1977)							
Anxiety	Holistic (14)	2.00 (.78)	1.36 (1.01)	.64	1.88^{1}	1.27	
	Social skills (14)	1.29 (.99)	1.29 (.91)	.00	0.0	1.37	< 1
Depression	Holistic (14)	.86 (1.03)	.43 (.65)	.43	1.25	.51	
	Social skills (14)	1.50 (.94)	.93 (1.14)	.57	2.10^{1}	.85	< 1
Retardation	Holistic (14)	.71 (1.07)	.14 (.36)	.57	2.51^{2}	.21	3.64^{3}
	Social skills (14)	1.07 (1.26)	.71 (.99)	.36	1.44	.64	
Delusions	Holistic (14)	2.86 (1.65)	1.57 (1.91)	1.29	2.30^{2}	1.53	
	Social skills (14)	2.64 (1.50)	1.50 (1.87)	1.14	2.28^{2}	1.55	< 1

Table 1. Pre- and posttreatment evaluations with the Symptom Checklist-90, Psychiatric Assessment Scale, Nurses Global Impressions Scale, and the Tennessee Self-Concept Test—*Continued*

Scales	Group (*n*)	Pre mean (SD)	Post mean (SD)	Δ	Within-group correlated *t*-test	Between-group ANOVA adjusted Post mean	Between-group ANOVA adjusted *F*
Hallucinations	Holistic (14)	1.71 (1.82)	1.21 (1.72)	.50	1.24	1.30	
	Social skills (14)	2.00 (1.84)	1.79 (1.67)	.21	.59	1.70	< 1
Incoherence	Holistic (14)	1.50 (1.51)	.57 (.93)	.93	2.25[2]	.54	
	Social skills (14)	1.21 (1.05)	.29 (.83)	.92	3.79[3]	.32	< 1
Flat affect	Holistic (14)	1.86 (.86)	1.07 (.92)	.79	2.80[2]	1.07	< 1
	Social skills (14)	1.86 (.86)	1.36 (.84)	.50	1.99[3]	1.36	
Poverty of speech	Holistic (14)	1.64 (1.15)	.71 (.91)	.93	3.04[3]	.71	
	Social skills (14)	1.57 (1.02)	.71 (.91)	.86	2.37[2]	.72	< 1
Total	Holistic (14)	13.36 (5.50)	7.07 (5.48)	6.29	3.71[3]	7.00	
	Social skills (14)	13.14 (5.11)	8.57 (6.36)	4.57	4.28[3]	8.64	< 1
Nurses Global Impressions Scale Guy (1976)							
Severity	Holistic (14)	4.07 (1.14)	2.79 (1.37)	1.28	3.80[3]	2.60	< 1
	Social skills (14)	3.50 (1.22)	2.79 (1.19)	.71	2.92[2]	2.97	
Improvement	Holistic (14)		3.57 (1.34)		.93[4]		
	Social skills (14)		3.14 (1.10)				
Tennessee Self-Concept Test Fitts (1972)							
Total P	Holistic (13)	296.15 (36.66)	317.08 (38.37)	20.93		Region of nonsignificance	
	Social skills (14)	331.00 (54.70)	332.86 (75.23)	1.86		188.9–549.6	

Note:—On the SCL-90, PAS, and NGI items, lower scores reflect lesser severity. On the TSC, higher scores reflect increased self-concept.

[1] $p < .10$.

[2] $p < .05$.

[3] $p < .01$.

[4] Between-group *t*-test.

had small *n*'s, the finding that 18 of 22 delta scores were larger in the holistic group suggests that, during the inpatient phase, the holistic program may have had a more positive overall impact.

While the number of relapses in the holistic and social skills groups were not significantly different, the 50 percent relapse rate in the social skills group compared with the 79 percent relapse rate in the holistic group may indicate an important difference. The social skills group appeared to fare better during the followup phase. Since the holistic patients did not continue practicing the stress reduction techniques in the community, their relapses cannot be attributed to the ineffectiveness of holistic techniques. These results suggest, however, that greater attention must be paid to ensuring generalization of holistic treatments in future studies of stress reduction techniques for the prevention of relapses in schizophrenia.

During the inpatient phase, there were no dropouts in the holistic group and, with one exception, the holistic patients participated actively. Previous studies of exercise (Kaplan, Mendelson, and Dubroff 1983) and relaxation techniques (Glueck and Stroebel 1975) with inpatient psychiatric populations have encountered severe problems with compliance and dropouts. In this study, the patients' compliance with the holistic regimen was probably due to combining the holistic techniques with contingency management techniques. Participation in the holistic "behaviors" was reinforced as part of the token economy. Thyer, Irvine, and Santa (1984) demonstrated that contingency management techniques can also be used in the community to increase the participation of schizophrenic patients in stress reduction activities.

While there were significant benefits to implementing a holistic program on a behavioral unit, especially in the area of compliance, the conditions for supporting a holistic treatment program were not ideal. For example, the reward system of the token economy may have sabotaged some of the treatment goals of the holistic program. Sessions on nutrition were conducted while patients munched on candy bars, drank coffee, and smoked cigarettes they received as reinforcers. White bread, canned vegetables, and chopped meat were the mainstays of the hospital food. This diet and the lack of supplemental vitamins are not considered optimal from the holistic perspective.

While the therapists conducted the holistic sessions efficiently and did not make disparaging remarks in front of the patients, only 2 of the more than 20 behaviorally oriented therapy and nursing staff members were knowledgeable about, or followers of, holistic treatment approaches. It seems likely that the treatment's impact was negatively influenced by the patients' awareness that the holistic techniques were not generally practiced by their therapists or nursing staff. In addition, the staff's policy of ignoring all discussion of hallucinations and delusions may have undermined the holistic treatment goal of facilitating acceptance and positive attitudes toward psychotic experiences.

Since this is the first report of a holistic program with schizophrenic patients, the findings regarding the use of controversial holistic treatments, e.g., jogging, meditating, and promoting a positive attitude toward the psychotic experience, are discussed in more detail.

Exercise. The results of this study showed that the schizophrenic patients participated in and improved their level of fitness through a structured exercise program. The trend on the PAS Motor Retardation item in favor of the holistic group may have been related to their participation in the exercise program.

Meditation. The holistic patients did not show any change in their ability to lower their blood pressure despite the meditation training. Change in blood pressure, however, has been found to be an insensitive measure of improvement in relaxation skills among normotensive subjects (Frumkin et al. 1978).

The use of meditation with schizophrenic patients has not been seriously investigated before. Anecdotal accounts of patients with undisclosed diagnoses and premorbid histories have reported severe depersonalization (Lazarus 1976) and "complete mental breakdown" (Carrington 1978) precipitated by meditation. These findings may have created concern among mental health professionals about the use of meditation with schizophrenic patients.

The schizophrenic patients in this holistic program and the patients in the only other study using meditation with this population (Glueck and Stroebel 1975) did not show distress or exacerbation of symptoms during or immediately after meditating. Therefore, further studies with meditation and other techniques that induce lower autonomic arousal would seem to be without significant risk, and might be particularly valuable for subgroups of schizophrenic patients prone to overarousal (Gruzelier and Venables 1975).

Growth and Schizophrenia Sessions. Some mental health professionals might have predicted that the positively oriented discussions of psychotic experiences with patients would contribute to their delusional systems. For example, Nydegger (1972) considers delusions and hallucinations to be types of behavior that are maintained by social attention. Although the "Growth and Schizophrenia" sessions provided social attention for the expression of hallucinatory experiences and delusional material, the holistic patients showed significant pre-post reductions on the PAS Hallucinations and Delusions scales. There were no significant differences on the psychotic symptoms variables between the social skills patients, whose discussions of hallucinations and delusions were put on extinction, and the holistic patients.

While the therapeutic benefits of expressing and exploring psychotic experiences were not established by this study, these results should alleviate the concern that structured, thoughtful exploration of psychotic experiences with patients will lead to exacerbation or persistence of symptoms.

Conclusion

The results from this study demonstrate that schizophrenic patients can be motivated to participate in an intensive regimen of stress reduction and attitude change activities, that holistic techniques in conjunction with medication and contingency management can produce positive outcomes, and that holistic programs can be implemented in conventional medical settings. For a maiden voyage, the holistic program performed well in comparison with a more established social skills training treatment. Not only did the holistic approach not prove harmful (as the literature might have predicted), but, during the inpatient phase, it actually appeared more helpful than the highly focused social skills treatment. The lack of followthrough and the high incidence of relapses among the holistic patients, however, indicate that future studies with holistic techniques should include more community-based programming.

Clinicians wanting to develop a holistic treatment program for schizophrenic patients need not limit themselves to the narrowly defined holistic interventions used in this study. In fact, the results from this study suggest that holistic techniques are best used in conjunction with other methods. Depending on a particular patient's needs, appropriate treatment might also include medication and social skills training.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Brown, G., and Birley, J. Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behavior,* 9:203–214, 1968.

Carrington, P. *Freedom in Meditation.* Garden City, NY: Anchor Press, 1978.

Cooper, K. *The Aerobics Way.* New York: M. Evans and Co., 1977.

Derogatis, L.; Lipman, R.; Rickels, K.; Uhlenhuth, E.; and Covi, L. The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behavioral Science,* 19:1–15, 1974.

Dodson, L., and Mullens, W. Some effects of jogging on psychiatric hospital patients. *American Corrective Therapy Journal,* 23:130–134, 1969.

Fitts, W. *The Self Concept and Psychopathology.* Dede Wallace Center Monograph IV. Nashville, TN: Counselor Recordings and Tests, 1972.

Frumkin, K.; Nathan, R.; Prout, M.; and Cohen, M. Nonpharmacologic control of essential hypertension in man: A critical review of the literature. *Psychosomatic Medicine,* 40:294–320, 1978.

Glueck, B., and Stroebel, C. Biofeedback and meditation in the treatment of psychiatric illness. *Comprehensive Psychiatry,* 16:303–321, 1975.

Griest, J.; Klein, M.; Eischens, R.; Faus, J.; Gurman, A.; and Morgan, W. Running as treatment for depression. *Comprehensive Psychiatry,* 20:41–54, 1979.

Gruzelier, J., and Venables, P. Evidence of high and low levels of physiological arousal in schizophrenics. *Psychophysiology,* 12:55–73, 1975.

Guy, W. *ECDEU Assessment Manual for Psychopharmacology: Nurses' Global Impressions Scale.* NIMH Publication No. (ADM) 76-338. Washington, DC: Superintendent of Documents, U.S. Government Printing Office, 1976.

Halpern, S. *Spectrum Suite.* Palo Alto, CA: Spectrum Research Institute, 1977.

Kaplan, K.; Mendelson, L.; and Dubroff, M. The effect of a jogging program on psychiatric inpatients with symptoms of depression. *Occupational Therapy Journal of Research,* 3:173–175, 1983.

Kerlinger, F., and Pedhazur, L. *Multiple Regression and Behavioral Research.* New York: Holt, Rinehart, and Winston, 1973.

Krawiecka, M.; Goldberg, D.; and Vaughn, M. A standardized psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica,* 55:299–308, 1977.

Lazarus, A. Psychiatric problems precipitated by transcendental meditation. *Psychological Reports,* 39:601–602, 1976.

Liberman, R.; Falloon, I.; and Aitchison, R. Multiple family therapy for schizophrenia: A behavioral, problem-solving approach. *Journal of Psychosocial Rehabilitation,* 4:60–77, 1984.

Lukoff, D., and Everest, H. The myths in mental illness. *Journal of Transpersonal Psychology,* 17:123–154, 1985.

Lukoff, D.; Snyder, K.; Ventura, J.; and Nuechterlein, K. Life events, familial stress, and coping in the developmental course of schizophrenia. *Schizophrenia Bulletin,* 10:258–292, 1984.

Mosher, L., and Menn, A. Soteria: An alternative to hospitalization for schizophrenia. In: Lamb, H., ed. *Alternatives to Acute Hospitalization.* San Francisco: Jossey-Bass, 1979. pp. 73–84.

Nydegger, T. The elimination of hallucinatory and delusional behavior by verbal conditioning and assertive behavior: A case study. *Journal of Behavior Therapy and Experimental Research,* 3:225–227, 1972.

Pelletier, K. *Mind as Healer, Mind as*

Slayer. New York: Dell Publishing Co., 1977.

Perry, J. *The Far Side of Madness.* Englewood Cliffs, NJ: Prentice-Hall, 1974.

Simonton, C.; Matthews-Simonton, S.; and Creighton, J. *Getting Well Again.* Los Angeles: J.P. Tarcher, 1978.

Spring, B., and Coons, H. Stress as a precursor of schizophrenia. In: Neufeld, R., ed. *Psychological Stress and Psychopathology.* New York: McGraw-Hill, 1982. pp. 13–53.

Thyer, B.; Irvine, S.; and Santa, C. Contingency management of exercise by chronic schizophrenics. *Perceptual and Motor Skills,* 58:419–425, 1984.

Vaughn, C.; Snyder, K.; Jones, S.; Freeman, W.; and Falloon, I. Family factors in schizophrenic relapse. *Archives of General Psychiatry,* 41:1169–1179, 1984.

Wallace, C. The social skills training project of the Mental Health Clinical Research Center for the Study of Schizophrenia. In: Curran, J., and Monti, P., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment.* New York: Guilford Press, 1982. pp. 57–89.

Welis, H., and Canfield, J. *One Hundred Ways to Enhance Self Concept in the Classroom.* Englewood Cliffs, NJ: Prentice-Hall, 1976.

Wing, J.; Cooper, J.; and Sartorius, N. *The Description and Classification of Psychiatric Symptoms.* London: Cambridge University Press, 1974.

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Incentives and Disincentives for Schizophrenia Research Careers

NIMH Activities

Abstract

A meeting was convened to discuss schizophrenia research career incentives and disincentives. A diverse group of scientists from different fields and in different career stages discussed the causes of and potential solutions to schizophrenia research manpower problems. The scientific and clinical, individual and institutional factors involved were identified and numerous disincentives to careers in schizophrenia research were noted. A number of possible incentives were also suggested and debated. There was general agreement on the following issues: (1) attracting and maintaining a larger group of young investigators is crucial; (2) multidisciplinary studies combining clinical and basic science approaches should be strongly encouraged; and (3) mechanisms for increasing funding and stimulating interest in schizophrenia research careers should be studied by the National Institute of Mental Health. Specific recommendations are now being assessed to determine how best to respond to the schizophrenia research manpower needs that were identified at the meeting.

The meeting on Incentives and Disincentives for Schizophrenia Research Careers was convened by the National Institute of Mental Health (NIMH) on July 12, 1985. An appendix listing participants appears at the end of this report.

Dr. Frazier opened the meeting, noting that we have purposely assembled a highly qualified and diverse group of scientists—at different stages in their careers, from different fields, and with different career experiences. He underscored the importance of the meeting in terms of his establishing schizophrenia research as a top priority for NIMH and in the context of the reorganization of NIMH. The Program in Schizophrenia, as an organizational entity, will be responsible for the full array of mechanisms for developing the field, from regular research grants to fostering overall research resources in this area, including programs for training young scientists.

Given the centrality of schizophrenia research to the NIMH mission, Dr. Frazier expressed deep concern about difficulties in attracting and retaining people in the field. Although specific data are unavailable, he noted, "I am chagrined personally and I believe that the perceptions of a good many people in the field are that too little is being done."

The ability to attract and retain the most promising scientists is an issue of major proportions. Given the long pipeline of research training leading to an independent research career, we must assume that the situation will worsen significantly over the next decade unless something is done to change the situation. It is imperative that any potential shortage of well-trained research personnel be addressed to prevent irreparable damage to future research efforts.

Apparent causes of schizophrenia research career disincentives are multiple and complex as noted in table 1. To some extent, they reflect generic disincentives for clinical research careers. In addition, the nature of the illness, the methodologies and approaches for its study,

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and the settings where schizophrenia research is performed all potentially limit the attractiveness, satisfaction, and career enhancement important for personal motivation. Importantly, many of these limitations are a function of institutional decisions that limit career advancement in areas of research unlikely to yield dramatic findings in a short time; place schizophrenia research in less attractive, off-campus settings; and limit funding for research requiring inpatient observation and intervention.

Dr. Frazier's remarks initiated spirited and far-ranging discussion. Brief summaries—often overlapping and by no means exhaustive—of major issues raised by the participants are recounted next.

First considered are issues related to analysis of the extent, significance, and reasons for the problem. Following are discussions of suggestions for reducing disincentives and developing new incentives for schizophrenia research.

Analysis of the Problem

Extent and Significance. "No survey of the field is necessary," claimed Dr. Detre, since a content analysis of several leading journals would show that a relatively small proportion of the articles are in the area of schizophrenia, reflecting a relative lack of high-quality research results. Dr. Carpenter pointed out that a dropoff in the literature of schizophrenia may be occurring because journal editors will no longer publish clinical case reports and overgeneralized assertions without adequate data. He said that it has become evident that there "was never a viable base of research." Dr. Friedhoff commented that part of the problem is that many investigators—particularly basic scientists—may be doing research relevant to schizophrenia but fail to conceptualize it as relevant. The focus must be on manpower, noted Dr. Holzman, but the problem is not that experienced researchers are moving away from the field; rather, "there is a failure to attract and keep young people."

Most participants felt that the personnel issue was central. Moreover, as Dr. Swisher noted, the problem must be conceptualized at three levels: (1) there is a declining interest among young physicians, generally, in clinical research careers; (2) there may be particular factors that make young psychiatrists even less likely to undertake preparation for research careers; and (3) even with the limited pool of psychiatric researchers, there are disincentives for studying schizophrenia.

Dr. Matthysse pointed out that, in the past year, there has been a 20 percent drop in schizophrenia research applications to the Scottish Rite Foundation. This would suggest that funding availability alone does not drive research interest but that "intellectual ferment" is a major factor. Just as schizophrenia is a heterogeneous disorder, the incentives and disincentives for schizophrenia research are complex and varied.

Nature of the Scientific Problem. Dr. Swisher noted, "Clearly, [schizophrenia] is a problem that looks intractable at the present time." Dr. Wyatt remarked that Dr. Kety's seminal analysis of the field in 1959 pointed out the extent of likely artifactual findings in the literature and the difficulties in carrying out scientifically valid research in the field. The nonspecificity of many findings in schizophrenia makes other areas more intellectually "clean" and satisfying. Dr. Jernigan felt that some "defections" to dementia research may be due to the presence of more replicable effects and the more specific links between the neuropsychometric pattern and the biological findings.

There was general agreement that future research on schizophrenia must be enriched by the basic sciences, especially the neurosciences and molecular biology. While some groups are pressing for more clinical or "applied science" efforts, there was a consensus among the participants that basic sciences (brain) research is crucial and that far more is needed in the future. Dr. Kety emphasized that more basic knowledge would be needed for application to clinical problems. Dr. Goodwin noted that in combining basic and clinical science, one must be extremely careful that one "doesn't miss areas that would be obvious to 'pure' basic scientists in the field and wind up with second-rate basic science."

Similarly, while there was a consensus that addressing the problem of heterogeneity was essential, there were differing views on how it should be done. As Dr. Detre noted, "The first sentence is always that schizophrenia is a heterogeneous disorder, but that is the last mention of it." Many felt that purely phenomenologic research is unlikely to develop homogeneous groups amenable to biological analysis. Others noted that there will always be some need to focus on diagnosis even if only to enable communication among investigators. Dr. Bowers noted that patients with schizophrenia clearly have problems in human relationships that "perhaps don't defy description, [but] certainly challenge description." He underscored the importance of psychosocial issues with respect to relapse and urged they be considered in conjunction with biological variables. Further complicating these issues is the relatively low prevalence of the disorder and development of diagnostic criteria that may too quickly have narrowed the spectrum of subjects for study. Dr. Matthysse cautioned, "We shouldn't be imprisoned by diagnostic language," and suggested that raw data elements should be separated from their assembly into higher order concepts.

An overwhelming problem is the lack of a comprehensive theory. Although the dopamine theory was useful at the time, noted Dr. Friedhoff, "we need new hypotheses to attract new people." Dr. Swisher agreed and pointed out that, in other fields, the development of a new hypothesis or a particular finding provides "a handle" for people to grab. Dr. Rieder cautioned about being too pessimistic in assessing the state of the field: "There are good studies and important new developments with many more strategies to pursue; there is, however, a manpower problem."

Nature of the Clinical Problem. Dr. Carpenter noted that there are relatively few gratifications in the treatment of schizophrenia, given the generally limited clinical success. Dr. Goodwin suggested that the growth and popularity of research in affective disorders may be less a reflection of the potential scientific advancement and more a function of the availability of more effective treatments. Dr. Bowers emphasized the stressfulness of clinical care for schizophrenic patients, particularly in research settings with unmedicated patients. Furthermore, it often is difficult to get patients and their families to participate in research. Several participants noted that, in the past, the field has played a role in stigmatizing the families of schizophrenic patients rather than trying to benefit from their experiences and ideas.

There is also, as Dr. Luchins emphasized, "a separation of clinical care and academic research." Many participants pointed to the rarity of links between academic and State institutions, or with other "institutions" (i.e., nursing homes, board and care facilities, etc.) that could provide access to patients. Furthermore, noted Dr. Swisher, not only are these patients behaviorally and geographically inaccessible to researchers, but there are legal barriers to their participation in research. Medical ethics has focused primarily on the individual, often giving inadequate attention to the potential risks or benefits to society as a whole, he said.

Individual Career Factors. Participants felt strongly that interests in research must be developed and nurtured early in the career path. Importantly, however, this must be done in a stable environment, with exposure to role models from a variety of disciplines capable of providing enthusiasm and confidence that something can be accomplished. To provide optimum experience and likelihood for a career in schizophrenia research, there must be specified attention, enthusiasm, access to patients with schizophrenia, and exposure to longitudinal designs, as Dr. Carpenter noted. The importance of access to clinical populations during the predoctoral period of study was also underscored.

Dr. Swisher distinguished "training" from "education" and suggested emphasizing the latter. A general understanding of biochemistry is more important than learning a specific technique. Others also cautioned against overemphasis on technology but underscored the importance of training clinical people in basic science areas. Dr. Berger pointed out the value of familiarity with advanced research technologies, not only in terms of conducting research in schizophrenia but also in establishing collegial links with other areas of research.

Careful attention must be paid to avoid burdening young scientists in a research career track with overwhelming administrative, teaching, or clinical duties. Dr. Jernigan noted that, in her experience, "career development funding can make all the difference in the world." Further, Dr. Holzman described a critical point in career development—approximately 7 years postdoctoral—when people seemed most vulnerable to dropping out of a research career track because of discouragement over getting independent funding, financial attraction of clinical practice, family obligations, and so forth. Careful attention must be paid to focusing on the special needs of young investigators at these "danger points." In addition, he advocated a "double career" strategy that allows people to make a less than 100 percent commitment to what may be viewed as an intractable problem. This may be important for both academic survival and morale.

Potential opportunities and problems should be considered at later career stages as well. Encouraging established scientists to consider how their area of study may be relevant to schizophrenia and enticing them to enter the field full time may yield new opportunities. Also, senior investigators may need to retrain and update their skills. There should also be opportunities for senior people to be creative in a "think tank" atmosphere.

Ultimately, the growth of the field is dependent upon the existence and support of role models or mentors who can attract, inspire, and teach younger people. Dr. Luchins emphasized the need for more such mentors in schizophrenia research, while Dr. Jernigan suggested that the lack of female role models is a major impediment to attracting more women into the field.

Institutional (Academic and Governmental) Factors. Clearly institutional factors at all levels play a major role in molding individual attitudes and decisions. For example, deinstitutionalization disseminated patients away from more concentrated facilities, making them less accessible for clinical studies. Also, the expense of inpatient studies (and particularly inpatient studies of schizophrenia) makes administrators at the level of the academic medical center reluctant to invest in this area, thus reducing young people's exposure to these areas of research. There is still too often a lack of commitment within departments of psychiatry to foster research and nurture young people interested in research. Dr. Burke pointed to American Association of Medical Colleges data indicating that a significantly lower proportion of full-time faculty in psychiatric departments (compared with faculty in other clinical departments) have had research training experience, are full-time researchers, or have applied and been funded for Federal research grants. Even in departments intensively invested in research, there may be an insufficient base of people specifically involved and interested in schizophrenia research. It is also important, as Dr. Berger noted, that there be an institutional commitment to fostering interdisciplinary research, "actively enlisting all disciplines from sleep physiologists to ophthalmologists and even mathematicians as schizophrenia researchers."

The role of the Federal research establishment and especially NIMH is critical. Clearly, the expansion of research at the National Institutes of Health (NIH) and the development of programs such as the National Institute on Aging Alzheimer's Disease Centers, along with the relative lack of growth in the NIMH budget, have attracted researchers to other fields. The developing relationship between the research community and family groups has potential for making gains in advocacy for more mental illness research funding.

As Dr. Kety noted, however, many people "might be interested if they were aware of NIMH's interest." Dr. Wyatt noted that the "watering down" of the NIMH mission by a focus on a broad array of psychological and social problems may have undermined attempts to make clear the seriousness of schizophrenia. The refocusing of the Institute over the past several years and the reorganization of NIMH now provide the opportunity to reemphasize schizophrenia research.

Developing Incentives and Reducing Disincentives

The afternoon session was devoted largely to "brainstorming" recommendations for addressing the problems summarized in table 1. While there was strong consensus on many of the suggestions, others generated considerable controversy.

Fostering Centers of Excellence. There was general agreement, with some caveats, on the need to bring together people from different disciplines and approaches, at different stages of their careers, within highly concentrated intellectual settings associated with a major academic center, to focus on schizophrenia. The stability, availability of appropriate technology, presence of enthusiastic senior mentors, local determination of science direction, and general intellectual excitement of such an enterprise provide an ideal environment for young investigators. Emphasis must be placed on having strong and continuing relationships with basic science departments as well as with teams of investigators studying other, similar problems (e.g., Huntington's disease, Alzheimer's disease). Care must be taken, however, that such centers demonstrate a clear commitment to recruiting and training young scientists in the field of schizophrenia research.

Supporting Costs of Care. One aspect of schizophrenia research, largely related to the concept of centers, is the need for some mechanisms to cover the costs of inpatient care. NIH's General Clinical Research Centers Program has included specific provisions for bed costs. The NIMH's Clinical Research Centers, while allowing such funding, have never specifically had it as a part of the program; center directors, in order to keep within the centers' $750,000 limit, have infrequently requested such funds. Nevertheless extremely tight restraints on length of stay, potential development of Diagnosis Related Groups, and the difficulties involved in carrying out many important studies requiring around-the-clock biological and behavioral monitoring (frequently with unmedicated patients) suggest a need for establishing some mechanism of support.

Developing State/Academic Relationships. Several participants emphasized the need to somehow add new luster to State hospitals by adding a scientific component. Not only does collaboration with academic centers provide added prestige and improved care and morale at State hospitals, but it may also provide researchers with access to patient populations and at least partial support of bed costs. Although there are significant problems in developing such an effort, a number of participants described their involvement in successful examples of academic-State collaboration. Among the problems noted were the difficulty in recruiting people to this kind of setting, the lack of incentives for State bureaucracies to facilitate these relationships, and, often, the lack of assurance of a long-term commitment by all parties (State bureaucracy, unions, academic institution) to the effort. Many felt that NIMH could help in providing additional incentives to facilitate these relationships by promoting joint applications by academic centers and neighboring State institutions. Additionally, consideration should be given to broadening the notion of "State institutions" to settings other than State hospitals, including nursing homes, board and care facilities, and the like. In addition, NIMH should encourage States to develop their own research support programs, such as the innovative one now being developed in California.

Table 1. Disincentives for schizophrenia research careers

1. Clinical and biological heterogeneity.
2. Poor correlation between clinical and biological findings.
3. Lack of specific "markers" for diagnosis and classification.
4. An insufficient amount of basic scientific knowledge available for application to clinical problems.
5. Over emphasis on methods to determine focus of scientific inquiry.
6. Chronic nature of the disorder requiring more difficult longitudinal approaches for many studies.
7. Scientific problems of studying patients while they are medicated.
8. Clinical problems of studying unmedicated patients.
9. Poor patient response to treatment.
10. Patient uncooperativeness—since many do not understand that they are ill.
11. Families "turned off" by mental health professionals' blaming them for causing patients' problems.
12. Beds for schizophrenia research subjects costing more than those for other patients (more nursing care needed).
13. Legal and Institutional Review Board-enforced restriction of access to research patients.
14. Risk to a research career from focusing on a chronic, complex disorder less likely to yield answers or multiple publications important for academic advancement.

Establishing Consortia and Other Collaborative Arrangements. While there was general agreement on the need for collaboration among various centers and investigators, there were sharp differences as to the nature of such collaboration. Some asserted that it might be appropriate to develop multisite studies to combine epidemiological and biological techniques, an approach similar to that taken by the National Cancer Institute in investigating low-prevalence disorders. Others felt that it was too early for fixed-protocol multicenter studies but that it would be important to develop networks whereby centers could maintain frequent communication and try to use standardized tools. At a minimum, NIMH should reactivate the earlier proposal for a consortium to study the problem of heterogeneity.

Big Science Versus Small Science. Concern was expressed that a focus on "big science" (i.e., centers or large collaborative projects) might "gobble up" a disproportionate amount of limited funds. It may be more productive to maintain a large number of researchers on the "front lines" to come up with a serendipitous finding. Others asserted that the "days of the 'Lone Ranger' are gone," and that, while misuse must obviously be avoided, maintaining a critical mass of researchers in elite academic institutions would be the most productive strategy. As one participant stated, "There cannot be a quality research program in every garage." Ultimately, these views are not mutually exclusive, and attention to both long-term and short-term goals and to "big" and "small" science are necessary. Several suggestions were offered to accelerate the pace of research activity, including smaller grants in the $15,000 to $100,000 range to individuals or small consortia, and aggressive outreach to identify investigators likely to be interested in the field.

Linking Basic and Clinical Science. There was overwhelming support for enhancing collaboration between basic scientists and clinical investigators, but there were varying suggestions on how to accomplish this goal. The problem of the "house biochemist" was alluded to in terms of the difficulties of maintaining first-rate basic science in an environment unconnected to other basic science colleagues. Yet more incentives must be developed to attract basic scientists—at all career levels—into research relevant to schizophrenia, and to develop collaborative relationships. This could be done in the context of centers, or through individual grant programs announced through Requests for Applications (RFA's) on basic science areas relevant to schizophrenia, or through small workshops (such as those held on Huntington's disease) that would bring together basic and clinical scientists. A necessary element of any strategy is identification of the scientists and fields of study with relevance to schizophrenia. It was suggested that asking grant applicants how their work might relate to schizophrenia would at least get people to think about these issues.

Nurturing Career Development. Given the reorganization of NIMH and the decentralization of programs for career development, it was urged that some mechanism to assure a focus on these issues and to explore aggressively new opportunities be maintained. There was universal agreement on the need to develop a cadre of young investigators by exposing people to research early in their careers, ideally in a major research center or similar "breeding place." Consideration should be given to developing summer and part-time stipend programs in which participating medical students could gain exposure to clinical research projects.

Research training should be an inherent and specifically identified part of any specialized center program that is developed. Career development programs focused on schizophrenia should be expanded to free up junior faculty to be more productive scientifically. Some mechanisms, such as the Physician Scientist Award, with its 2- to 3-year requirement for basic science experience, while appropriate for a select few, may discourage some people. Approaches similar to the Geriatric Mental Health Academic Award might be considered for schizophrenia. Liberal use of the National Research Service Awards mechanism also should be considered. The recent increase in the stipend level makes it potentially more attractive, and the low, fixed, indirect cost rate makes it a more efficient use of funds. Attention to the critical "7-year mark" in career development might include removing the distinctions between the level I and level II of the Research Scientist Development Award. In addition, we should consider programs of supplemental salary support for investigators participating in State-academic collaborative programs to enhance their attractiveness and overcome recruitment problems of these programs. Also, we must consider using career development approaches for investigators at advanced stages in their careers who might profitably shift their focus into schizophrenia research. Finally, studies investigating the various paths and pitfalls in the career development of schizophrenia researchers compared with those of other psychiatric and biomedical researchers might be useful.

Fostering Intellectual Ferment. Establishing a sense of excitement and intellectual ferment in a field is of prime importance in attracting people to it. Obviously, much of this kind of excitement must be generated from the actual findings of basic and clinical investigators; nevertheless, NIMH can apply its convening authority or support others to sponsor conferences that would enhance a sense of excitement in the field. Such approaches could include large international conferences modeled on the two earlier Rochester conferences, which created a sense of ferment in the field; smaller workshops modeled on the Hereditary Disease Foundation workshops on Huntington's disease, which linked small groups of scientists in different areas, at different career stages, and also provided some modest funds for start-up projects; and other approaches modeled on the NIMH Division of Biometry and Epidemiology's effort in consultation-liaison psychiatry research, which had pairs of investigators from two different disciplines jointly develop preliminary proposals and participate in a workshop with expert consultants and other pairs of investigators. It was suggested that NIMH should provide some reward for convening conferences and the like; to do it properly is hard work, but it can be extremely productive. As part of this effort, enhancing relationships between NIMH and both professional and lay organizations should be a high priority. Among examples cited was the role of the American Heart Association in the growth of cardiac research.

Revising Grant Review and Funding Processes. The current environment of research support throughout biomedical research is not conducive to funding new and innovative research. Generally, reviews of research proposals "follow the most orthodox lines—everybody is looking for a 'safety net.' " Dr. Detre felt that innovative approaches to schizophrenia are too often "shot down" and that there needs to be greater willingness to try unusual or "long shot" approaches. It was suggested that funding limitations may be leading review groups to rate highly only those studies likely to "succeed." This could favor methodological refinements over a search for more basic mechanisms.

It was not entirely clear how this trend could be reversed. Some suggested substantial changes in the structure and process of review; others suggested it might be best to attempt some modest experiments in the review process and also await the results of NIH studies of this issue. Others felt the best approach might be to orient and educate the Initial Review Group members more effectively. Similarly, no consensus was reached on whether some type of preferential funding for schizophrenia research should be developed in response to the priority score inflation problem afflicting all NIH and Alcohol, Drug Abuse, and Mental Health Administration Institutes. Some felt that specific mechanisms or RFAs, setting aside small amounts of funds for innovative approaches in schizophrenia, would be important as a kind of "venture capital" approach. Others cautioned that the development of a formal "set-aside program" might undermine respect for the field as a whole and attract second-rate investigators who "couldn't get funded elsewhere." More important was the concern that this might touch off a "scramble for set-asides" and "endless politicking" by special interest groups. This type of "Balkanization problem" has afflicted the mental health field in the past; exacerbating that problem during a time of relative quiescence might undermine the current potential for an overall boost for the field.

Assuring Stability. Careful attention must be paid to supporting current investigators in areas related to schizophrenia and to enhancing their abilities in attracting young scientists to the field. Mechanisms to provide support for a longer period should be considered—3 years may not be sufficient. In addition, it is essential that NIMH's continuing commitment to stimulate additional high-quality research in schizophrenia, both extramurally and intramurally, be made concrete and widely publicized.

Conclusion

There was general agreement that the workshop was stimulating and productive. The variety of perspectives from researchers at different career stages was useful in identifying schizophrenia research career disincentives and suggesting a variety of possible incentives. There was general agreement that schizophrenia research can be more difficult to carry out than research on other mental illnesses for a number of scientific, clinical, academic, career, and institutional reasons. There was also agreement that attracting young investigators to the field is a key to improving the prospects for productive research. Numerous specific recommendations are being discussed and assessed by NIMH to determine how best to implement changes that will have a significant effect on this field.

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First Person Account: A Parent's View on Enforcing Medication

by Eleanor Slater

The article that follows is part of the *Schizophrenia Bulletin's* ongoing *First Person Accounts* series. We hope that mental health professionals—the *Bulletin's* primary audience—will take this opportunity to learn about the issues and difficulties confronted by consumers of mental health care. In addition, we hope that these accounts will give patients and families a better sense of not being alone in confronting the problems that can be anticipated by persons with serious emotional difficulties. We welcome other contributions from patients, ex-patients, or family members. Our major editorial requirement is that such contributions be clearly written and organized, and that a novel or unique aspect of schizophrenia be described, with special emphasis on points that will be important for professionals. Clinicians who see articulate patients, with experiences they believe should be shared, might encourage these patients to submit their articles to *First Person Accounts*, Schizophrenia Research Branch, NIMH, 5600 Fishers Lane, Rm. 10C-06, Rockville, MD 20857.—*The Editors*.

My daughter developed paranoid schizophrenia over the last 15 years. Two years ago, she stopped taking her medication and began sliding back into a familiary pattern of delusions and accusations. She had begun making frantic calls to the police and was about to be evicted from her apartment for harassing the other tenants when she physically attacked her fiance and was taken to a community hospital in her catchment area. At the recommitment hearing after the initial 5-day observation period, she was disturbed enough to be held involuntarily for another 20 days.

After the hearing, her fiance and I spoke to a psychiatrist and were shocked to discover that the community hospital did not force patients to take medication. The psychiatrist said they would talk my daughter into taking it. From 13 years of experience with her problem, I knew they couldn't convince her, in her disordered state, to take medication. Luckily, we were able to find another community hospital that would make sure she got the medication she needed, and we reached an agreement to have her transferred there.

Watching someone you care about disintegrate over a period of months into someone you can't recognize, someone made fearful and frustrated by her own imaginings, is painful. To be forced, as her fiance was, to have a loved one committed because of physical violence is deeply traumatic for everyone involved. The most disheartening thing was finding out she wouldn't get the help she needed at the end of all that.

I was so upset by our experience that I wrote to the Commissioner of Mental Health for Pennsylvania, questioning the policies of this particular hospital. He replied that he thought a patient committed by the court for treatment should be treated and that if there was a question about the advisability of forcing medications, a second doctor should be consulted. His office, however, could not interfere with a community hospital's policies.

I was surprised to learn that many States do not force medication. I would guess this policy might be the result of hospitals being sued by patients or patients' rights groups. The families have been so frustrated by this very serious problem that

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they have sometimes even considered taking legal steps to see that their family member was effectively treated with medication.

As a regular policy, not forcing psychotic patients to take medication would seem to create many problems for hospitals. How do hospitals deal with patients who may harm themselves or others? Should they be confined in restraints, as they were years ago? Should they be kept in isolation, which requires large facilities? Or will they end up being discharged back into the community as untreatable?

One of the things that our support group, Parents of the Adult Mentally Ill (PAMI), does is try to implement sensible treatment based on our own years of experience with mentally ill children. Most, if not all, of us tried for a long time to talk our sons and daughters out of their delusions before we came to accept that reasoning just doesn't help. In the same way, we know how impossible it is to convince someone who is mentally ill to take medication when the person is becoming irrational. The problem isn't one of intelligence; most of our children have some college education and some, like my daughter, have college degrees. The problem is in the nature of the illness itself. According to Dr. E. Fuller Torrey (1983) of St. Elizabeths Hospital in Washington, D.C.:

> People with schizophrenia have diseases of the brain; they cannot give informed consent. . . . Supporting this position is a study of chronic schizophrenic patients showing that only 27% of them understand that they needed medication. [p. 191]

As a parent, I also know that medication is not perfect and that the side effects can be distressing. When my daughter goes back on her medication, I feel bad seeing her shuffling or experiencing involuntary arm and mouth movements. These symptoms usually subside over time; but she also gains weight, and she hates being heavy. I think she hates taking medication most of all because she is, in a sense, admitting she is mentally ill, something she very much wants to deny.

But the alternative is much worse. She becomes more and more paranoid and delusional. I can only imagine what hell her life must be when she tells me people are trying to kill her, or that she knows I'm trying to commit suicide, or that evil people won't let her sleep at night. I can see her distress as her world begins crashing in on her and she needs help. I don't think she needs the right to be crazy or that she, or any other mentally ill patient, needs the right to commit suicide or act in a way she will later regret.

One thing that some family support groups have done to try to balance the need for medication with its adverse side effects has been to launch a letter-writing campaign to drug companies urging them to spend more time and money on research. I understand that some of the newer medications have been improved. On the other hand, while many parents think hospitals should enforce giving medication to mentally ill patients who have had to be committed to their care, we would also like to see hospitals question or eliminate the practice of giving massive doses of medication immediately on admission. This can cause patients to fear the medications. Although this is done less frequently, drugs should be used as discriminatingly as possible to help the patients.

My daughter has been hospitalized a number of times over the last 15 years, and it takes at least a month for the medication to begin to turn her thinking around. Then she beings to show a remarkable change in her thinking and in her personality. She becomes a sweet and loving person, reminiscent of the daughter I used to know. If a proposed ruling countermands enforcing medication in this State, it frightens me to think that she and thousands of others may have no help when they become irrational. I don't want to see that wasteland of frustration and torment become a way of life for her, and for thousands like her.

Reference

Torrey, E.F. Legal and ethical dilemmas and schizophrenia. In: Torrey, E.F. *Surviving Schizophrenia: A Family Manual.* New York: Harper & Row, Publishers, 1983. pp. 182-195.

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Eleanor Slater is one of the founders of the first support group in Pittsburgh—PAMI (Parents of the Adult Mentally Ill). She is on the executive board of United Mental Health and is Program Chairperson of ADMIT, an advocacy group representing the support groups in the Pittsburgh area.

Bibliography and Abstracts on Schizophrenia

The abstracts that appear below are drawn from the computer-based information storage and retrieval system operated by the National Clearinghouse for Mental Health Information, and from the Mental Health Abstracts database, produced by IFI/Plenum Data Company, 3202 Kirkwood Highway, Wilmington, DE 19808. They were selected for inclusion here on the basis of their direct relevance to schizophrenia: judgments about the quality of the article or book abstracted have been left to the reader. Foreign titles have been translated and the city of the journal's origin noted. Languages that use neither the Latin nor Fraktur (German) alphabets (e.g., the Slavic languages) have been transliterated. Titles of articles, books, and infrequently cited journals have been translated from these languages, and all other identifying information appears in English in their citations.

The articles described in these abstracts are not available from either the Clearinghouse or the Schizophrenia Research Branch, but are available for access through Dialog Information Retrieval Systems in reference departments of major university libraries and public libraries.

Attention, Perception, Cognition

6632. Balogh, D.W., and Leventhal, D.B. (Ball State University, Muncie, IN 47306) The use of temporal and amplitude cues by schizophrenics, psychiatric controls, and aged normals in auditory lateralization. Journal of Nervous and Mental Disease, 170(9):553–560, 1982.

The ability of paranoid and nonparanoid schizophrenic patients, psychiatric controls, and aged controls to use temporal and amplitude cues to make judgments about the direction of auditory stimuli on an auditory lateralization task is investigated. Twelve subjects in each group were tested on three lateralization conditions: temporal-fixed, temporal-shifted, and amplitude. The subjects' task was to identify the apparent direction (left or right) of a train of clicks presented binaurally through headphones. In the temporal conditions interaural click onset differences provided the lateralization cues, while in the amplitude condition interaural amplitude differences provided the cues. In the temporal-fixed condition, onset asynchrony was constant for the duration of each stimulus. In the temporal-shifted condition, onset asynchrony was introduced midway through each stimulus presentation. A forced-choice random staircase method was used to determine the 70 percent correct response threshold for each subject. Results indicated that paranoids had particular difficulty with the temporal-fixed condition relative to the temporal-shifted condition. The opposite was true for the nonparanoids. Both control groups showed no significant difference in performance on the two temporal conditions. Psychiatric controls performed significantly better than aged controls in both the fixed and shifted conditions. There were no significant differences among groups on the amplitude condition. The data were discussed in terms of schizophrenic patients' clinical symptoms and previous perceptual research with schizophrenic patients. (23 references)

6633. Bernstein, A.S.; Frith, C.D.; Gruzelier, J.H.; Patterson, T.; Straube, E.; Venables, P.H.; and Zahn, T.P. (Department of Psychiatry, State University of New

York, Brooklyn, NY 11203) An analysis of the skin conductance orienting response in samples of American, British, and German schizophrenics. Biological Psychology, 14:155-211, 1982.

The existing literature involving the phasic orienting response (OR) in schizophrenia by examining the skin conductance component (SCOR) reports conflicting results with divergent implications for the nature of the attentional dysfunction in these patients. This report addresses this issue by applying a common set of response definitions and uniform statistical-analytic procedures to the previously gathered electrodermal data obtained independently in each author's laboratory. A total of 14 studies is involved, drawn from six laboratories in the United States, United Kingdom, and West Germany. Collectively, these studies examine chronic and acute schizophrenics, males and females, those receiving and not receiving neuroleptic drugs. (60 references)

6634. Callaway, E., and Naghdi, S. (Langley Porter Psychiatric Institute, University of California, San Francisco, Medical Center, San Francisco, CA 94143) An information processing model for schizophrenia. Archives of General Psychiatry, 39(3):339-347, 1982.

An information processing model for schizophrenia is presented to overcome paradoxes that are problematic for diagnosis. It is contended that an information processing disorder must be present for a person to be diagnosed as schizophrenic, and that it is present when schizophrenia symptoms occur along with other diseases. The proposed model suggests that schizophrenia symptoms occur along with other diseases and that schizophrenics have a deficiency in information processing that can be characterized as conscious, serial, and limited in channel capacity. In contrast, those processes that seem automatic, unconscious, parallel, and almost unlimited in capacity are seen as being normal or supernormal. Characteristics of the limited channel capacity processes (LCCPs) in schizophrenia are described, and results are reviewed from studies of auditory evoked potential and delayed recovery cycles which may be relevant to schizophrenia. In addition to making predictions about the disorder, the model also is considered helpful in researching three areas where the empiric base is especially weak: LCCP plasticity, state-trait ambiguity, and lack of comparisons between paradigms. (61 references)

6635. Galbraith, K.J., and Steffy, R.A. (Hamilton Psychiatric Hospital, Hamilton, Ontario, Canada) Redundancy deficit in the reaction time of normal subjects under standard and meprobamate-impaired conditions. Canadian Journal of Behavioural Science, 14(1):71-81, 1982.

Latency and redundancy deficit, two indexes of reaction time performance that reliably show impairment in process schizophrenics, were assessed using normal subjects with no psychiatric history. Subjects were tested under standard/optimal and drug-impaired conditions to evaluate whether an expected increase in latency would be accompanied by a correlated increase in redundancy deficit. In other research the redundancy deficit index has been found to be independent of latency and also has shown special promise as a marker of schizophrenic information processing difficulty. The present findings confirmed the independence of these two markers in that no increased redundancy deficit was observed despite a significant increase in latency. The findings also revealed a greater degree of redundancy deficit in normals under the optimal conditions than had been expected. In response to this observation, a methodological problem with trial arrangements was discussed and a thorough review of studies which tested redundancy deficit in normal subjects was conducted. (33 references)

6636. Hasse-Sander, I.; Gross, G.; Huber, G.; Peters, S.; and Schüttler, R. (Psychiatrische Klinik, Universitat Bonn, D-5300 Bonn-Venusberg, West Germany) Psychological testing in basic stages and pure residual syndromes of schizophrenic diseases. Archiv für Psychiatrie und Nervenkrankheiten, 231(3):235-249, 1982.

Three groups of schizophrenic diseases which cannot be differentiated with regard to psychopathological cross-section syndromes were investigated using psychological tests. One group (22 cases) of reversible postpsychotic basic stages and two groups of pure residual syndromes with an average duration of disease of 9.3 years (20 cases) and 17.5 years (21 cases) were studied. All three groups deviated significantly from the norm. The pathological values were most marked in performance on the HAWIE (Hamburg-Wechsler Adult Intelligence Scale). All groups showed a significant lowering of performance compared with the verbal scale (which corresponds approximately to the premorbid intelligence level), the concentration lapse test, and measures of sensorimotor reaction time by the Wiener reaction test. They also scored lower on the Benton test and on examinations with a writing scale. Reversible postpsychotic asthenic basic stages were not significantly differentiated in psychological tests from pure residual syndromes which were compared with regard to sex and age at disease onset and (in the pure residual group) with regard to age and school success. Decreased performance on the reaction test, the concentration test, and in the nonverbal portion of the HAWIE correlated with symptoms identified by other tests. The positive correlation was more distinct in reversible postpsychotic basic stages than in pure residuals. The findings support the global hypothesis that deficiencies of schizophrenic stages with a component of reversible or irreversible pure potential reduction are based on disturbances of perception and interpretation of information. They also support the assumption of no fundamental differences between cerebro-organic or psychosyndromes of schizophrenic diseases determined by the dynamic and cognitive disorders of pure deficiency. (31 references)

6637. Kay, S.R. (Albert Einstein College of Medicine, Bronx, NY 10461) Conceptual disorder in schizophrenia as a function of encoding orientation. Journal of Nervous and Mental Disease, 170(3):154-163, 1982.

This study evaluated the hypothesis that schizophrenic conceptual disorder derives from abnormal verbal encoding, geared to salient affective and physical cues at the expense of conceptual attributes. Subjects were 63 adult psychotic inpatients, including 42 schizophrenics and 21 nonschizophrenics of similar age, sex, race, and chronicity. They underwent 12 trials on Wickens' release from proactive interference (PI) procedure to measure reliance on three dimensions for encoding: conceptual (taxonomic class), affective (evaluation axis of the semantic differential), and physical (rhyming sounds). The release effect for schizophrenics, compared with controls, was found to be significantly weaker on the conceptual dimension and correspondingly stronger on the affective dimension. Whereas controls exhibited the normal attraction to conceptual over other cues, schizophrenics presented a flat profile. Nonparanoid and thought-disordered schizophrenics showed particular deficits on the conceptual dimension. All schizophrenic groups contrasted controls by their diminished PI release with less salient concepts. The results thus suggested that encoding by schizophrenics is uniquely oriented to stimulus salience rather than semantic relevance. A fundamental deficiency in processing of cues essential for conceptual operations, accordingly, seems to underlie the conceptual dysfunction in schizophrenia. The implications were discussed in relation to diversities in the cognitive literature and prevalent theories. (66 references)

6638. Reich, S., and Cutting, J. (Dept. of Neurology, London Hospital, Whitechapel, London E1 2AD, England) Picture perception and abstract throught in schizophrenia. Psychological Medicine, 12(1):91-96, 1982.

Acute schizophrenics were compared with depressed, normal, and brain-damaged subjects on their ability to appreciate a meaningful picture. Their responses were measured for level of abstraction (how well the theme was conveyed), for strategy (whether details were reported before or after global interpretation), and for appropriateness of content. Schizophrenics were less abstract than normals, more abstract than damaged subjects, but no different from depressives on this measure. Their strategy was different from normal subjects, but comparable with that of depressive subjects. The majority of schizophrenics gave inappropriate responses. It is suggested that meaningful picture interpretation might be a useful tool for evaluating the distortion of meaning which characterizes schizophrenia. (22 references)

6639. Saccuzzo, D.P.; Braff, D.L.; and Sprock, J. (San Diego State University, San Diego, CA 92182) The effect of mental retardation and schizophrenia on information processing. Journal of Nervous and Mental Disease, 170(2):102-106, 1982.

Schizophrenic persons with average intelligence and dual-diagnosis mentally retarded schizophrenic persons were tested in a forced-choice letter discrimination task to examine the relationship between schizophrenia and retardation relative to the information-processing theory. The subjects were eight schizophrenic persons of average or better intelligence, eight mentally retarded schizophrenic persons, and a control group of eight minor depressive individuals who matched the nonretarded schizophrenic group for intelligence. The groups did not differ significantly on the minimum exposure duration needed to identify an unmasked target stimulus at criterion levels of accuracy. When masked stimuli were employed, however, the depressive group had significantly more correct detections than both schizophrenic groups. More importantly, the performance of the two schizophrenic groups did not differ significantly. Data indicated that schizophrenic deficits in information processing are independent of intellectual factors. Thus, vulnerability to a masking stimulus in schizophrenic persons can be attributed to the pathology of schizophrenia. This vulnerability indicates that schizophrenic patients are slow information processors. (31 references)

6640. Schneider, R.D., and Cauthen, N.R. (Calgary General Hospital, Calgary, Alberta, Canada) Locus of reaction time change in schizophrenics and normal subjects. Journal of Nervous and Mental Disease, 170(4):231-240, 1982.

Simple reaction time (RT) obtained from trials presented in isotemporal series (preparatory intervals of the same duration) were partitioned into premyographic and myographic time components for 20 schizophrenic and 20 normal subjects according to a previously developed procedure. This procedure allowed examination of the relative contributions made by these RT components to (a) overall mean reaction time and (b) the crossover pattern of responding as first observed by David Shakow. In addition, a within-subject comparison of the traditional pressure-release motor task and an alternative press-only motor task was made. Differential predictions were made on the effect of the task manipulation from (a) the loss of major set and (b) the reactive inhabitation interpretational positions. It was found that the schizophrenics' crossover pattern of responding was specific to the press-release task. Furthermore, it was apparent that both premyographic and myographic components of the RT interval contributed to (a) their overall longer mean RTs, as well as (b) their increased magnitude of crossover. The results support the view that inhibitory processes triggered by the increased levels of arousal during the isotemporal series are responsible for the schizophrenics' crossover pattern of responding on the press-release task. Loss of major set explanation is not incompatible with a reactive inhibition explanation of the crossover effect. (25 references)

Biology

6641. Adler, L.E.; Franks, R.D.; Freedman, R.; Pachtman, E.; Pecevich, M.; and Waldo, M.C. (Denver Veterans Administration Medical Center, Denver, CO 80220) Neurophysiological evidence for a defect in neuronal mechanisms involved in sensory gating in schizophrenia. Biological Psychiatry, 17(6):639-654, 1982.

The action of central nervous system inhibitory neuronal mechanisms was tested in acutely psychotic unmedicated schizophrenic patients and in normal controls. An early positive component of the auditory average evoked response recorded at the vertex 50 msec after a click stimulus was studied. Stimuli were delivered at 10-second intervals to establish a baseline response. Inhibitory mechanisms were then tested using a conditioning-testing paradigm by assessing the change in response to a second stimulus following the first at either 0.5-, 1.0-, or 2.0-second intervals. At the 0.5-second interval, normal controls had over 90 percent mean decrement in response, whereas schizophrenics showed less than a 15 percent mean decrement. At 2-second intervals, responses from normals were still 30-50 percent diminished, but those from schizophrenics showed an increased response to the stimulus compared to baseline. The data suggest that normally present inhibitory mechanisms are markedly reduced in schizophrenics. Failure of these inhibitory mechanisms may be responsible for the defects in sensory gating, an important part of the pathophysiology of schizophrenia. (29 references)

6642. Albus, M.; Ackenheil, M.; Engel, R.R.; and Muller, F. (Dept. of Psychiatry, University of Munich, Munich, West Germany) Situational reactivity of autonomic functions in schizophrenic patients. Psychiatry Research, 6(3):361-370, 1982.

In a study designed to evaluate the state of arousal and the autonomic reactivity to experimental conditions in schizophrenic patients, 12 acute, unmedicated schizophrenic patients with paranoid hallucinatory symptomatology and 63 healthy normal control subjects were administered four standardized tasks: cold pressor test, noise, mental arithmetic, and active relaxation. Biochemical (norepinephrine and cortisol) and physiological (electromyogram, electroencephalogram, skin conductance response, skin conductance level, finger pulse amplitude, finger temperature, heart rate, respiratory volume, pulse wave velocity, and electrogastrogram) parameters were measured simultaneously. Schizophrenic patients showed elevated levels of norepinephrine and cortisol, as well as heightened responsivity on measures of electromyographic activity, skin conductance level, and heart rate and reduced responsivity to conditions of stress. It is concluded that schizophrenic patients show higher nonspecific activation and reduced ability to react to external stimulation, perhaps induced by lack of inhibition of the reticular formation by the limbic system. (26 references)

6643. Andreason, N.C.; Dennert, J.W.; Olsen, S.A.; and Damasio, A.R. (Dept. of Psychiatry, University of Iowa Hospitals and Clinics, 500 Newton Rd., Iowa City, IA 52242) Hemispheric asymmetries and schizophrenia. American Journal of Psychiatry, 139(4):427-430, 1982.

Hypothesized abnormal left hemispheric function, deviations from normal functional asymmetry, or normal asymmetry reversals in schizophrenia were investigated in 43 right-handed schizophrenic patients and 40 right-handed controls. Frontal and occipital petalia and frontal and occipital width of each hemisphere on computerized tomographs were measured. There were no significant differences in structural asymmetry between the groups. Eight left-handed schizophrenics may have had abnormal functional asymmetries; their mean ventricular-brain ratio, a possible indirect indicator of cerebral atrophy, was higher than that of right-handed patients. (13 references)

6644. Arora, R.C., and Meltzer, H.Y. (Dept. of Psychiatry, University of Chicago, Pritzker School of Medicine, Chicago, IL 60637) Serotonin uptake by blood platelets of schizophrenic patients. Psychiatry Research, 6(3):327-333, 1982.

Active uptake of serotonin (5-hydroxytryptamine; 5-HT) by blood platelets of acute and chronic schizophrenic patients was compared with that of normal controls. Unlike previous reports, no significant difference in the kinetic parameters (K_m and V_{max}) of 5-HT uptake between schizophrenic patients and normal controls was observed, although a trend toward decreased V_{max} in the acute schizophrenics was present. Since decreased V_{max} of platelet 5-HT uptake has been found in patients with bipolar, unipolar, and schizoaffective depression, further study of the usefulness of platelet 5-HT uptake as a biological marker for major depressive illness is indicated. Abnormalities of 5-HT uptake do not appear to contribute significantly to the increased platelet 5-HT levels reported in schizophrenic patients. (39 references)

6645. Baron, M.; Perumal, A.S.; Levitt, M.; and Cannova, G. (New York State Psychiatric Institute, 722 W. 168th St., New York, NY 10032) Platelet monoamine oxidase in schizophrenia with beta-phenylethylamine and benzylamine as substrates. Biological Psychiatry, 17(4):479-483, 1982.

The relationship of β-phenylethylamine (PEA) concentration to platelet monoamine oxidase (MAO) activity was studied and the optimal PEA concentration for MAO assay was sought. Platelet MAO activity levels using PEA and benzylamine as substrates were examined at optimal concentrations in 10 chronic schizophrenic males and 10 age- and sex-matched normal controls. Results show that a relatively narrow range of PEA concentration yields maximal MAO activity. The significant correlations between MAO activities obtained with PEA and benzylamine and the similarity in the slopes of the regression lines in patients and controls indicate that substrate-related differences between schizophrenics and normal controls do not exist in the present population. Results reaffirm the importance of using optimal substrate concentrations in measuring platelet MAO activity. (11 references)

6646. Benes, F.; Cohen, B.M.; Jones, B.D.; Lemay, M.; Lipinski, J.F.; and Sunderland, P. (No address given) Normal ventricles in young schizophrenics. British Journal of Psychiatry, 141:90-93, 1982.

The computed tomographic scans of 11 schizophrenic patients and 26 controls were evaluated for both linear (Evan's and cella media ratios) and volume (planimetry and grid ratios) measurements of ventricular size. There were no differences between the two groups on any of the measures obtained. These results are related to previous reports showing ventricular enlargement in schizophrenics. The relatively younger age and briefer period of hospitalization in the present sample are suggested as variables possibly associated with these findings. (18 references)

6647. Bochkarev, V.K., and Panyushkina, S.V. (Lab. neyrofiziologii, Institut psikhiatrii AMN SSSR, Moskva, USSR) Study of the dynamics of the integral parameters of the background EEG during treatment with neuroleptics. Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 82(2):258-264, 1982.

The electroencephalogram dynamics of schizophrenic patients from the acute psychotic state until remission were studied by computerized component analysis during treatment with chlorpromazine, leponex, and combinations of neuroleptics, and were examined in relation to the concepts of activation processes. The possible role of those processes in the neurophysiological mechanisms of the therapeutic effect of neuroleptics is discussed. (16 references)

6648. Buchsbaum, M.S.; Ingvar, D.H.; Kessler, R.; Waters, R.N.;

Cappelletti, J.; van Kammen, D.P.; King, A.C.; Johnson, J.L.; and Manning, R.G. (National Institute of Mental Health, Section on Clinical Psychophysiology, Biological Psychiatry Branch, Bethesda, MD 20892) Cerebral glucography with positron tomography: Use in normal subjects and in patients with schizophrenia. Archives of General Psychiatry, 39(3):251-259, 1982.

Local cerebral uptake of deoxyglucose labeled with fluorine 18 was measured by positron emission tomography in eight patients with schizophrenia who were not receiving medication and in six age-matched normal controls. Subjects sat in an acoustically treated, darkened room with eyes closed after injection of 3-5 mCi of deoxyglucose 18F. After uptake, seven to eight horizontal brain scans parallel to the canthomeatal line were done. Scans were treated digitally, with a 2.3-cm strip peeled off each slice and ratios to whole slice activity computed. Patients with schizophrenia showed lower ratios in the frontal cortex, indicating relatively lower glucose use than normal controls. This was consistent with previously reported studies of regional cerebral blood flow. Patients also showed diminished ratios for a 2.3-cm square that was positioned over central gray matter areas on the left, but not on the right side. These results are only preliminary; issues of control of mental activity, brain structure identification, and biologic and anatomic heterogeneity of schizophrenia remain to be explored. (50 references)

6649. Chouinard, G.; Annable, L.; Boisvert, D.; Bradwejn, J.; Creese, I.; and Jones, B. (Allan Memorial Institute, Montreal, Quebec, Canada H3A 1A1) High neuroleptic plasma levels in patients manifesting supersensitivity psychosis. Biological Psychiatry, 17(7):849-852, 1982.

Tardive dyskinesia is believed to result from a dopamine (DA) receptor supersensitivity in the neostriatum that develops after prolonged DA receptor blockade by neuroleptic medication. It is proposed that similar changes occur in the mesolimbic pathway in response to chronic DA receptor blockade by these drugs and that a sudden return of psychotic symptoms following neuroleptic withdrawal or dosage decrease could be the clinical expression of mesolimbic postsynaptic DA receptor supersensitivity. This phenomenon is termed "supersensitivity psychosis." The use of radioreceptor assay of neuroleptics is reported in two patients manifesting symptoms of supersensitivity psychosis. (10 references)

6650. DeLisi, L.E.; Freed, W.J.; Gillin, J.C.; Kleinman, J.E.; Bigelow, L.B.; and Wyatt, R.J. (Adult Psychiatry Branch, Div. of Special Mental Health Research, NIMH, St. Elizabeths Hospital, Washington, DC 20032) *p*-Chlorophenylalanine trials in schizophrenic patients. Biological Psychiatry, 17(4):471-477, 1982.

p-Chlorophenylalanine (PCPA), an inhibitor of tryptophan hydroxylase, the enzyme involved in the rate limiting step of the synthesis of 5-hydroxytryptophan, was administered in a double-blind trial to seven actively symptomatic, chronic schizophrenic patients. The Brief Psychiatric Rating Scale (BPRS) was recorded twice daily for each patient by trained nurse raters and weekly by a research psychiatrist, all of whom were blind to the medication status of the patients. Mean total BPRS scores, as well as most individual symptom clusters, showed no significant differences between placebo and active PCPA trial periods for all seven patients as a group. It is concluded that PCPA at a dose of 3,000 mg/day did not produce significant clinical improvement in these patients despite a previous report of success. The apparent discrepancy may have resulted from differences in patient selection. Failure of PCPA to initiate clinical change after inhibition of the rate-limiting enzyme makes the serotonin and indoleamine hypothesis of schizophrenia less plausible. (33 references)

6651. Garbutt, J.C.; van Kammen, D.P.; Levine, R.A.; Sternberg, D.E.; Murphy, D.L.; Ballenger, J.; Bunney, W.E., Jr.; and Lovenberg, W.M. (National Institute of Mental Health, Bldg. 10, Rm. 4N214, 9000 Rockville Pike, Bethesda, MD 20892) Cerebrospinal fluid hydroxylase cofactor in schizophrenia. Psychiatry Research, 6(2):145-151, 1982.

One aspect of monoamine physiology in schizophrenia was examined when cerebrospinal fluid levels of pterin cofactor were measured in 11 off-medication schizophrenic patients and compared with 24 normal control subjects. No significant differences were found. Platelet monoamine oxidase activity correlates significantly with pterin levels in male schizophrenic subjects and in female controls. (29 references)

6652. Gattaz, W.F.; Gattaz, D.; and Beckmann, H. (Psychiatrische Klinik, Zentralinst. für Seelische Gesundheit, Postfach 5970, D-6800 Mannheim 1, West Germany) Glutamate in schizophrenics and healthy controls. Archiv für Psychiatrie und Nervenkrankheiten, 231(3):221-225, 1982.

Cerebrospinal fluid (CSF) glutamate levels were measured in 28 paranoid schizophrenic patients and 15 healthy controls. Fifteen of the patients were being treated with neuroleptic drugs and 13 were not receiving any drugs. No significant difference was found between glutamate level in patients not receiving neuroleptics and in controls. CSF glutamate was significantly higher in patients taking neuroleptics than in controls ($p < .001$) or in patients not receiving neuroleptics. These findings, as well as other data from the literature, indicate the enhanced levels of cerebral glutamate may be significant for the antipsychotic efficacy of neuroleptic drugs. (12 references)

6653. Gerner, R.H., and Sharp, B. (Dept. of Psychiatry and Biobehavioral Science, Neuropsychiatric Institute, University of California at Los Angeles, Los Angeles, CA 90024) CSF beta-endorphin-immunoreactivity in normal, schizophrenic, depressed, manic, and anorexic subjects. Brain Research, 237(1):244–247, 1982.

β-Endorphin immunoreactivity was measured in cerebrospinal fluid (CSF) of 75 medication-free subjects: normal, depressed, manic, schizophrenic, and anorexic subjects. No significant differences in β-endorphin immunoreactivity were found. Affinity extraction chromatography revealed β-lipotropin and β-endorphin, but no apparent precursors. Results suggest that a simple hypothesis of excess or diminished central β-endorphin immunoreactivity levels is unlikely to account for these major psychiatric disorders. (13 references)

6654. Gerner, R.H., and Yamada, T. (Dept. of Psychiatry and Biobehavioral Sciences, Neuropsychiatric Institute, University of California, Los Angeles, CA 90024) Altered neuropeptide concentrations in cerebrospinal fluid of psychiatric patients. Brain Research, 238(1):298–302, 1982.

Immunoreactive somatostatin, bombesin, and cholecystokinin (CCK) were measured in the cerebrospinal fluid of normal subjects and patients with anorexia nervosa, depression, mania, and schizophrenia. Somatostatin-like immunoreactivity was decreased in anorexic and depressed patients. Bombesin-like immunoreactivity tended to be decreased in schizophrenics. CCK-like immunoreactivity did not differ between groups. Results suggest that neuropeptides may have a role in the regulation of human behavior. (26 references)

6655. Ghadirian, A.M.; Annable L.; and Chouinard, G. (Allan Memorial Institute, Montreal, Quebec, Canada) Sexual dysfunction and plasma prolactin levels in neuroleptic-treated schizophrenic outpatients. Journal of Nervous and Mental Disease, 170(8):463–467, 1982.

A random sample of schizophrenic outpatients, 26 men and 29 women, treated with neuroleptics was selected from a long-term followup clinic. The patients rated their current sexual functioning on specially designed scales; 54 percent of male and 30 percent of female patients reported impaired sexual functioning during neuroleptic treatment, and 91 percent of female patients reported changes in menstruation. Partial correlation coefficients were calculated between sexual dysfunction score and plasma prolactin level adjusting for age. Sexual dysfunction was associated with high plasma prolactin levels in male patients but not in female patients. High prolactin tended also to be associated with menstrual disturbances. Antiparkinsonian medication was not correlated with either sexual dysfunction or menstrual disorder score. (20 references)

6656. Gulmann, N.C.; Orbaek, K.; and Wildschiodtz, G. (County Hospital Nordvang, Copenhagen, Denmark) Alteration of interhemisphere conduction through corpus callosum in chronic schizophrenia. Biological Psychiatry, 17(5):585–594, 1982.

The contralateral and ipsilateral somatosensory evoked potentials (SEP) after stimulation of the right and left median nerve were recorded in 10 patients with chronic schizophrenia and in 10 normal subjects. The ipsilateral SEP depends on the function of the corpus callosum. The latency-delay from peak 5 in the contralateral SEP to the analogous peak in the ipsilateral SEP was significantly reduced in the patients, but only from the left to the right hemisphere. This peak presumably represents interhemisphere transfer and function of corpus callosum. The amount of synaptic activity—measured by the mean amplitude of the SEP—in the right hemisphere, relative to the activity in the left hemisphere after stimulation of the right median nerve, was also greater in the patients. These differences could be interpreted as an abnormal function of the corpus callosum and as a breakdown of the lateral specialization of the left cerebral hemisphere in schizophrenia. (21 references)

6657. Iacono, W.G. (University of British Columbia, Vancouver, British Columbia, Canada) Bilateral electrodermal habituation-dishabituation and resting EEG in remitted schizo-

phrenics. Journal of Nervous and Mental Disease, 170(2):91-101, 1982.

This study examined bilateral electrodermal responding, heart rate, and resting electroencephlogram (EEG) in schizophrenic patients who were not psychotic at the time of testing. Twenty-four carefully diagnosed, remitted schizophrenics and 22 medical outpatient controls were exposed to 17 pure tones; the 16th tone differed in frequency and duration from the others. Before the tone series, subjects engaged in 2 minutes of respiratory maneuvers followed by a 5-minute rest period during which EEG was recorded. Schizophrenic patients divided evenly into responding and nonresponding groups and skin conductance responding was associated with a higher rate of spontaneous activity, elevated tonic conductance levels, and more response during the respiratory exercises. When those responding in the control and schizophrenic groups were compared for number and amplitude of tone-elicited responses, no group differences emerged. There were no differences in dishabituation between these two groups and a variety of analyses failed to reveal any evidence of electrodermal or cerebral asymmetry. There were no differences between the two schizophrenic groups in measures of heart rate or resting EEG; however, the schizophrenic patients as a group produced less EEG α and more δ than the normal subjects. These results identified in psychotic, hospitalized patients represent stable traits characteristic of schizophrenia. (66 references)

6658. Jankovic, B.D.; Horvat, J.; and Jakulic, S. (Immunology Research Center, Belgrade, Yugoslavia) Delayed skin hypersensitivity reactions to human brain S-100 protein in psychiatric patients. Biological Psychiatry, 17(6):687-697, 1982.

To confirm and extend previous observations of the correlation between cell-mediated immunity and psychiatric diseases, 511 patients with schizophrenia, cerebral atrophy, dementia, depression, and mental retardation, and 32 control subjects and 27 control patients were skin-tested with human brain S-100 protein and human liver protein. Of psychiatric patients tested, 70.2-93.1 percent developed positive skin hypersensitivity reactions to S-100 protein, while 2.8-20.7 percent of patients reacted to liver protein. Of control subjects, 6.2-7.4 percent responded to S-100 protein, and 7.4-9.4 percent to liver protein. The findings indicate that cell-mediated immune processes may be involved in brain mechanisms underlying cerebral atrophy, depression, schizophrenia, and mental retardation. (50 references).

6659. Jimerson, D.C.; van Kammen, D.P.; Post, R.M.; Docherty, J.P.; and Bunney, W.E., Jr. (National Institute of Mental Health, Biological Psychiatry Branch, Building 10, Room 2D46, 9000 Rockville Pike, Bethesda, MD 20892) Diazepam in schizophrenia: A preliminary double-blind trial. American Journal of Psychiatry, 139(4):489-491, 1982.

A preliminary double-blind trial of high-dose diazepam (250-300 mg/day) was conducted in five schizophrenic and one schizoaffective inpatients. The schizoaffective patient and one of two schizophrenic patients showed limited decreases in psychosis ratings. In the three other patients, sedation and ataxia necessitated limitation of the diazepam dose. These data, together with the lack of success with γ-amino-butyric acid (GABA) agonist administration in schizophrenia, suggest that currently available agents which act at the diazepam and GABA receptors or their complex are not notably effective antipsychotic agents. (10 references)

6660. Jorgensen, O.S.; Goldschmidt, V.V.; and Vestergaard, B.F. (Dept. of Child Psychiatry, Rigshospitalet, 9 Blegdamsvej, DK-2100 Copenhagen, Denmark) Herpes simplex virus (HSV) antibodies in child psychiatric patients and normal children. Acta Psychiatrica Scandinavica, 66(1):42-49, 1982.

The prevalence of herpes simplex virus (HSV) antibodies has been investigated in 123 child psychiatric patients and 86 normal children. HSV antibodies were measured by the enzyme-linked immunosorbent assay (ELISA) technique. The prevalence of HSV antibodies in different diagnostic groups (conduct disorder, emotional disorder, hyperkinetic syndrome, anorexia nervosa, infantile autism, and borderline schizophrenia in childhood) was compared with age-matched normal children, but no significant differences were found. (19 references)

6661. Kaiya, H.; Nakamura, S.; Namba, M.; and Yoshida, H. (Dept. of Neuropsychiatry, Gifu University School of Medicine, Gifu, Japan) Plasma glutamate decarboxylase activity in neuropsychiatry. Psychiatry Research, 6(3):335-343, 1982.

Plasma glutamate decarboxylase (GAD) activity was measured in patients with endogenous psychoses and neurologic diseases. Unmedicated schizophrenic patients showed no difference in plasma GAD levels compared to controls. Administration of neuroleptics together with anticholinergic agents increased plasma GAD activity in schizophrenic patients. Compared to controls, patients with major depression and bipolar illness showed significantly lower GAD activity. Antidepressants and minor tranquilizers had no effect on plasma GAD activity. Relatively lower GAD activity was shown in neurotic patients. The enzyme activity in plasma of patients with Huntington's Chorea (HC) was lower than control levels. The plasma GAD concentrations correlated with cerebrospinal fluid concentrations in five HC patients. (33 references)

6662. Kemali, D.; Del Vecchio, M.; and Maj, M. (Clinica Psichiatrica, Prima Facoltà di Medicina e Chirurgia, Università di Napoli, Piazza Mizaglia 2, i-80138 Naples, Italy) Increased noradrenaline levels in CSF and plasma of schizophrenic patients. Biological Psychiatry, 17(6):711-717, 1982.

Noradrenaline (NA), dopamine (DA), and adrenaline (A) concentrations were determined by means of a specific, sensitive radioenzymatic method in plasma, cerebrospinal fluid (CSF), and urine of schizophrenic patients and in a group of age- and sex-matched controls with no history of mental disorder. NA levels were significantly increased both in plasma and CSF of schizophrenics, particularly in paranoids. The increase in plasma NA concentration can probably be ascribed to the higher unspecific arousel level of these patients, whereas the increase in CSF NA levels probably reflects a rise of central noradrenergic activity. The physiopathological significance of this overactivity is questionable. (31 references)

6663. Kinnell, H.G.; Kirkwood, E.; and Lewis, C. (Botley's Park Hospital, Chertsey, Surrey KT16 0QA, England) Food antibodies in schizophrenia. Psychological Medicine, 12(1):85-89, 1982.

The validity of the suggestion that food allergy is relevant to the manifestation of schizophrenic illness was investigated. Antibodies to oats, wheat gluten, chicken, calf, and milk protein were measured in 98 schizophrenic patients and in 90 unaffected close blood relatives. The majority of subjects were male outpatients on neuroleptic medication. Antibodies were detected in only a minority of patients. (25 references)

6664. Luchins, D.J. (University of Chicago, Chicago, IL 60637) Developments in biological psychiatry: Clinical applications to the major psychoses. Hospital & Community Psychiatry, 33(5):355-361, 1982.

Recent developments in biological psychiatry offer clinicians the opportunity to apply new techniques to the diagnosis and treatment of mental disorders. For instance, the dexamethasone suppression test has been used in the differential diagnosis of depression, and high doses of propranolol have been reported to improve schizophrenic symptoms. Studies now suggest that lithium may be equally effective in treating schizoaffective and affective disorders. Receptor binding techniques provide new information on the mechanism of action of psychotropic drugs and allow the clinician to assess the likelihood of specific side effects of drugs within a class. Other recent developments include the use of computered tomography with patients who exhibit schizophrenic behavior. (66 references)

6665. Meldrum, B. (Dept. of Neurology, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, England) GABA and acute psychoses. Psychological Medicine, 12(1):1-5, 1982.

Biochemical and pharmacological observations relevant to a role of γ-amino-butyric acid (GABA) in schizophrenia and acute psychoses are reviewed. Overall, clinical studies of GABA agonists or GABA-mimetics show no therapeutic actions against the positive symptoms of acute schizophrenia and, thus, provide no support for the GABA deficiency hypotheses of schizophrenia. By contrast, high doses of drugs known to act at GABA receptors other than the receptor responsible for postsynaptic inhibition all are capable of inducing acute psychoses, which vary from an apparent exacerbation of schizophrenia syndrome in patients with acute schizophrenia to an organic confusional state in patients with a variety of neurological disorders. Thus, an excessive local release of GABA, or hypersensitivity of a subgroup of GABA receptors, or an endogenous GABA agonist might be responsible for some features of acute psychotic syndromes. (59 references).

6666. Meltzer, H.Y.; Busch, D.A.; Robertson, A.; and Tricou, B.J. (Department of Psychiatry, University of Chicago, Chicago, IL 60637) Effect of (des-tyr)-gamma-endorphin in schizophrenia. Psychiatry Research, 6(3):313-326, 1982.

Des-tyrosine-γ-endorphin (DTγE), a derivative of γ-endorphin which has been reported to have some neuroleptic-like properties, was administered to eight hospitalized schizophrenic patients (six chronic, one subacute, one acute) in an open-study. Following an initial drug-free period, patients were given DTγE for 12 days in doses ranging from 1 to 10 mg/day. Two of the patients were markedly improved after receiving DTγE. The improvement was sustained for 2 months in one subject, while the other deteriorated to pretreatment status within 48 hours of the discontinuation of DTγE. Of the other six patients, one showed moderate improvement, three showed minimal improvement, and two showed no change. Improvement was mainly in the area of social functioning; change in positive psychotic symptoms was less noticeable. The positive results obtained in this study in some subjects could have been nonspecific effects, rather than pharmacological action, since social functioning, the main area of improvement, may be especially sensitive to expectancy effects in open trials. Nevertheless, further study of DTγE in acute schizophrenics for longer periods appears indicated. (28 references)

6667. Merrin, E.L. (University of California, San Francisco, School of Medicine, San Francisco, CA 94122) An evaluation of evidence for dominant lobe dysfunction. Journal of Nervous and Mental Disease, 169(7):405-416, 1981.

The literature implicating a disturbance located in the left cerebral hemisphere in the pathogenesis of schizophrenia is reviewed. Studies of the psychiatric sequelae of lateralized organic cerebral disease were considered, as well as neurological, neurophysiological, and neuroanatomic studies of psychiatric patients. In general, abnormalities in cerebral hemispheric asymmetry appear to be common among psychiatric patients, particularly those with personality disorders, some affective disorders, childhood autism, and at least some schizophrenia including milder, less chronic illnesses. A specific relationship between the side of unilateral cerebral impairment and diagnosis is not consistently supported by the literature, however. The implications of these findings are discussed in terms of neurodevelopmental issues in schizophrenia. (157 references)

6668. Mintz, M.; Myslobodsky, M.S.; and Tomer, R. (Tel Aviv University, Ramat Aviv 69978, Israel) Neuroleptic-induced lateral asymmetry of visual evoked potentials in schizophrenia. Biological Psychiatry, 17(7):815-828, 1982.

Electrophysiological studies have shown asymmetric brain reactivity in schizophrenia, supporting the hypothesis of left hemisphere hyperarousal in this disease. It may be assumed, therefore, that neuroleptic treatment restores hemispheric balance in schizophrenic patients by selectively suppressing the hyperactive left hemisphere. This hypothesis was tested by comparing bilaterally recorded visual evoked potentials (VEP) in 9 nonmedicated patients, 29 schizophrenics treated with various neuroleptic drugs, and 34 normal controls. In medicated patients, later VEP components showed enhancement over the right hemisphere as a function of the overall dose (chlorpromazine equivalent) of neuroleptics. Reversed asymmetry was seen in drug-free patients. In view of this result, some amendments to the concept of left hemisphere hyperactivity in schizophrenia are proposed. (41 references)

6669. Nasrallah, H.A.; McCalley-Whitters, M.; and Jacoby, C.G. (Psychiatry Service, VA Medical Center, Iowa City, IA 52240) Cerebral ventricular enlargement in young manic males: A controlled CT study. Journal of Affective Disorders, 4(1):15-19, 1982.

Computed tomographic head scans of 24 young manic males were compared with those of 27 matched control subjects, and 55 chronic schizophrenic patients to compare cerebral ventricular size. Cerebral ventricular size was significantly larger in the manic and schizophrenic patients, compared to controls. There was no difference in size between the manic and schizophrenic groups, suggesting that cerebral ventricular enlargement may be a nonspecific neuroanatomical correlate of psychotic disorders. It is suggested that family history of psychosis, past history of perinatal brain injury, infantile encephalitis or head injury, and electroencephalogram abnormalities should be recorded on all patients with mania or schizophrenia. (14 references)

6670. Osborne, M.; Crayton, J.W.; Davis, J.M.; and Javaid, J. (University of Chicago, Chicago, IL 60601) Lack of effect of a gluten-free diet on neuroleptic blood levels in schizophrenic patients. Biological Psychiatry, 17(5):627-629, 1982.

This study examined the long-term effect of a gluten-free diet on blood levels of the neuroleptic butaperazine in five chronic schizophrenic patients. Patients who had failed to respond to traditional neuroleptic treatment were placed on a constant 80-160 mg/day dose of butaperazine for the duration of the study. (9 references)

6671. Puente, A.E.; Heidelberg-Sanders, C.; and Lund, N. (Dept. of Psychology, University of North Carolina, Wilmington, NC 28406)

Detection of brain damage in schizophrenics as measured by the Whitaker Index of Schizophrenic Thinking and the Luria-Nebraska Neuropsychological Battery. Perceptual and Motor Skills, 54(2):495–499, 1982.

Neuropsychological performance of schizophrenics with and without brain damage was measured by the Whitaker Index of Schizophrenic Thinking and the Luria-Nebraska Neuropsychological Battery. Inpatient volunteers (11 brain damaged and 15 nondamaged) with a diagnosis of schizophrenia were administered both tests individually. Results indicate that none of the five scores of the Whitaker differentiated between the two groups. Of the 14 summary scales of the Luria-Nebraska, 8 differentiated between groups: motor, tactile, visual, writing, arithmetic, right hemisphere, left hemisphere, and pathognomic scales. While cognitive variables, as measured by the Whitaker, may be impaired further with organicity, other variables measured by the Luria-Nebraska were more effective in differentiating between groups. (6 references)

6672. Schubert, D.S.P.; Brocco, K.; Miller, F.T.; and Patterson, M. (Dept. of Psychiatry, Cleveland Metropolitan General Hospital, 3395 Scranton Rd., Cleveland, OH 44109) Serum creatine phosphokinase levels in patients meeting the St. Louis Research Diagnostic Criteria for schizophrenia. American Journal of Psychiatry, 139(4):491–493, 1982.

Serum creatine phosphokinase levels were examined in 18 patients meeting St. Louis Research Diagnostic Criteria (RDC) for schizophrenia and 36 comparison subjects with other RDC diagnoses. Mean initial serum creatine phosphokinase levels of schizophrenics were significantly higher than those of subjects in other diagnostic groups. There were no corresponding elevations in the serum levels of nondrinking alcoholics or patients with primary or secondary affective disorders. It is suggested that increased creatine phosphokinase levels in schizophrenic patients may be due to a sudden increase in the substance in the dopaminergic or limbic system due to increased muscle membrane permeability or to sympathetic nervous system or adrenergic dominance. (12 references)

6673. Schweitzer, L. (Baylor College of Medicine, Houston, TX 77025) Evidence of right cerebral hemisphere dysfunction in schizophrenic patients with left hemisphere overactivation. Biological Psychiatry, 17(6):655–673, 1982.

A number of laterality studies using initial lateral eye movements as a measure of unbalanced frontal hemisphere activity have demonstrated that schizophrenics look to their right more often than normals or depressives when thinking about spatial and/or emotional material. This increase in right gaze responses represents increased left hemisphere responsiveness for spatial and emotional material. Thus, it was predicted that schizophrenics would be more receptive to their left hemisphere and would detect rapidly appearing spatial stimuli presented to their left hemisphere more accurately than to their right hemisphere. Further, it was expected that schizophrenics would perform better than controls when identifying rapidly presented shapes in the left hemisphere. Using visual half-field presentation of verbal and spatial material, the experimental prediction of increased left hemisphere accuracy for shape detection within the schizophrenic group was corroborated. However, there was an unanticipated, significant right hemisphere deficit for spatial identification between the schizophrenic group and the normal controls, suggesting that schizophrenics may have a primary deficit in their right hemisphere. The apparent left hemisphere increase in activity for spatial material reported by Gur and Schweitzer may thus be a compensatory mechanism for a primary failure of the schizophrenic's right hemisphere to maintain normal attention and vigilance. (26 references).

6674. Sternberg, D.E.; Charney, D.S.; Heninger, G.R.; Leckman, J.F.; Hafstad, K.M.; and Landis, D.H. (Connecticut Mental Health Center, 34 Park St., New Haven, CT 06519) Impaired presynaptic regulation of norepinephrine in schizophrenia: Effects of clonidine in schizophrenic patients and normal controls. Archives of General Psychiatry, 39(3):285–289, 1982.

Recent studies which have found elevated levels of norepinephrine (NE) in cerebrospinal fluid and brain specimens from schizophrenics are discussed. Presynaptic inhibitory α-2-adrenergic receptors regulate NE release in the brain. Schizophrenic patients and age-matched normal controls were used to test the hypothesis that the functional sensitivity of this presynaptic regulation of NE is impaired in schizophrenia. The ability of clonidine, an α-2 agonist, to lower plasma levels of the NE metabolite 3-methoxy-4-hydroxyphenylglycol (MHPG) and to lower blood pressure (BP) in patients and controls was evaluated. Clonidine produced a significant decrease in plasma MHPG levels in controls, but

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not in schizophrenic subjects. It decreased BP in both groups. The results suggest that there is a functional subsensitivity of the inhibitory presynaptic α-2-adrenergic receptor in schizophrenia which may relate to an impaired regulation of NE turnover. (67 references)

6675. van Kammen, D.P.; Docherty, J.P.; Marder, S.R.; Rayner, J.N.; and Bunney, W.E., Jr. (National Institute of Mental Health, Bldg. 10, Room 4N-214, 9000 Rockville Pike, Bethesda, MD 20892) Long-term pimozide pretreatment differentially affects behavioral responses to dextroamphetamine in schizophrenia: Further exploration of the dopamine hypothesis of schizophrenia. Archives of General Psychiatry, 39(3):275-281, 1982.

In 10 of 30 schizophrenic subjects treated with pimozide for 5 weeks, 20 mg of dextroamphetamine sulfate induces an increase in synthesis. The number of patients becoming more psychotic with the dextroamphetamine challenge was not significantly different from the number who worsened after dextroamphetamine challenge when pretreated with placebo. Half of the subjects who showed a psychotic response to dextroamphetamine during placebo pretreatment responded to dextroamphetamine with an increase in psychosis after pimozide treatment. Dextroamphetamine induced a worsening in subjects who had improved with pimozide. The stability of the preinfusion condition was more important to the type of response to dextroamphetamine than long-term pretreatment with a dopamine receptor blocker. The activation euphoria response to dextroamphetamine was unaffected by pimozide pretreatment. This suggests that the change in psychosis and activation may be regulated by different mechanisms. The findings are seen as questioning the postulated relationship between the antipsychotic drug response and dopamine receptor blockade. (102 references)

6676. van Kammen, D.P.; Docherty, J.P.; Marder, S.R.; Schulz, S.C.; Dalton, L.; and Bunney, W.E., Jr. (National Institute of Mental Health, Bldg. 10, Room 4N214, 9000 Rockville Pike, Bethesda, MD 20892) Antipsychotic effects of pimozide in schizophrenia: Treatment response prediction with acute dextroamphetamine response. Archives of General Psychiatry, 39(3):261-266, 1982.

Acute behavioral response to 20 mg of dextroamphetamine i.v. predicts week 4 antipsychotic response to double-blind pimozide treatment. Patients whose psychotic condition improved with dextroamphetamine administration showed more antipsychotic response to pimozide therapy than those whose condition worsened or did not change. Multiple regression analysis indicated amphetamine-induced response predicted pimozide response after 4 weeks, but week 5 pimozide response was more accurately predicted by prepimozide psychosis ratings. The results provide evidence that mechanisms underlying early and late pimozide response are not necessarily identical. Because patients who did not respond to dextroamphetamine administration still improved with pimozide therapy, the data do not support the concept that schizophrenia can be divided into two groups (dopamine sensitive or dopamine insensitive) but instead suggest that dopamine responsiveness changes over time. Clinical application is not warranted until studies with larger samples replicate these findings. (49 references)

6677. Watanabe, M.; Funahashi, T.; Nakazawa, T.; Noguchi, T.; Nomura, S.; Suzuki, T.; and Tsukada, Y. (Fujita-Gakuen University School of Medicine, 1-98 Dengakugakubo, Kutsu-kake-cho, Toyoake, Aichi 470-11, Japan) Antithymic antibodies in schizophrenic sera. Biological Psychiatry, 17(6):699-710, 1982.

Sera from normal controls and schizophrenics were examined for antithymic activity, employing a cytotoxicity test with C3H mouse thymocytes. The average level of antithymic activity in schizophrenics was considerably higher than that of controls. The antithymic activity in newly admitted unmedicated patients was not statistically different from that of hospitalized medical patients. There was no difference in antithymic activity within the hebephrenic, catatonic, and paranoid subdivisions of schizophrenia. The antithymic titer also did not correlate with the psychopathological status as assessed by the Brief Psychiatric Rating Scale total score. The antithymic activity against C3H mouse thymocytes in both the schizophrenic and control sera was completely absorbed with mouse brain tissue homogenate (where Thy-1 antigen was present) and liver homogenate (where Thy-1 was absent), but not with human brain and liver. It seems unlikely, therefore, that antithymic factor in human sera contains antibodies against Thy-1 (brain-associated thymic) antigen. Antithymic activity is not considered specific for schizophrenic illness, and the high antithymic activity found in schizophrenics might be produced against unknown xenoantigens, probably as a result of a nonspecific dysfunction of the immunological system in such patients. (25 references)

Case Studies

6678. Fromm-Reichmann, F. (No address given) Frieda Fromm-Reichmann discusses the "Rose Garden" case. Psychiatry: Journal for the Study of Interpersonal Processes, 45(2):128-136, 1982.

Fromm-Reichmann's successful psychoanalytic treatment of a schizophrenic adolescent female, who later wrote "I Never Promised You a Rose Garden" under the name of Hannah Green, is discussed in a 1956 lecture given at the Ypsilanti Psychiatric Institute. The patient was admitted to the hospital following 2 years of psychiatric treatment for self-mutilation, hallucinations, and fear of persecution by men. Treatment was based on the assumption that the patient must accept illness as an expression and part of the self, and must befriend it and integrate it like other experiences. The clinical application of this assumption is illustrated in anecdotal materials concerned with the patient-analyst relationship, the patient's relationship with her family, her loneliness, her fantasies, her difficulties with the female sexual role, and the significance and motivations of her compulsive over-eating and self-mutilation. At the end of treatment, the patient had shown great improvement; she went on to write her own treatment history, made friends, married, and started a family.

6679. Moss, H.B., and Green, A. (8922 Fleetwing Ave., Los Angeles, CA 90045) Neuroleptic-associated dysphagia confirmed by esophageal manometry. American Journal of Psychiatry, 139(4):515-516, 1982.

A case is described of neuroleptic-associated dysphagia in a chronic schizophrenic male, in whom manometry was used to document changes in esophageal motility during manipulation of the patient's medication. Upper esophageal dystonia was the etiology most consistent with manometric findings. As neuroleptic-associated dysphagia could be a potentially fatal side effect of pharmacotherapy for schizophrenia, further research to elucidate the epidemiology and pathophysiology of this phenomenon is needed. (10 references)

6680. Pies, R. (Dept. of Psychiatry, State University of New York Upstate Medical Center, 750 East Adams St., Syracuse, NY 13210) Capgras phenomenon, delirium, and transient hepatic dysfunction. Hospital & Community Psychiatry, 33(5):382-383, 1982.

A case is presented which shows that Capgras phenomenon may occur in patients with superimposed functional and organic mental disorders, that specific pharmacologic and pharmacokinetic factors may contribute to the syndrome, and that the delusion of doubles may persist in attenuated form even after the primary organic brain syndrome has subsided. While the Capgras syndrome appeared in the context of a mixed organic-functional illness, the syndrome was clearly separable from the patient's chronic pathology. The case demonstrates the need for sophisticated prescription of psychotropics, and the potential hazards of polypharmacy. Finally, the patient's hepatic dysfunction ought to remind clinicians that the metabolism and pharmacokinetics of psychotropic drugs must be carefully considered (7 references)

6681. Roth, L.H.; Appelbaum, P.S.; Huber, G.; Reynolds, C.F.; and Sallee, R. (University of Pittsburgh School of Medicine, Pittsburgh, PA 15261) The dilemma of denial in the assessment of competency to refuse treatment. American Journal of Psychiatry, 139(7):910-913, 1982.

The case of a woman with paranoia who denied she was ill and refused medication is presented to illustrate the dilemma in evaluating and treating patients who deny their illness. The issue of whether a patient who denies his or her illness is truly legally competent to refuse or consent to treatment is examined. It is concluded that the assessment of such competency requires a consideration of the accuracy of the patient's "appreciation of the nature of his or her situation" and that safeguards need to be built into a system for evaluating competency along these lines to prevent abuse. (13 references)

Cross-Cultural Studies

6682. Kauders, F.R.; Hammond, K.W.; and MacMurray, J.P. (Loma Linda University Medical Center, Loma Linda, CA 92354) Male predominance among Palauan schizophrenics. International Journal of Social Psychiatry, 28(2):97-102, 1982.

Data gathered in a 3-month field study of known schizophrenics in the Palau Islands reveal a 4:1 ratio of males to females. It is postulated that relatively rapid sociocultural changes over the last two decades have loosened traditional external restraints on behavior, destabilized male role and gender identities, and created an environment that has been particularly schizophrenogenic for males. Statistical analysis of the age distribution of the schizophrenic patients is used to support the hypothesis. Evidence is also presented indicating that the gender distribution is not artifactual. (15 references)

6683. Ona, P.A. (University of Benin, Benin City, Nigeria) Cultural relativity of first rank symptoms in schizophrenia. Journal of Social Psychiatry, 28(2):91–95, 1982.

This study assessed the extent and usefulness of Schneider's first-rank symptoms in the diagnosis of schizophrenia from a cross-cultural point of view. Case notes of patients admitted to the mental health ward of the University of Benin teaching hospital, Benin City, Nigeria, were analyzed. Of 78 schizophrenic patients studied, 47 (60.3 percent) had first-rank symptoms. This retrospective study attempts to replicate the studies of Mellor and Zarrouk to determine the following: (1) To what extent are first-rank symptoms manifested by schizophrenic patients in Nigeria? (2) Are first-rank symptoms an uncommon occurrence in a Nigerian population? (3) Is there a difference in occurrence of symptoms in the Nigerian population, compared with Mellor's European studies and studies by Zarrouk? (4) What is the cross-cultural validity of Schneider's first-rank symptoms in the diagnosis of schizophrenia? (10 references)

6684. Yaroush, R.A. (Institute of Cognitive Science, Box 345, University of Colorado, Boulder, CO 80309) Application of the Whitaker Index of Schizophrenic Thinking to a non-English-speaking population. Journal of Clinical Psychology, 38(2):244–252, 1982.

A German version of the Whitaker Index of Schizophrenic Thinking (WIST) was administered to West Germans diagnosed as acute or chronic schizophrenics and to groups of psychiatric and normal controls. The index values on the WIST differentiated the performance of the two schizophrenic groups from normal controls. Acute patients (unlike chronic schizophrenic patients) improved significantly from one test administration to the second. These results replicate the outcome of the original standardization study. The usefulness of the WIST definition of schizophrenic thinking which is based on nonlinguistic, cognitive variables, and the applicability of the WIST for cross-cultural diagnosis are discussed. (11 references)

Description

6685. Adebimpe, V.R.; Chu, C.C.; Klein, H.E.; and Lange, M.H. (University of Missouri, Columbia School of Medicine, Columbia, MO 65201) Racial and geographic differences in the psychopathology of schizophrenia. American Journal of Psychiatry, 139(7):888–891, 1982.

The psychopathology of black and white and of rural and urban schizophrenic patients is compared. Using a structured interview, psychiatrists rated 273 schizophrenic patients consecutively admitted to seven hospitals and mental health centers over 3½ years. Results indicate that important symptoms were more severe in black than in white schizophrenic patients: black patients were more angry, impulsive, hallucinating, dysphoric, and asocial. A greater number of important symptoms were found to be more intense in rural than in urban schizophrenic patients: rural patients were more angry, aggressive, silly, negativistic, and uncooperative; but urban patients were more anxious, rigid, ambivalent, and asocial. (16 references)

6686. Burnham, R.T. (No address given) Emotional styles in process-reactive schizophrenics. Dissertation Abstracts International, 42(10): 4183-B, 1982.

The emotional styles of process and reactive schizophrenics were compared using 60 hospitalized schizophrenic patients. Upon admission, process schizophrenics were more depressed, hostile, anxious, distrustful, and withdrawn than reactive schizophrenics. Both groups changed as a function of time in the hospital but reactive schizophrenics changed more rapidly. On discharge, process schizophrenics, despite significant changes, were still described as unsociable, withdrawn, distrustful, and depressed while reactive schizophrenics more closely resembled the norm. The emotions profile index was shown to differentiate process from reactive schizophrenia upon admission with 75 percent accuracy.

6687. Gardos, G.; Cole, J.O.; and Labrie, R.A. (McLean Hospital, Belmont, MA 02178) A 12-year follow-up study of chronic schizophrenics. Hospital & Community Psychiatry, 33(12):983–984, 1982.

Data from a 12-year followup study by the Boston State Hospital cohort of a National Institute of Mental Health-sponsored collaborative study of high-dose chlorpromazine for chronic schizophrenia is summarized. Followup was considered particularly valuable because the initial population was relatively homogeneous and representative of physically healthy, young, chronically hospitalized schizophrenic patients; repeated measures of symptoms and social adjustment spanning 12 years were available; and the cohort was involved in the deinstitutionalization movement during the followup period. (5 references)

6688. Higdon, J.F. (Psychiatry Department (Medical Psychology), University of Missouri, Columbia, MO 65201) Roles of power, sex, and inadequate attractiveness in paranoid women. Psychological Reports, 50(2):399-402, 1982.

Six Thematic Appreception Test cards, Leary Interpersonal Check Lists, and questionnaires about sexual attitudes were administered to 10 paranoid schizophrenic females, 10 nonparanoid schizophrenic females, and 10 nonschizophrenic females. The subjects did not differ significantly on preoccupations with power, homosexuality, or sex-role confusion, except that paranoids attributed significantly greater power to themselves and schizophrenics attributed significantly greater power to men. The paranoid phenomenon in females, then, does not represent a mirror image of the paranoid phenomenon involving power conflict in males. Rather, in females, the paranoid phenomenon may result from some other factor, such as a feeling of deficiency in feminine attractiveness (i.e., the hypothesis of sex-role inadequacy by Wolowitz). (9 references)

6689. Hirsch, S.R. (No address given) Depression "revealed" in schizophrenia. British Journal of Psychiatry, 140:421-423, 1982.

The association of depressive symptoms with schizophrenia is considered, and recent studies of this relationship are examined. Three types of depression associated with schizophrenia are discussed: pharmacogenic depression, akinetic depression, and postpsychotic depression. Studies which investigated the course of depressive symptoms in schizophrenia are cited. Results show a high frequency of depressive symptoms in schizophrenia, occurring whether patients are on neuroleptic medication or not, which tend to recur and to remit after treatment has begun. These findings suggest that depressive symptoms are an integral part of the schizophrenic syndrome and must share with schizophrenia common pathophysiological processes. The need for examination of the underlying relationship between depression and schizophrenia is required. (13 references)

6690. Howard, M.E. (University of Missouri at Columbia, Columbia, MO 65201) Effects of drinking duration, aging, and schizophrenia diagnosis on neuropsychological test performance of alcohol abusers. Dissertation Abstracts International, 42(9):3824-B, 1982.

The effects of duration of problem drinking, aging, and the presence of a diagnosis of schizophrenia on the neuropsychological test performance of hospitalized alcohol abusers was studied. One hundred and eighty male inpatient alcohol abusers were assigned to one of 12 experimental groups categorized by duration of drinking (long and short), age (younger, middle-aged, and older), and diagnosis of schizophrenia (present or not). Test performance of subjects was measured by factor scores from factor analysis of Swiercinsky's Modified Halstead-Reitan Neuropsychological Battery, scores on nine tests from the battery which are related to alcohol abuse, and Wechsler Adult Intelligence Scale performance and verbal intelligence differences. Alcohol abusers showed significantly altered neuropsychological test performance in association with duration of problem drinking and age, but not with diagnosis of schizophrenia. Factor scores were found to be more useful in identifying the differential effects of duration of problem drinking and aging than the test scores. Alcohol abusers show some neuropsychological deficits similar to premature aging; however, alcohol abuse impairs verbal memory function while aging does not.

6691. Jauhar, P., and Weffer, M.P.I. (No address given) Psychiatric morbidity and time zone changes: A study of patients from Heathrow Airport. British Journal of Psychiatry, 140:231-235, 1982.

In a 2-year period, 186 patients were admitted from Heathrow Airport to the nearest psychiatric hospital. Affective illness was related to time zone change. Depression was diagnosed significantly more often on flights from east to west; east to west versus west to east; and north to south, combined with south to north versus east to west. Hypomania was inversely related to depression in an east-to-west comparison. No other associations with direction of travel were seen in other diagnoses. Ninety-three patients (50 percent) were diagnosed as schizophrenic; 24 of these had been aimlessly wandering. Twenty patients had been admitted at least once before under similar circumstances. Schizophrenic patients from Heathrow constituted 20 percent of the total number of schizophrenic patients admitted to the hospital during the study period. (20 references)

6692. Leiter, E. (Bronx Psychiatric

Center, Bronx, NY 10461) The effects of subliminal activation of aggressive and merging fantasies in differentiated and non-differentiated schizophrenics. Psychological Research Bulletin, 22(7):1-21, 1982.

Sixty schizophrenic patients who had been initially assessed on several measures of differentiation were exposed to three conditions in a subliminal psychodynamic activation experiment. One condition was designed to activate aggressive fantasies, a second symbiotic fantasies, and the third was a neutral control condition. The major results showed that (1) the aggressive condition intensified pathology; (2) the symbiotic condition reduced pathology for the more differentiated schizophrenics but increased it for the less differentiated: the criterion score for degree of differentiation came from the adjective rating scale and is a measure of sense of separateness; (3) the other more general measures of differentiation from the rod and frame test, embedded figures test, Rorschach and figure drawings did not predict performance after the symbiotic condition. (35 references)

6693. Manschreck, T.C.; Maher, B.A.; Rucklos, M.E.; and Vereen, D.R. (Dept. of Psychiatry, Massachusetts General Hospital, Fruit St., Boston, MA 02114) Disturbed voluntary motor activity in schizophrenic disorder. Psychological Medicine, 12(1):73-84, 1982.

Disturbances in voluntary motor activity and their relationship to assessed features of schizophrenic disorder (subtype diagnosis, formal thought disorder) were investigated in schizophrenic and affective symptomatology, neurological soft signs, delusions, and affective disorder patients. Subjects receiving neuroleptic drugs were examined carefully for side effects to ensure that such effects were not mistaken for disturbances associated with the schizophrenic disorder itself. Results indicate that voluntary motor activity disturbances that are not attributable to side effects or known neurological disorder occurred in virtually all cases of conservatively defined schizophrenia. The observed abnormalities were short lived; clumsiness was the most frequently observed anomaly. Affective blunting and neurological soft signs also correlated strongly with disturbed motor features. There was a significant and positive association of motor abnormality and disturbances in thought, a finding congruent with the attention deficit hypothesis of schizophrenic thought disorder. Thus, certain thinking and motor abnormalities in schizophrenics may have a common pathogenetic basis. (42 references)

6694. Marsden, C.D. (Dept. of Neurology, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, England) Motor disorders in schizophrenia. Psychological Medicine, 12(1):13-15, 1982.

The Manschreck et al. (1982) study of disturbed motor behavior in schizophrenia is critically discussed. Manschreck et al. claim that disturbances in voluntary motor activity not attributable to drug effect or known neurological disorder occur in virtually every case of conservatively defined schizophrenia; they view this finding as compatible with the attention deficit hypothesis which predicts that motor and thought disturbance should coincide in schizophrenia. The methods employed to detect these so-called motor disturbances are discussed, and attention is drawn to: (1) the uncertainty of claims that the sorts of florid motor disturbances now associated with organic brain disease existed in schizophrenic patients before the advent of neuroleptic treatment; (2) the subtle role of neuroleptic drugs in causing voluntary motor activity disturbances; and (3) difficulties inherent in using apparently validated rating scales as objective methods of assessing motor disorder. (5 references)

6695. Mellor, C.S. (No address given) The present status of first-rank symptoms. British Journal of Psychiatry, 140:423-424, 1982.

The clinical value of first-rank symptoms of schizophrenia is assessed. Frequency of occurrence of these symptoms, their ability to differentiate between schizophrenia and affective disorder, the method of eliciting them, and the identity of symptoms are considered. Schneider's claims about first-rank symptoms find only limited support from the more recent literature. However, these studies rarely acknowledge the problems of defining and eliciting these symptoms, and their conclusions should not be unreservedly accepted. The choice of particular diagnostic schema of schizophrenia will be determined by its usefulness until some external validating criterion for this disorder is discovered. (15 references)

6696. Mester, R. (Eitanim Psychiatric Hospital, Doar Na Shimshon, Jerusalem, Israel) The psychodynamics of the dental pathology of chronic schizophrenic patients. Israel Journal of Psychiatry and Related Sciences, 19(4):255-261, 1982.

The oro-dental condition of hospitalized chronic patients is frequently severe and often neglected. Dental problems may provide useful information for psychotherapy and might be very influential in the rehabilitation process. The psychodynamics of dental and gum pathology in the hospitalized patient population and their implications for psychotherapy, management, and rehabilitation are discussed. (13 references)

6697. Owens, D.G.; Johnstone, E.C.; and Frith, C.D. (Div. of Psychiatry, Clinical Research Centre, Northwick Park Hospital, Watford Rd., Harrow, Middlesex, England) Spontaneous involuntary disorders of movement: Their prevalence, severity, and distribution in chronic schizophrenics with and without treatment with neuroleptics. Archives of General Psychiatry, 39(4):452-461, 1982.

Spontaneous involuntary movement disorder was assessed in 411 hospitalized patients with chronic schizophrenia, 47 of whom had never taken neuroleptic medication, using two standardized recording techniques. The prevalence of abnormality depended on the severity criteria but was high, with half of the sample on the Abnormal Involuntary Movement Scale and two-thirds on the Rockland Scale rating 3 or more on one item or more. Comparison of those with and without a history of neuroleptic treatment showed few significant differences in prevalence, severity, and distribution of abnormal involuntary movements. It is concluded that spontaneous involuntary disorders of movement can be a feature of severe, chronic schizophrenia unmodified by neuroleptic drugs. (53 references)

6698. Parker, G. (University of New South Wales, New South Wales, Australia) Re-searching the schizophrenogenic mother. Journal of Nervous and Mental Disease, 170(8):452-462, 1982.

Numerous studies of schizophrenics have defined a dominant, overprotective, but basically rejecting mother as the schizophrenogenic mother. While such a maternal style has been incriminated as a causal influence in the development of schizophrenia and/or a response to schizophrenia or prodromal schizophrenic disturbance in a child, case-control studies raise doubt as to whether such a maternal style is overrepresented in the families of schizophrenic patients. The evolution of the concept and its critical evaluation, principally by American workers, is reviewed and integrated with British studies on expressed emotion. The British studies have looked principally at course rather than onset of schizophrenic disturbance and have demonstrated that exposure to high levels of expressed emotion in a key relative is predictive of schizophrenic relapse. It is suggested that the major components of expressed emotion—critical comments and overinvolvement—parallel the rejecting and overprotective characteristics imputed to the schizophrenogenic mother in the literature. While expressed emotion has been shown to predict relapse, causality has not, as yet, been demonstrated. Several noncausal links are explored. Because such research may promote key advances in the clinical management of schizophrenic patients, the review draws attention to the way in which findings from the regions complement each other. (51 references)

6699. Peteroy, E.T., and Pirrello, P.E. (Behavior Change Institute, Suite 112, 4400 Broadway, Kansas City, MO 64111) Comparison of MMPI scales for black and white hospitalized samples. Psychological Reports, 50(2):662, 1982.

Minnesota Multiphasic Personality Inventory (MMPI) confusion, schizophrenia, and hypomania scales were compared for 30 white and 20 black inpatients at a mental health center. Nonsignificant *t*-ratios were found for males and females on these scales, and data for the sexes were combined into racial groups for analysis. No significant differences between the two racial groups were found. The homogenized nature of this hospitalized population may account for this finding. It is suggested that further exploration of racial bias in the MMPI should continue to focus on matching groups for psychopathology. (2 references)

6700. Ramsay, R.A.; Ananth, J.; Engelsmann, F.; Ghadirian, A.M.; Krakowski, A.J.; and Wittkower, E.D. (McGill University, Montreal, Quebec, Canada) Schizophrenia and psychosomatic illness. Journal of Psychosomatic Research, 26(1):33-42, 1982.

To explore the possibility of a mutual exclusive relationship between schizophrenia and certain psychosomatic disorders, 354 adult schizophrenia patients, both acute and chronic, were studied for lifetime prevalence of peptic ulcer, bronchial asthma, neurodermatitis, and rheumatoid arthritis. Significant evidence of mutual exclusivity was found only in the case of peptic ulcer in the overall sample. Prevalence was higher in younger, more acute patients than in older, chronic patients, but this finding was difficult to interpret. Parallel inquiry into the prevalence of schizophrenia and the same disorders among close family members of the patients yielded inconclusive results. (3 references)

6701. Reid, W.H.; Moore, S.L.; and Zimmer, M. (Nebraska Psychiatric Institute, 602 South 45th St., Omaha, NE 68106) Assessment of affect in schizophrenia: Reliability data. Journal of Nervous and Mental Disease, 170(5):266–269, 1982.

Empirically defined and statistically independent concepts of emotional range, mobility, appropriateness, and communicability were rated by 30 professionals and paraprofessionals from special videotapes of 22 chronic schizophrenic subjects. Reliability of the ratings, measured by correlations among and across parameters and rater types, was notably better than that of the same raters using traditional subjective clinical judgment. There was little disagreement among psychiatrists, psychology interns, junior medical students, or social workers/nurses, nor were any of those groups significantly more reliable than the average for all raters. Significant trends toward low reliability of the appropriateness parameter are pointed out and possible clinical relevance is discussed. (7 references)

6702. Rudden, M.; Frances, A.; and Gilmore, M. (New York Hospital-Cornell Medical Center, Payne Whitney Psychiatric Clinic, New York, NY 10021) Delusions: When to confront the facts of life. American Journal of Psychiatry, 139(7):929–932, 1982.

Research indicates that patients do not hold delusions with as fixed a certainty as has been believed. Confrontation with reality may have an important role in the evaluation and treatment of delusional patients. Four factors may help predict the value of reality confrontation in a given clinical situation: (1) how understandable the delusion is in the context of the patient's life, (2) the degree of conviction with which the patient holds the delusion, (3) the phase in development of the delusional beliefs, and (4) the patient's diagnosis. (15 references)

6703. Salama, A.E.A.A., and Sizemore, D.M. (Southern Illinois University School of Medicine, Springfield, IL 62708) Observations on suicide among hospitalized schizophrenic patients. Hospital & Community Psychiatry, 33(11):940–941, 1982.

Suicide prevention is one of the most serious problems in the management of schizophrenic patients. To enhance the ability to assess suicide potential among hospitalized schizophrenic patients, three very similar schizophrenic men who committed suicide over a period of 18 months were studied at a medium-sized state mental health facility in central Illinois. (9 references)

6704. Sperry, R.B., Jr. (No address given) Myth and psychological integration in schizophreniform disorders. Dissertation Abstracts International, 42(9):3835–B, 1982.

The relationship between the imagery contained in the delusions and hallucinations of schizophreniform disorder and various cultural renewal myths was examined. It is suggested that schizophreniform disorder may have a psychological etiology and may be a constructive, integrative process, and that the ideation in such integrative processes is analogous to cultural renewal themes. Case data were obtained during Jungian existential individual therapy sessions with hospitalized patients. Subjects' imagery took the form of various myths of renewal, and the ideation appeared to be associated with psychological developmental issues and problematic interpersonal relationships. Contrasting interpretations of the data are also discussed. Limitations include the continuing debate about the differential diagnosis of schizophrenia and schizophreniform disorder and an inability to attribute the results to any specific therapeutic procedure.

6705. Starker, S., and Jolin, A. (Portland Veterans Administration Medical Center, Portland, OR 97207) Imagery and hallucination in schizophrenic patients. Journal of Nervous and Mental Disease, 170(8):448–451, 1982.

Volitional auditory imagery and spontaneous imagery/fantasy are examined in schizophrenic vs. nonschizophrenic psychiatric inpatients. Imaginal aspects of schizophrenia are also studied with regard to concurrent presence or absence of hallucination. Comparisons of imaginal function between diagnostic groups and among hallucinatory subgroups turned up few significant differences. Findings do not support the Mintz and Alpert enhanced vividness model of schizophrenic hallucination, but do provide some support for an imagery deficit model. (19 references)

6706. Taylor, P.J.; Dalton, R.; Fleminger, J.J.; and Lishman, W.A. (Institute of Psychiatry, De Crespigny Park, London SE5 8AF, England) Differences between two studies of hand preference in psychiatric patients. British Journal of Psychiatry, 140:166–173, 1982.

Significant differences in handedness patterns between groups of psychiatric patients and normal controls were identified in two recent British studies, with substantial disagreement in some important findings. Most of the discrepancies were attributable to the different application of a simple classification of handedness data, and the remainder to differences in sample size. Diagnosis, sex, and age were found to have a similar effect on handedness in both studies. Neurotic patients were similar to controls regardless of classification, whereas mixed-handedness in personality disorder depended on it. There was no overall excess of left-handedness among schizophrenic subjects, but trends toward excess sinistrality in men and full dextrality in women approached significance. (16 references)

6707. Wilkinson, D.G. (King's College Hospital, Denmark Hill, London SE5 9RS, England) The suicide rate in schizophrenia. British Journal of Psychiatry, 140:138–141, 1982.

A retrospective, case-register-based inquiry on 39 first-admission schizophrenics is presented. Three of the 39 schizophrenics committed suicide. It is noted that schizophrenic patients often talk about, and attempt, suicide. Of the four natural deaths, one exhibited evidence of severe self-neglect and another was not accurately diagnosed for diabetes mellitus due to the mental disturbance present. Clinical features of the suicides include irritable outbursts, histories of previous suicide attempts, and relapses with hallucinations and delusions shortly before death. It is suggested that such deaths are more likely to occur during the early part of treatment for schizophrenia. (10 references)

6708. Williams, J.B.W. (Columbia University, New York, NY 10027) Psychosocial stressors and major depression, schizophrenia, and schizophreniform disorder. Dissertation Abstracts International, 42(9):4154–A, 1982.

The relationship between severity and type of psychosocial stressors and major mental disorders was investigated in data for 247 patients with major depressive disorder, 247 patients with schizophrenia, and 112 patients with schizophreniform disorder. Stressors were classified on the basis of an entrance or exit from patients' social field, desirability, control, number of life change units entailed, and area of life affected. These variables were compared across diagnostic groups for individuals with and without associated personality disorders. Consistent with previous findings, patients with major depressive disorders experienced a greater number of stressors, undesirable events, entrances, and uncontrollable events than patients with schizophrenia. For schizophrenics, the highest level of adaptive functioning in the past year and level of severity of stressors experienced before episode onset were positively correlated, while for major depressives the correlation was negative. The results for schizophreniform disorder were equivocal, with similar results to major depression on some stressor dimensions, and midway between the other groups on others. Implications for social work practice and further life-event research are discussed.

6709. Wold, P.N.; Rosenfield, A.G.; and Dwight, K. (355 Thayer St., Providence, RI 02906) The relationship between work impairment and diagnosis. Rhode Island Medical Journal, 65(4):161–164, 1982.

The relationship between work performance and diagnosis was studied in a community mental health clinic population. Schizophrenia and bipolar affective disorder were found to impair work performance significantly more than other diagnostic entities. Subjects were 218 consecutive admissions who were classified in 13 diagnostic categories. Each subject was rated as to whether or not work impairment was present. Results indicate that work performance is not necessarily correlated to severity of symptoms. (16 references)

Diagnosis

6710. Asnis, L.C.; Baron, M.; and Gruen, R. (Division of Psychogenetics, New York State Psychiatric Institute, 722 W. 168th St., New York, NY 10032) Diagnostic overlap in schizophrenia research: Relation to outcome predictors and family history. Psychiatry Research, 6(3):345–353, 1982.

Five sets of criteria for the diagnosis of schizophrenia were compared in 47 hospitalized patients who fit New York Research Diagnostic Criteria (RDC) for chronic schizophrenia. The other diagnostic systems used were the flexible system, Feighner criteria, New Haven Schizophrenia Index (NHSI), and Taylor Abrams (TA) criteria. The NHSI, the broadest system examined, fit all of the patients studied. In contrast, only 30 patients (63 percent of the sample) were diagnosed schizophrenic using TA criteria. When the Strauss-Carpenter prognostic scale was used, patients who fit TA criteria for schizophrenia did not differ from the remainder of the sample in the prognostic scores. In addition, the two groups did not differ in the familial rate of schizophrenia spectrum disorders. The usefulness of narrow versus broad diagnostic criteria in schizophrenia research is discussed. (33 references)

6711. Bilett, J.L.; Jones, N.F.; and Whitaker, L.C. (School of Professional Psychology, University of Denver, Denver, CO 80208) Exploring schizophrenic thinking in older adolescents with the WAIS, Rorschach, and WIST. Journal of Clinical Psychology, 38(2):232-243, 1982.

Questions of the possibility of diagnosing mental disorder in adolescents were examined. Judgments of specific responses from the Rorschach, the Wechsler Adult Intelligence Scale, and the Whitaker Index of Schizophrenic Thinking (WIST) were made by 10 expert clinicians regarding 12 adolescents aged 16 to 19. Judgments were surprisingly accurate in discriminating among schizophrenics, nonschizophrenic hospitalized adolescents, and a normal control group. Results indicate that schizophrenic thinking is not necessarily characteristic of adolescents and that information from the WIST may be helpful in identifying schizophrenic adolescents. (34 references)

6712. Craig, T.J.; Goodman, A.B.; and Haugland, G. (Information Sciences Division, Rockland Research Institute, Orangeburg, NY 10962) Impact of *DSM-III* on clinical practice. American Journal of Psychiatry, 139(7):922-925, 1982.

Criterion-based diagnoses (*DSM-III*) was compared with clinical diagnoses (*DSM-II*) for 102 psychiatric inpatients. The introduction of specified diagnostic criteria represented a refinement of existing diagnostic practices rather than a qualitatively different approach to diagnoses; however, in the schizophrenia and affective disorder categories, nonwhites and women were more often assigned to a worse prognostic category by the *DSM-II* system than were white men with similar symptoms. The introduction of *DSM-III* criteria may ensure more appropriate diagnosis and treatment for nonwhites and women. (22 references)

6713. Lacks, P. (Washington University, St. Louis, MO 63130) Continued clinical popularity of the Bender-Gestalt Test: Response to Bigler and Ehrfurth. Professional Psychology, 13(5):677-680, 1982.

Neuropsychologists are critical of the continued popularity of single neuropsychological tests, such as the Bender-Gestalt Test (BGT), when more comprehensive test batteries are available. Such criticism ignores evidence favoring a general effect view of brain dysfunction and the reality of the typical testing situation in which psychologists are asked to screen for organicity in psychiatric settings that have high base rates for schizophrenia. Extensive batteries of neuropsychological tests are appropriate for detailed neuropsychological evaluation; however, the brief and inexpensive BGT is more appropriate as a screening test for the most typical referral for differential diagnosis. (21 references)

6714. Lewine, R.R.J.; Kirchhofer, M.; Monsour, A.; Renders, R.; and Watt, N. (No address given) The empirical heterogeneity of first rank symptoms in schizophrenia. British Journal of Psychiatry, 140:498-502, 1982.

First-rank symptoms have assumed an important role in the assessment of schizophrenia. Only recently, however, have there been empirical studies of their reliability and validity. This study examined the relationship between first-rank and other psychiatric symptoms in 100 schizophrenic patients. The results are consistent with other research reports suggesting that first-rank symptoms do not represent a homogeneous group of symptoms within an individual patient. (10 references)

6715. Shukla, G.D.; Sahu, S.C.; Tripathi, R.P.; and Gupta, D.K. (No address given) A psychiatric study of amputees. British Journal of Psychiatry, 141:50-53, 1982.

Psychiatric manifestations were studied in 72 right-handed amputees in the post-operative period. In addition to the phantom limb phenomena observed in nearly four-fifths of the cases, nearly two-thirds had psychiatric symptoms in the form of depression (45 patients), anxiety (38), crying spells (38), insomnia (34), loss of appetite (23), suicidal ideas (21), and psychotic behavior (2). Right-arm amputees experienced phantom phenomena and insomnia significantly more often than left. Nearly one-fifth of the cases were diagnosed as psychotic depressive reactive, two-fifths as depressive neurotic, and two—both right upper limb amputees—as schizophrenic. (15 references)

6716. Young, M.A.; Meltzer, H.Y.; and Tanner, M.A. (Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 60612). Operational definitions of schizophrenia. What do they identify? Journal of Nervous and Mental Disease, 170(8):443-447, 1982.

Several studies have shown that different systems for diagnosing schizophrenia produce diagnoses with relatively low agreement. This, however, does not necessarily imply that the different systems are identifying conceptually different diagnostic groups. They may, in fact, identify a single entity, but with different degrees of accuracy. Using multiple diagnostic systems, 196 inpatients were evaluated. The pattern of relationship among the diagnoses was studied with latent class analysis, a statistical methodology not previously applied to these types of data. Results indicated that the diagnoses of the Research Diagnostic Criteria, Flexible 6, Schneider, and 1978 Taylor and Abrams diagnostic systems all estimated a single underlying diagnosis. The Taylor and Abrams system identified this core diagnosis with high accuracy. The results also suggest that blunted affect and absence of an affective syndrome are related to latent class schizophrenia, while the presence of only nonspecific delusions and/or hallucinations is not related. The appropriateness of the latent class definition of schizophrenia in selecting patients for research is discussed. (18 references)

Epidemiology

6717. Szymanski, H.V.; Gutterman, N.; Salter, V.; and Schulberg, H.C. (State University of New York at Buffalo, Buffalo, NY 14214). Estimating the local prevalence of persons needing community support programs. Hospital & Community Psychiatry, 33(5):370–373, 1982.

As community support programs for the chronically mentally ill expand, it becomes increasingly important to determine the number of individuals qualifying for these services. Although national prevalence data are currently available, they have only limited usefulness for program planning at State and local levels. Because of the distinctive circumstances affecting each community's chronically ill population, their number, and their need for services, three methods to identify a patient cohort that approximates the local prevalence of persons needing community support programs are proposed. The first method identifies persons who have been hospitalized previously and who currently require outpatient psychiatric care. The second identifies persons previously hospitalized who require another hospitalization during a specified period of time. The third method identifies persons who are currently in outpatient treatment with a diagnosis of schizophrenia. Choice of method depends on definition of chronic mental illness, type of data available, local treatment philosophies, and support system structure. (15 references)

The Family

6718. Angermeyer, M.C. (No address given) The association between family atmosphere and hospital career of schizophrenic patients. British Journal of Psychiatry, 141:1–11, 1982.

The content of special 30-minute discussions between each of 30 acute male schizophrenics and their parents was analyzed to determine features of parental personality. Two years after discharge, 13 subjects were readmitted. Compared with those not readmitted, fathers expressed more outward-directed hostility and hostility projected onto others, and mothers expressed more inward-directed hostility as well as guilt anxiety and shame anxiety. Both parents were more emotionally unstable during the course of the discussion. The emotional interaction between mothers and sons was of symmetrical type in contrast to the complementary type shown between mothers and sons who had not been readmitted. (25 references)

6719. Baron, M.; Asnis, L.; and Gruen, R. (Div. of Psychogenetics, New York State Psychiatric Institute, 722 W. 168th St., New York, NY 10032) Schizophrenia: A comparative study of patients with and without family history. British Journal of Psychiatry, 140:516–517, 1982.

To test the hypothesis that schizophrenia can be differentiated on the basis of family history, the authors compared two matched samples of 20 patients each, distinguished by the presence or absence of family history of schizophrenia. Family history was not associated with onset characteristics, symptom picture, phenomenological subtypes of schizophrenia, prognostic indicators, or global assessment score for psychosocial functioning. (9 references)

6720. Midelfort, C.F. (3312 Ebner Coolee Rd., La Crosse, WI 54601) Use of the family in the treatment of schizophrenic and psychopathic patients. Journal of Marital and Family Therapy, 8(2):1–11, 1982.

An ego-oriented family approach to severe psychiatric disturbance in one family member is discussed. The dynamics of family therapy, which utilize the potent influences of the family as experienced in childhood, also use the family as it is today with its mature responsibilities. Cases are presented to illustrate treatment of schizophrenic and psychopathic patients within the family approach. Schizophrenic patients who were too threatened by the hospital and the psychiatrist to participate in therapeutic interviews were allowed to have a relative present. This method was extended to the treatment of patients with psychopathic personalities. The initial purpose in these experiments was to increase the control over the patients' behavior between interviews. The use of these methods in over 100 cases yielded encouraging results. Family therapy represents a combination of psychoanalytic dynamics, a practical application of social psychiatry, and some of the techniques of psychobiology. The approach gives an understanding of psychopathology and psychotherapy that may be an added aid in these difficult fields of study.

Genetics

6721. Baron, M. (Div. of Psychogenetics, New York State Psychiatric Institute, 722 W. 168th St., New York, NY 10032) Genetic models of schizophrenia. Acta Psychiatrica Scandinavica, 65(4):263–275, 1982.

In a study of genetic models of schizophrenia, multiple threshold models of inheritance were applied to a large sample of Franz Kallmann's (1938) pedigree data on schizophrenia. Paranoid and nonparanoid subtypes are represented in the models at different thresholds on a continuum of genetic environmental liability. Single major locus and multifactorial polygenic inheritance were ruled out as modes of transmission. These findings suggest that the paranoid/nonparanoid dichotomy cannot be used as a genetic threshold determinant in the population studied. (56 references)

6722. Baron, M.; Gruen, R.; Asnis, L.; and Kane, J. (Div. of Psychogenetics, New York State Psychiatric Institute, 722 W. 168th St., New York, NY 10032) Schizoaffective illness, schizophrenia and affective disorders: Morbidity risk and genetic transmission. Acta Psychiatrica Scandinavica, 65(4):253–262, 1982.

In a study of morbidity risk and genetic transmission in schizoaffective illness, schizophrenia, and affective disorders, data were analyzed for first-degree relatives of schizoaffective proband and matched controls, bipolars, unipolars, and schizophrenics. The familial pattern of affective and schizophrenic subtypes of schizoaffective disorder resembled the familial pattern of affective and schizophrenic probands, respectively. The overall risk for the spectrum of schizoaffective and affective disorders was higher among relatives of schizoaffective manic as compared to relatives of schizoaffective depressive probands, although the difference was not significant. When tested for consistency with multiple threshold hypotheses of genetic transmission, schizoaffective illness qualified as neither a more extreme form of affective illness nor as a disorder that occupies an intermediate position between bipolar and unipolar disorders or is genetically milder than affective disorder. The implications of diagnostic subtyping for genetic research in the major psychoses are discussed. (24 references)

6723. McGuffin, P.; Holland, A.; and Reveley, A. (No address given) Identical triplets: Non-identical psychosis? British Journal of Psychiatry, 140:1–6, 1982.

In contrast to all previous reports of psychoses in genetically identical siblings, identical triplets aged 28 were discordant by major Kraepelinian type of psychosis. Two had been diagnosed schizophrenic while the third was diagnosed manic depressive. Re-evaluation of their illnesses using standardized methods and diagnosis by blind raters suggested that the discordance was not due to simple misdiagnosis or different diagnostic bias between psychiatrists. These triplets illustrate the shortcomings of a strictly applied Kraepelinian dichotomy and show that a genetic perspective is important in attempting to resolve the problems inherent in classifying atypical psychoses. (20 references)

6724. Ratner, C. (Humboldt State University, Arcata, CA 95521) Do the studies on identical twins prove that schizophrenia is genetically inherited? International Journal of Social Psychiatry, 28(3):175–178, 1982.

Ten studies on identical twins reared apart are reviewed to determine if they substantiate a genetic component of schizophrenia. These studies have not eliminated probable social causes of schizophrenia in the twins, and therefore the origins of schizophrenia remain unsolved. (3 references)

6725. Shur, E. (Maudsley Hospital, London SE5 8AF, England) Season of birth in high and low genetic risk schizophrenics. British Journal of Psychiatry, 140:410–415, 1982.

Hypotheses proposed to account for the unusual seasonal birth patterns observed in schizophrenic populations were tested. The competing hypotheses were tested by retrospectively studying the season of birth in 975 schizophrenic subjects divided according to family history of psychiatric illness. Information was analyzed from case notes, item sheets, and questionnaires sent to general practitioners. The results are inconclusive, but a trend for high genetic risk cases to be born less often in the first quarter of the year was found. Although no clear support could be provided for any season of birth hypothesis, it is suggested tentatively that a seasonal constitutional damage factor may be responsible for the excess of births described in schizophrenic populations in the early months of the year. (28 references)

Prognosis

6726. **May, P.R.A.; Itil, T.; Lee, M.A.; van Putten, T.; and Yale, C. (Neuropsychiatric Institute, Center for Health Sciences, University of California at Los Angeles, Los Angeles, CA 90024) A preliminary attempt to relate individual differences in EEG test dose response to clinical effect. Biological Psychiatry, 17(5):599-603, 1982.**

Can early electroencephalogram (EEG) changes produced by antipsychotic drugs predict the outcome of treatment and aid selection of the right drug, even perhaps the right dose, for a schizophrenic patient? Studies on chronic dosage showed that schizophrenic patients with low voltage fast β activity had a better outcome than those with high voltage slow alpha waves. EEG profiles are similar after single doses of neuroleptics in normal volunteers and sustained dosage in schizophrenic patients. If predictive value could be shown for early EEG changes, this would parallel findings on subjective response to a test dose, early clinical change, and early plasma level, all useful predictors of clinical outcome after continued drug treatment. Early changes in laboratory performance tests were not predictive. (17 references)

6727. **Parnas, J.; Schulsinger, F.; Teasdale, T.W.; Schulsinger, H.; Feldman, P.M.; and Mednick, S.A. (Psykologisk Institut, Psykiatrisk Afdeling, Kommunehospitalet, 1399 Copenhagen, Denmark) Perinatal complications and clinical outcome within the schizophrenia spectrum. British Journal of Psychiatry, 140:416-420, 1982.**

In a prospective study of offspring of schizophrenic mothers, perinatal complications reported in midwife protocols were analyzed for those offspring who, as adults, were diagnosed as schizophrenic, borderline schizophrenic, or as not suffering from mental illness. The schizophrenics were found to have had the most complicated births, and the borderlines were found to have had the least complicated births. This difference is interpreted in terms of a diathesis/stress model. It is proposed that birth complications can decompensate borderline individuals toward schizophrenic breakdown. (19 references)

6728. **Tsuang, M.T. (No address given) Memory deficit and long-term outcome in schizophrenia: A preliminary study. Psychiatry Research, 6(3):355-360, 1982.**

Twenty-two schizophrenic patients who presented memory deficit at admission were followed up over a 30- to 40-year period along with 178 schizophrenic patients without memory deficit. Memory deficit at admission predicted poor outcome in marital, occupational, and psychiatric outcome categories. The symptoms and signs of memory deficit were assessed without knowledge of the patient's status at admission. A significantly higher proportion of the memory deficit group continued to show both symptoms and signs of memory deficit at followup. Thus, the presence of memory deficit in schizophrenics was a highly stable variable across a 30- to 40-year period. Implications of the preliminary results of this study are discussed and future studies are suggested. (28 references)

Psychiatry and the Law

6729. **Szasz, T. (Dept. of Psychiatry, Medical School, State University of New York, Syracuse, NY 13210) The psychiatrist in court: People of the State of California v. Darlin June Cromer. American Journal of Forensic Psychiatry, 3(1):5-46, 1982.**

The trial testimony of a psychiatrist in a murder trial where a plea of not guilty by reason of insanity was entered, is presented with analyses by psychiatrists Donald Lunde, Selwyn Smith, and Joseph Finney. The issue revolved around the ability of a defendant who had been diagnosed as schizophrenic to take responsibility for her acts. In the testimony, it is suggested that a diagnosis of schizophrenia is independent from a determination that a person can control his or her actions and that mental illness should not be considered a disease. Analyses of the testimony suggest that idiosyncratic views on psychiatry were presented, rather than an expert interpretation of the facts of the particular court case. It is also suggested that, if the defendant in the case had had adequate representation, a finding of not guilty by reason of insanity would have resulted. (3 references)

Psychological Theory

6730. Foster, R.M.P. (No address given) The effects of subliminal tachistoscopic presentation of drive-related stimuli on the cognitive functioning of paranoid and nonparanoid schizophrenics. Dissertation Abstracts International, 42(10):4190-B, 1982.

Using two psychodynamic conceptualizations of the paranoid mechanism—Freudian psychoanalytic theory, and the shame/humiliation theory advanced by Arieti and Colby—an investigation was conducted of whether paranoid schizophrenics can be distinguished from nonparanoid schizophrenics on the basis of specific emotional conflicts, and whether these conflicts are directly related to the cognitive pathology manifested by this diagnostic group. Exposure to shame/humiliation conflict produced no significant increases in psychopathology within the paranoid schizophrenic group. Upon exposure to homosexual conflict, only paranoid schizophrenics responded with an increase in concrete thinking pathology. The paranoid subjects showed no significant homosexual activation effects in any of the seven other cognitive symptoms.

Treatment

6731. Alanen, Y.O.; Rakkolainen, V.; and Laakso, J. (Turun Yliopisto Dept. of Psychiatry, University of Turku, Kurjenmaentie 4, SF-20700 Turku 70, Finland) Illness models of schizophrenia and the appropriateness of treatment. Nervenarzt, 53(3):150-153, 1982.

Medical, individual, and transactional models for the proper treatment of schizophrenic patients are described with reference to the results of inpatient and outpatient treatment of 100 schizophrenic patients (ages 16 to 46) in Turku, Finland. The most desirable treatment approach incorporates elements of all three models. The medical model for schizophrenia considers the disease as a disturbance which can be interpreted with psychological concepts. The transactional model addresses patient-centered and illness-centered issues and views schizophrenia as an illness rooted in the patient's past which has a network of complex interrelationships with the patient's present. Each of these treatment methods is sufficient for treating schizophrenic psychoses. However, because of the complex interrelationships associated with schizophrenia, an integrated treatment model brings together all three models in response to the patient's needs. Sixty percent of the cases discussed showed improvement as defined by established therapeutic guidelines, with one-third of the patients treated medically over a period of 2 years, and another third receiving individual therapy at least once a week, including family therapy. (4 references)

6732. Balon, R., and Kabes, J. (Psychiatric Research Institute, Prague, Czechoslovakia) A comparison of various dosages of long-acting lithium carbonate. Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova, 82(2):264-266, 1982.

Long-acting lithium carbonate was administered orally twice a day (once in the evening and once in the morning) to three groups of patients (n = 64) with schizophrenia and schizoaffective psychoses, and the efficacy was compared. The maximum daily dose of the drug did not exceed 2,000 mg, giving a maximum lithium level in the blood plasma of 0.7-1.3 millimoles per liter. The average doses were 1,500 mg per day in the first group, 1,295.4 mg in the second group, and 1,470.5 mg in the third group. Results show that it was sufficient to administer the drug once a day, preferably in the morning.

6733. Caton, C.L.M. (New York State Psychiatric Institute, New York, NY 10032). Effect of length of inpatient treatment for chronic schizophrenia. American Journal of Psychiatry, 139(7):856-861, 1982.

Brief hospitalization policies have accompanied the deinstitutionalization movement, although length of inpatient stay in facilities under State and local administration remains quite varied. A 1-year study of 119 chronic schizophrenic patients hospitalized in New York City between 1977 and 1979 revealed that length of inpatient stay had no relationship to subsequent hospitalization, treatment compliance, or clinical or social functioning in the community. Rather, a patient's postdischarge experiences with treatment and significant others in the community were critical in determining subsequent use of inpatient services. This finding points to the need for wider application of briefer hospitalization policies for dischargeable chronic schizophrenic patients. (20 references)

6734. Coons, D.J.; Hillman, F.J.; and Marshall, R.W. (Alaska Psychiatric Institute, Anchorage, AK 99502) Treatment of neuroleptic malignant syndrome with dantrolene sodium: A case report. American Journal of Psychiatry, 139(7):944-945, 1982.

A case of neuroleptic malignant syndrome is presented in which treatment with dantrolene sodium was associated with improvement. Further exploration of this treatment is proposed. (10 references)

6735. Chouinard, G., and Annable, L. (McGill University, Dept. of Psychiatry, AMI Pharmacology Research Unit, 1033 Pine Ave. West, Montreal, Quebec, Canada H3A 1A1) Pimozide in the treatment of newly admitted schizophrenic patients. Psychopharmacology, 76(1):13-19, 1982.

Pimozide, a specific dopamine blocking agent, was compared with chlorpromazine in a 4-week double-blind study of the treatment of 40 schizophrenic patients newly admitted to hospital through the emergency room. Dosage was adjusted according to therapeutic effect and during the final week ranged from 10-70 mg/day for pimozide and 600-1,500 mg/day for chlorpromazine. Pimozide was found to exert less overall therapeutic effect than chlorpromazine, particularly in highly agitated patients. Women responded better to either treatment than men. A weighted mean of the doses given to male and female patients during the final week suggests that, in the treatment of acutely ill patients, the mg dose equivalency of pimozide in terms of chlorpromazine is approximately 1:25, considerably lower than estimates from maintenance studies. Pimozide induced significantly more parkinsonian symptoms but fewer autonomic side effects than chlorpromazine. The weaker presynaptic dopamine blocking effect of pimozide might be responsible for its reduced potency in the treatment of acute schizophrenic symptoms. (30 references)

6736. Epstein, N. (Children's Psychiatric Center-Community Mental Health Center, Eatontown, NJ 07724) A residence for autistic and schizophrenic adolescents. Social Casework, 63(4):209-214, 1982.

A community-based residence for male adolescents with autism and schizophrenia who require intensive training in basic life skills is described. At the Children's Psychiatric Center-Community Mental Health Center, Eatontown, N.J., group therapy is used to promote eventual semiindependent living. Mastery over idiosyncratic behavior and learning how to obtain and hold a job constitute the main achievements of intervention. The inculcation of public behavior by the children is the preliminary task, and bizarre behavior is not tolerated in the community. Residents are exposed to constant repetition of basic house rules related to daily living. The housefather has a central role in the residence as a stable, warm figure who has set a standard for appropriate interventions. Shopping in the community, weekly group therapy sessions which emphasize eventual entrance into the world of work, and a school that provides intensive remedial education and vocational/functional training are components of the program. (4 references)

6737. Glazer, W.M., and Sheard, M.H. (Yale University, 34 Park St., New Haven, CT 06519) Relapse in patients with shifting RDC diagnoses treated with lithium alone. Journal of Clinical Psychiatry, 43(4):134-136, 1982.

Relapse in four patients identified by retrospective history as having had affective episodes at one time in their life and schizophrenic episodes at another was studied when lithium was substituted for antipsychotic medications for 13 months. Results show two patients relapsed on lithium alone, one relapsed on no medications, and one did well on lithium alone. The phenomenology observed in the three relapsing cases includes mania (as defined by Research Diagnostic Criteria) rapid onset with confusion, sexually regressive behavior, and improvement with reinitiation of antipsychotic medication. Caution is advised in the use of lithium alone for patients fitting this diagnostic description. (10 references)

6738. Glick, I.D.; Klar, H.M.; and Showstack, J.A. (Cornell University Medical College, New York, NY 10021) Toward the definition and delivery of appropriate care. American Journal of Psychiatry, 139(7):908-909, 1982.

Some of the accepted "truths" of psychiatric practice are questioned such as "more is better" and "inpatient care is preferable to outpatient care," and programs based on controlled studies of both clinical outcome and cost efficacy are advocated. Findings from these studies should guide political positions and be incorporated into training a new generation of psychiatrists who will continue to explore, discover, and refine treatment approaches toward a new definition and delivery of appropriate community standards of care. (10 references)

6739. Hagstrom, G.C. (No address given) A microanalysis of Direct Confrontation Psychotherapy with schizophrenics: Using neurolinguistic programming and Delsarte's system of expression.

Dissertation Abstracts International, 42(10):4192-B, 1982.

Direct Confrontation Psychotherapy was analyzed in an investigation of verbal, paralanguage, and nonverbal processes occurring between patient and therapist during four treatment sessions. Four psychotherapy sessions of a chronic schizophrenic and direct confrontation psychotherapist were videotaped beginning with the first therapy session, followed by sessions 1 month, 1 year, and 2 years later. The results indicate interpersonal communicative changes in the schizophrenic patient over the 2-year period, while the therapist remained consistent. Changes in the schizophrenic patient's verbal content revealed a more varied use of complete sentences, more dynamic use of paralanguage, and a more integrated use of nonverbal movement expressions. The methods used by the psychotherapist are described in relation to the theory of Direct Confrontation Psychotherapy. This study is the first complete theoretical and descriptive representation of Direct Confrontation Psychotherapy.

6740. Herz, M.I.; Simon, J.C.; and Szymanski, H.V. (School of Medicine, State University of New York at Buffalo, Buffalo, NY 14214) Intermittent medication for stable schizophrenic outpatients: An alternative to maintenance medication. American Journal of Psychiatry, 139(7):918–922, 1982.

A pilot study of an alternative to maintenance medication for stable schizophrenic outpatients was conducted. Doses in 19 patients were gradually reduced to zero over 8 weeks, and medication was then given only when a patient experienced early signs of relapse. The patients attended weekly group therapy and were closely monitored for prodromal signs, especially at times of stress; significant others helped observe the patients. Five patients experienced increased symptoms during the drug washout period and were dropped from the study; of the remaining 14, 10 remained stable on the intermittent medication protocol. (20 references)

6741. Janakiramalan, N.; Channabasavanna, S.M.; and Narasimhamurthy, N.S. (National Institute of Mental Health and Neurosciences, Bangalore, India) ECT/chlorpromazine combination versus chlorpromazine alone in acutely schizophrenic patients. Acta Psychiatrica Scandinavica, 66(6):464–470, 1982.

The response to electroconvulsive therapy (ECT) or no ECT at two levels of chlorpromazine was studied in a 2×2 factorial experiment on 60 acutely schizophrenic patients hospitalized for 6 weeks. ECT enhanced the therapeutic response at the 300-mg level of chlorpromazine but neither ECT/chlorpromazine combination offered any significant therapeutic advantage over 500 mg of chlorpromazine alone. (13 references)

6742. Liebowitz, M.R. (Columbia University College of Physicians and Surgeons, New York, NY 10032) Newer uses for older psychotropic medications. Hospital & Community Psychiatry, 33(4):282-286, 1982.

New uses are still being discovered for a number of psychotropic agents—e.g., the efficacy of the tricyclic antidepressants for panic disorder and agoraphobia with panic attacks; the use of the monoamine oxidase inhibitors for the above disorders and for atypical depression and hysteroid dysphoria; the use of propranolol for anxiety disorders and for uncontrollable violent outbursts; the antianxiety and antipanic effects of clonidine; and the usefulness of lithium in treating schizophrenia and schizoaffective disorder and for emotionally unstable character disorders. In addition to strengthening the therapeutic armamentarium, the discovery of new drug response patterns helps generate or strengthen hypotheses about the pathophysiology of various psychiatric disorders. (47 references)

6743. Matthews, J.G. (Auburn University, Auburn, AL 36830) The process and outcome of therapy of outpatient schizophrenics by A and B therapists: Does psychotherapy add to the effects of psychopharmacotherapy? Dissertation Abstracts International, 42(10):4198-B, 1982.

The question whether psychotherapy can add appreciably to the treatment of schizophrenic patients who are concurrently receiving medication when the patients are treated by A or B therapists was studied. Forty-two patients diagnosed as schizophrenics and matched for age, sex, education, and socioeconomic status were randomly assigned to one of three treatment conditions: medication and psychotherapy with A therapists, medication and therapy with B therapists, or medication alone. Process and outcome for all three groups were evaluated by therapist, patient, and a family informant. Analyses of covariance computed for the various outcome measures indicate considerable clinical support for the hypothesis that psychotherapy can add appreciably to the effects of medication for schizophrenic patients if the therapists are differentiated by type. However, there were no significant differences in the process analyses of the therapeutic relationships of the A and B therapists with their patients.

6744. Moffic, H.S. (Dept. of Psychiatry, Baylor College of Medicine, Houston, TX 77030) A preliminary report on effects of initiating medication groups at a mental health clinic. Hospital & Community Psychiatry, 33(5):387, 1982.

To analyze the effects of medication groups, a mental health clinic tested 30 patients with a primary or secondary diagnosis of schizophrenia. The patients were randomly selected from the four medication groups at the clinic. Data on three treatment variables—rehospitalization, average daily dosage of medication prescribed (in chlorpromazine equivalents), and number of appointments—were compared for the 18 months before and the 18 months after establishing the groups. Such an approach enabled the patients to serve as their own controls. The rehospitalization rate markedly decreased from 27 percent before the groups were established to 10 percent afterwards. An unexpected finding was a concomitant decrease in the amount of medication prescribed. Only two patients exhibited tardive dyskinesia. Total kept appointments nearly doubled with the medication groups. (1 reference)

6745. O'Connell, R.A. (St. Vincent's Hospital and Medical Center, New York, NY) A review of the use of electroconvulsive therapy. Hospital & Community Psychiatry, 33(6):469–473, 1982.

Electroconvulsive therapy (ECT) is an effective psychiatric treatment for severe depression, mania, catatonic states, and, to a lesser degree, some schizophrenias. Complications are minimal and the risk of death lower even than that for inadequately treated depression. However, some medical centers in the United States do not use ECT. The neurochemical mechanisms that underlie ECT remain unknown, but a cerebral seizure is essential. The average number of treatments is seven, spread over 2 to 3 weeks. All candidates for ECT should undergo a complete medical evaluation and give informed consent. The current use of ECT, clinical indications and contraindications, complications, techniques, mechanisms of action, and some related ethical and legal issues are presented. (15 references)

6746. Pecknold, J.C.; McClure, D.J.; Allan, T.; and Wrzesinski, L. (St. Mary's Hospital, 3830 Lacombe Ave., Montreal, Quebec, Canada H3T 1M5) Comparison of pimozide and chlorpromazine in acute schizophrenia. Canadian Journal of Psychiatry, 27(3):208–212, 1982.

Pimozide and chlorpromazine were compared in a 4-week double-blind study to test the hypothesis that pimozide, a powerful dopamine receptor blocker, is more effective in treating acute schizophrenia than chlorpromazine. Twenty male and female subjects hospitalized for acute schizophrenia were treated on an individual titrated dosage of either 300 mg of chlorpromazine or 10–70 mg of pimozide. On the Brief Psychiatric Rating Scale, the chlorpromazine subjects significantly improved after 1 week, whereas the pimozide subjects showed no statistical improvement until week 3. By the end of the treatment period, no significant differences were apparent between the two groups. On the Clinical Global Impression Scale, a significant difference between groups was found at week 4—a greater improvement in the chlorpromazine subjects. Fifteen adverse reactions were noted for the pimozide group as compared to eight for the chlorpromazine group. It is concluded that chlorpromazine has an earlier onset of action than pimozide in acute schizophrenia, despite the fact that it has a weaker effect on the dopamine receptor than pimozide. The findings suggest the need for a critical reexamination of the dopamine theory of schizophrenia. (13 references)

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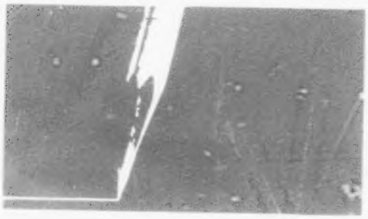

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About the Cover

The *Schizophrenia Bulletin* features art done by current and former mental hospital patients. The drawing that appears on the cover was sent to us by Jean K. Bouricius, the artist's mother. The artist, Kevin Bouricius, has provided the following description of his painting:

This painting represents two delusions—one was that I was a powerful giant who could do everything but make people happy, and the other was that my body was on fire. Sometimes these two delusions occurred together. The lantern in the giant's hand represents his search for meaning in life. This picture was painted not long after the death of John Lennon, and I feel that the painting may represent Lennon, who was a giant of a man on fire with creativity.

Semantic Barriers to Family and Professional Collaboration

by Agnes B. Hatfield

At Issue

The At Issue section of the Schizophrenia Bulletin *contains viewpoints and arguments on controversial issues. Articles published in this section may not meet the strict editorial and scientific standards that are applied to major articles in the* Bulletin. *In addition, the viewpoints expressed in the following articles do not necessarily represent those of the staff or the Editorial Advisory Board of the* Bulletin.—The Editors.

Abstract

Mental health professionals have begun to show interest in working collaboratively with families of mentally ill people in the interest of the patient's well-being. Many professionals are aware, however, that considerable alienation exists. One source of difficulty may be the language used to describe families. Drawing from the field of general semantics, the author examines recent writings for examples of language that may appear confusing or offensive to families. It was found that difficulties result from professionals' tendencies to confuse fact with inference, to stereotype families by placing them in either/or categories, to use judgmental language, and to use language of control that contradicts their goals of collaboration or partnership.

The movement toward community care of persons with serious mental disorders has brought together mental health professionals and families in ways that were not anticipated. A number of writers have stated recently that if the patient is to do well in the community, then genuinely collaborative relationships must be developed between the practitioners who treat patients and their relatives who look after them (Hatfield 1979, 1981; Beels and McFarlane 1982; Bernheim 1982; Holden and Lewine 1982). It has only been in recent years that the idea of "collaboration" has had much expression. Probably few professionals now practicing have been trained to think of families of mentally ill persons as partners in the caregiving process and even fewer learned effective strategies for working with these families. This is a time when the barriers to collaboration remain high, but it is also a time when both groups are seeking better relationships.

Unfortunately, there is a dearth of research which reveals the family's perspective on mental illness. Thus, there is little help for professionals who try to understand the world view of the family. Where the family experience has been described by families who have mentally ill relatives, it has been demonstrated that the burdens are high, the complexities and confusion great, and the stresses overwhelming at times. Families are truly victims of severe psychiatric disorder (Clausen and Yarrow 1955; Creer and Wing 1974; Doll 1976; Hatfield 1978; Arey and Warheit 1980). When asked to identify their needs for help, families indicate they need to understand mental illness, to know appropriate expectations, and to learn practical management skills.

Most traditional family therapies and psychoanalysis did not respond to these needs. Instead, practitioners of these theoretical persuasions saw families as the source of pathology in their relatives and believed that help for the mentally ill person first

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required treating the sick family. In a half century of search for the nature of this pathology, theorists have cycled through a great number of explanations of which the concepts of symbiotic or rejecting mothers, marital schism and skew, communication deviance, enmeshed families, and distorted hierarchical relations each had a period of popularity. Today's practitioners have considerable difficulty establishing trust with families because the field of mental health carries a legacy of negative views toward families.

Some current leaders in the field are vigorous in their disavowal of these damaging theories and propose alternate ways for viewing and helping families (Beels and McFarlane 1982; Anderson 1983; Terkelsen 1983). Using various forms of psychosocial education, a number of practitioners are now giving significant attention to the practical problems of living with a mentally ill relative (Falloon et al. 1981; Goldstein and Kopeiken 1981; Anderson 1983). Although few assessments of family satisfaction with these treatments have been reported, it is expected that families would respond to them well. Still there is reason to believe that many families experience alienation from the mental health profession. We need to understand why this occurs.

Families feel considerable stigmatizing at the hands of professionals. Through such organizations as the National Alliance for the Mentally Ill, they are asserting their need to be seen in a fair and accurate way. When other devalued groups—blacks, women, and the elderly—demanded a change in the way they were viewed, they raised consciousness through attention to language. It is with that same intent to raise consciousness that we have chosen to focus on language used to describe families of the mentally ill. Insights from the field of general semantics were used to guide this study. A close look at the words being used to describe families may reveal beliefs and attitudes that are negative toward families, or perhaps just as important, beliefs and attitudes that families think professionals hold. General semantics is a powerful tool that can help us analyze the speech we use. It can lead us to question our assumptions or encourage us to select language to convey our meanings and describe our observations more accurately.

According to a leading general semanticist, Hayakawa (1964), general semantics is "the study of human interaction through communication," and further, "communication sometimes leads to cooperation and sometimes to conflict" (p. iv). Johnson (1962), another expert in the field, provides additional support for the usefulness of examining language:

> We are a symbolic class of people. To say that we are human is to say, above all, and with incalculable significance, that our problems, as individuals, as groups, and as a world culture are symbolic problems . . . . They are problems of meaning, of evaluation, or orientation, processes which, on human levels, are predominantly symbolic in character. [p. 111]

Words have the power to mold men's thinking, channel their feelings, and direct their actions. Conduct and character are largely determined by words we use to describe ourselves and the world around us. The way we use words largely shapes our beliefs, prejudices, ideals, and aspirations (Johnson 1962).

The language of the social sciences has particular pitfalls, for it has less precise and common understandings than are found in the physical sciences. In the discussion that follows, common problems in language usage are identified to serve as guides in the analyses of recent professional literature about treatments. Some of the most common problems are:

- Confusion of fact and inference
- Use of two-valued orientations
- Prevalence of the language of judgment
- Use of language of control
- Occurrence of the double bind

There has been no attempt to sample widely the problems in language usage. The purpose in this article is to identify problems that exist and to show how they may be alienating families. The writings we chose to examine were published in the last 3 or 4 years and were authored by people who indicated that it was their purpose to work effectively with families. We believe that it is this group of practitioners who may be most interested in pursuing all avenues to better relationships.

Confusion of Fact and Inference

In accurate speech, a clear separation must be made between fact and inference. Facts are a precise report of what we have seen and done. They must be capable of verification (Ogden and Richards 1956; Hayakawa 1964; Chase 1966). When one goes beyond that which is directly observable and verifiable, one is making statements of inference. When some variation of the verb "to be" is used, the writer is presumably making a statement of fact. Chase (1966) warns of the potential pitfalls in the ubiquitous use of this verb form. He says that "one should handle the little word 'is' like a stick of dynamite. It presumes a statement of fact and therefore is an event subject to operational verification" (p. 82). A statement of inference goes beyond the data and draws conclusions, makes interpretations, and conveys judgments.

There was a pronounced tendency in the literature reviewed for statements of inference, belief, and opinion to be offered as though they were facts. The work of Madanes (1982) furnishes us with one prime example. In describing the relationship between a disturbed young person and his or her family, Madanes says, "The young person's disturbing behavior *is* helpful to the parents" (p. 50); "The disturbed behavior *is* the basis of power [over the parents]" (p. 50); "When a family with a severely disturbed youth comes to therapy there *is* a split between parents" (p. 54); and "The disturbing behavior *perpetuates* the problem" (p. 54, emphasis added). An uncritical reader could be led to think that Madanes is stating fact. it is never clear how or to what extent any of the statements made above have been verified. When statements of belief of opinion are repeated often enough, they can become reified and accepted as fact. This gives us a distorted body of knowledge, and when a perceptive person discovers there are no verifiable data to support assertions made, the mental health profession loses credibility.

An especially disturbing example of statements of inference being made as though they were fact follows: "Symptomatic behavior, at times escalating until hospitalization is needed, *is* a mechanism by which a dysfunctional family system regulates itself to resist change" (Scharfstein and Libbey 1982, p. 561, emphasis added). This assertion, probably not possible to prove with directly verifiable observations, is given to the reader as though it were fact. Of special concern, is the fact that Scharfstein and Libbey presented these unsubstantiated opinions to families who had just brought a disturbed member to the hospital and when they were in a fragile state. They were told, as if it were fact, that "important interactions between everyone in the family [have] contributed to the decision that one member would be hospitalized" (p. 562). "The hospitalization," they declare, "*was* the family's solution to the pain experienced by all members" (p. 563, emphasis added). There is no evidence that intensive observations have been made or careful case histories taken. Rather, it appears, any beleaguered family would have these judgments placed upon them. How do the families respond? The authors say that families report a great deal of shame, embarrassment, and guilt, and believe themselves to be unworthy of the staff's kind attention. It is hard to believe that approaches such as these lead to true collaboration. We often hear families say that they "feel accused and judged guilty without even a trial."

A couple more examples will suffice to demonstrate how opinion, belief, and judgment can be misleadingly represented as fact. McFarlane (1982) states, "The results of family visits to the patient are well-known to most clinicians; too often visits *are* followed by relapse" (p. 106, emphasis added) and "It is implicit in this type of group [one with chronically mentally ill members] that most of the families *are* quite disturbed" (p. 115, emphasis added). This seems a strong and potentially damaging statement to make about anyone, and McFarlane does not support it.

A final illustration is drawn from the work of Leff, Kuipers, and Berkowitz (1983). They state, "A major problem in attempting to move a patient from a parental home *is* that if either parent is emotionally involved, he will not allow the patient to leave. Furthermore, the patient also contributes to the bond and *is* often as emotionally dependent on the relative as the relative *is* on him or her" (p. 180, emphasis added). Leff and his colleagues state as an additional factor that there is a dearth of sheltered accommodations, and that vacancies are rarely available when needed. This is a very strange twist of logic. Families and patients are criticized for their excessive dependence on each other, when lack of community resources makes it impossible for them to be otherwise. This sounds a bit like a classic case of "blaming the victim" (Ryan 1973). Furthermore, one wonders how the authors get their "facts" when the opportunity is not there to test out willingness to separate.

Confidence in mental health research depends on accurate communication. When opinion and belief are presented as facts, credibility suffers. It is not that opinion should never be used; it is just that there should be no doubt in the reader's mind that that is what it is. While perhaps somewhat cumbersome, words such as "it seems to me," "I think that," "in my opinion," or "as I see it" must be stated. Furthermore, writers who discipline themselves to separate fact from belief and opinion may find themselves going back to sources to check the evidence for the ideas they hold.

Two-Valued Orientations

Human characteristics rarely, if ever, occur as all or none capacities;

rather, they occur on a continuum. The English language, nevertheless, is heavily biased toward "either-orness" and the social science literature reflects this kind of thinking. Durin (1983) calls two-valued orientation "an analytic crudity" (p. 41) that is totally divorced from reality. Characteristics of people exist in amounts, degrees, or gradations but almost never in polarities. Family therapists, however, frequently do see families in terms of "either-orness," that is, in terms of polarities or two-valued orientations. Note the following:

> It might be hypothesized that many families eventually respond in *one of two ways* Brown and his colleagues have described as components of high EE [expressed emotion]. They have become over involved and intrusive, constantly attempting to monitor and protect the patient from themselves or the environment; *or* they become frustrated, angry, rejecting, and withdrawn from both the patients and systems that treat them. [Anderson 1983, p. 101, emphasis added]

An increasingly popular concept in research and treatment is the concept of the family member's "expressed emotion" (EE). The tendency is to think of this characteristic in polarities. People either have "high EE" or "low EE." No recognition is given to the likelihood of EE being qualitatively different or occurring in widely varying amounts. Snyder and Liberman (1981) explain this two-valued concept as follows:

> The number of criticisms together with the quality of emotional over-involvement expressed by relatives designates them as high or low EE. A score of 6 or more criticisms, or 4 or more on the 6-point emotional over-involvement scale, places a family in the "high" EE category. Relatives with 5 or fewer criticisms and ratings of 3 or less on emotional over-involvement comprise the "low" EE group. [pp. 50-51]

A difference of just one point can place a family at one pole or the other. Such analytic crudities are being used to develop big research projects (Anderson, Hogarty, and Reiss 1981; Berkowitz et al. 1981; Goldstein 1981; Anderson 1983). One wonders why such reductionist concepts as high and low EE are used to categorize complex human beings. We suggest that it is used either as an economical device or that researchers do not understand the dynamics of these families well enough to attempt more detail. Unfortunately, such crude concepts give the impression that more is known than really is. Families may be alienated by these labels which, in essence, tell them they are "good families" or "bad families."

The polarities "functional" and "dysfunctional" are commonly used to describe families. Falloon et al. (1981) speak of "dysfunctional patterns of communication." McFarlane (1983) notes "blatant forms of family dysfunction." Scratton (1982) infers something called "systems dysfunction." Is it not more accurate to the facts of all family life that families carry out their functions with varying degrees of adequacy, and that they are not static as implied in the words quoted, but, rather, are in constant flux and change? More is at stake than words; words influence the way professionals see families, what they expect of them, and how they treat them.

The words "enmeshment" and "disenmeshment" occur frequently (McFarlane 1983; McFarlane and Beels 1983; Beels and McFarlane 1982; Goldstein and Dyche 1983). Although no definition was given, these terms appeared to refer to a family's tendency to be overly involved in its own members with a consequent isolation from the rest of the world. McFarlane (1983) sees "family enmeshment as a key factor in schizophrenia" and the goal in family treatment as "disenmeshment" (p. 147). Can we really say that families are "enmeshed" or they are "disenmeshed"?

Harbin (1982*b*) speaks of "problem families," "disturbed families," and "resistant families" (pp. 4-22). These are static concepts that characterize poorly the dynamic, fluid character of human life. They label rather than describe. The same thing can be said of "normal or pathological" (McFarlane 1982, p. 118) and "overinvolved," "underinvolved," and "pseudo-involved" (Harbin 1982*a*, p. 145). "Words," Hayakawa (1962) reminds us, "are extremely powerful tools." How we talk and think about others determines how we act toward them (Lee 1962).

Language of Judgment

In the preceding section, it was proposed that considerable disservice is done to families by using two-valued orientations of them. Of even greater concern was the almost exclusive focus on negative characteristics. If writers were aware of positive characteristics in the people they served, they did not mention them. In addition to the frequent use of "enmeshment," "dysfunction," and "pathogenesis," there were other such pejorative terms as "pernicious" and "acrimonious" to describe family environments (Goldstein 1981, p. 1) and "noxious" to describe family members (Clarkin and Glick 1982, p. 167). McFarlane (1982, p. 107) speaks of "psychotic-level" families.

Heinrichs and Carpenter (1983) advocate assessing strengths and limitations of both the patient and the family. In the two cases they reported for illustrative purposes, however, their language tended to be judgmental and negative. In case example I,

> Mark's father was a bright but neurotically tormented man gripped by obsessions and inhibitions. . . .Mark's mother appeared weary, detached, and embittered. . .both parents felt hopeless about Mark's chances of recovery and resentful that needing to care for him would always plague their lives. . .they acted as if they were being intentionally punished. . . . Every time Mark did show improvement. . .his parents responded as if it were just a cruel torment to raise their hopes and then plunge them into deeper despair. [p. 284]

There was no mention of strength in this family nor was there any recognition that this might, indeed, be an expected response to a situation in which the 32-year-old son had had a "long and stormy history of schizophrenia with many episodes of psychosis" (p. 284).

In case example II, Heinrichs and Carpenter (1983) describe a mother as "an extremely difficult and overbearing woman. She had a remarkably severe inability to hear even significant communications without distortions and confusion. . .initial sessions made it clear how utterly maddening it was for anyone to deal with her" (p. 385). Observation and description with a minimal level of inference and judgment is a necessary foundation for working with families. Perhaps, it is because this work is so frustrating and demanding that good scientists lose sight of this fact.

Words like "dysfunctional," "noxious," "pernicious," "pathological," and "disturbed" are emotionally toned labels and must surely make families feel rejected and disapproved. Hayakawa (1964) says that when a person uses affect-laden judgments, the most appropriate question is "Why do *you* feel that way?"

The judgment of blame is a matter for discussion in much literature. Weinstein (1982) suggests that telling families they were a causal factor in their relative's mental illness does not constitute blame as long as the practitioner recognizes that they did not do so intentionally. It is our belief that families will feel blamed under such circumstances and that intention is not the issue. Wynne (1983) states, "There is a human tendency that, if anyone goes wrong, to blame oneself, someone else, or something else" (p. 258). He says it is a natural and inevitable human response and that health professionals should avoid the self-flagellation of thinking that they are the only ones who blame families. This is an interesting statement but offers little hope for overcoming the serious schism between families and professionals.

Language of Control

On the one hand there is a tendency in current literature to talk about the family as a part of the team and of the need to work cooperatively and collaboratively with it; on the other hand, however, much language suggests that professionals are more often seeking to control families. As Coleman (1983) so insightfully concludes, "at the heart of the matter lies a struggle for control—the use of symbols to guide, manage, dominate, direct, or regulate the perceptions of others" (p. 402).

Technical language acts as a badge; it generates authority and sets the rules for response. In the process, technical language erects barriers and boundaries that can be counterproductive. By using technical language, professionals take away understanding from clients and in that way control them. Language may confer prestige upon its users and arouse respect and awe among those who do not understand it, but it also creates high anxiety and a sense of helplessness among the uncomprehending (Coleman 1983). Technical language enables persons to talk to themselves and others of a like mind in a language comprehensible only to each other. Such persons are often unable or unwilling to revise their language; thus, the possibility of shared perceptions is limited. Such words as "triangulation," "ecosystemic," "differentiation," and the like are what Postman (1976) calls euphemisms. They are auspicious or exalted terms used to define common experiences. Some people use these words, presumably, in order to influence the way other people regard them. This, Postman calls "impression management" and he quotes G.B. Shaw as saying that "all professions are a conspiracy against the laity." However, Postman notes, such elitism can have serious consequences when it is necessary for the laity to know what is going on.

Postman further points out that the setting in which a service is given is a semantic environment which controls the behavior of others. Who, he asks, can enter a doctor's office and construe his or her role as a customer? Since therapy is usually given to families in offices or clinics like those of physicians, we think Postman's analogy is very much to the point. Clients, he says, are reminded of their situation as a student called upon to explain some difficulty to the principal. Clients do not want to take up too much of the doctor's (or worker's) time. They are eager to do exactly what they are told. Professionals go to extensive lengths to generate and sustain such a metaphor. Offices are designed to create the same sense of mystery, anxiety, and guilt as the principal's office. The patient is summoned when the professional is ready, the decisions are always mysterious, and the patient is rarely clear as to how he or she will be improved by the procedure. Every semantic environment is projected as being something other than it is, Postman (1976) maintains.

Haley (1962) wrote a clever piece in which he points out how psychoanalytic settings make the superior position of the analyst almost invincible. First, the client must voluntarily come to the analyst, thus conceding his inferior position. The analyst uses many maneuvers to create helplessness, such as, "I wonder if that is the way you really feel." If therapy is going badly, the patient is made to feel at fault. We suspect that if Haley made a similar analysis of most family treatments, he would also find they are designed for authoritarian control. The practice of using video cameras, one-way vision screens, and telephones to call in directions to the therapist in contact with the family gives considerable power of control over the family.

Language of control was common in the literature reviewed. Professionals talk about "handling" families (Harbin 1982*b*; Scharfstein and Libbey 1982), of using "paradoxical treatment" as a means of control (McFarlane 1983), and of "reframing" so that the family is forced to define the problem or see the situation in a way useful to the professional (Clarkin and Glick 1982; Scharfstein and Libbey 1982). Although Anderson (1983) and her co-workers advocate "mutual agreement about the goals, content, length, rules and methods of therapy," they also state they *refuse* to entertain unrealistic goals, *require* families to postpone "major separation issues," and *will not entertain* such ideas. Harbin (1982*b*) informs families that they are expected to be part of the treatment team and *must orient themselves to a new definition of the problem* (presumably the definition held by the professional). Whoever defines a problem has a great deal of power, for everything else follows from it, including the nature and length of treatment and the evaluation of outcome.

How much of the difficulty between families and professionals is a power struggle, with overwhelming need of each to control, is not known, but there is evidence that "no win" situations do develop. There seems to be considerable concern over "resistant families" (Beels and McFarlane 1982; McFarlane 1982; McFarlane and Beels 1983). Harbin (1982*a*) discusses at some length the power struggles that go on in State institutions between families and staff.

It was interesting to find out that there were professionals who did not consider the authoritarian role either desirable or inevitable. Bell (1982) made cross-cultural studies of families with relatives in hospitals and reported that families in many parts of the world were welcomed as participants in the care of their relative. Sussman (1982) sees the necessity and the possibility for more "equitable and dignified family-bureaucratic organization linkages and relationships" (p. 277). He feels the role and accompanying expertise of each are different, but that each serves to complement the other. Bruggen and O'Brien (1982) are highly critical of families who hand over all decisionmaking to professionals and of professionals who insist on their superior capacity to know what is good for others.

Double Bind in Family Treatment

McFarlane (1983) may be correct that "the family is being double-bound by the therapy system" (p. 234). As he further points out, to the extent the treatment system is double-binding the family, no change is likely, the family is continually forced to defend itself, and no therapeutic alliance can develop. Probably most obvious is the contradiction that is apparent when professionals say they value families as a part of the treatment team but see them almost exclusively in negative terms. They criticize traditional theorists for their tendency to blame families but are unable to conceal their own attitudes of blame. With such mixed messages, families are certain to be defensive and therapeutic alliances unlikely.

In our discussion of power and control, there was much evidence of professionals giving mixed signals to families. The tendency was to bring families into an authoritarian environment, define the problem the way the professional sees it, and yet expect of the family a real take-charge stance in the home. There is tyranny in the power to define a problem and make it stick, Postman (1976) insists, and to that extent there is a tyrannical situation in most family therapy. Unquestioning and passive participants in the clinic are likely to be seen as good clients, but will families unlearn this role fast enough to take charge of a difficult situation at home?

Madanes (1982), for example, defines the goals of therapy for the family and uses the considerable power of her clinical approach to compel families to take charge of their mentally ill son or daughter and require him or her to behave properly. One wonders if families who succumb to the power of the therapist can then assert themselves in relation to their offspring. Conflict occurs, Postman (1976) reminds us, when there are incompatible metaphors, i.e., when there is incongruity so that reasonable actions of one person will inevitably conflict with reasonable actions of another.

There is also incongruity in recent statements of McFarlane and Beels (1983) where they make unquivocal statements that the important goal is to become working allies *with* families of schizophrenic patients, but having said that, revert to authoritarian language in describing what to do *to* families. They conclude their work with a description of an elaborate decision-tree model in which the professional makes the decisions about what kinds of treatment the family is to undergo.

Unparalleled in the literature reviewed were the mixed messages in the Scharfstein and Libbey (1982) work, discussed earlier in this article. These authors express the laudable intention of making families feel at home in the hospital setting, but in what they characterize as "the initial traumatic period of the ward," families are directly implicated in causing the mental illness and subsequent hospitalization of their ill relative. The families' expressions of pain and embarrassment, and their feelings of being unworthy of any help, were considered to be a basis for an alliance with families.

Summary and Conclusions

The willingness of a new generation of family therapists to work with families in a cooperative way is acknowledged. It is also acknowledged that such a significant change from traditional relationships is not easy to develop and that there are serious barriers to overcome. Our approach to further understanding was to analyze samples of language used for clues to the attitudes, thoughts, and behaviors of professionals in relationship to families. This analysis revealed that there are considerable semantic barriers to collaboration and that probably there is more at issue than misapplied words. Rather, there are fundamental problems implicit in the perceptions and attitudes of those who work with families.

Change, if it is to come, may require that professionals examine their own needs and problems, and that they question their training and the theories that color their perceptions and guide their practices. Practitioners may be much more bound to traditional family theories than they acknowledge. Theories rigidly held can hold back progress toward new types of relationships. The antidote to unscientific stances, Postman (1976) advises, is our ability to conceive of false ideas—that is, to seek reasons for not believing something. This is hard for highly motivated, well-intentioned practitioners to do. Certainty is desperately needed, for families come to them with huge and ever present problems. There is pressure to oversimplify, to select from observations of families that which confirms previously held assumptions, with consequent failure to note the complexities of the family dilemma—the paradoxes, contradictions, and competing explanations.

Analysis of language points up some of the difficulties, but the solutions may be quite basic. What is lacking, we believe, is an adequate theoretical framework to replace those that are now being discarded. Without a theoretical framework, a researcher may be unaware of his own perspectives, and he may believe he comes to the situation without preconceived knowledge or bias. It is essential that researchers hear themselves, know their own biases, make them explicit, and continuously question them.

In a recent book chapter entitled "Science and Psychiatry," Wyatt (1985) examines some of the difficulties psychiatrists face. It is their desire to simplify complex problems, he believes, that leads to dogma and using single facts as proof. Perhaps it is society's expectations and misunderstandings of the powers of psychiatry, he notes, that lead psychiatrists to establish closed systems and premature judgments. Wyatt faults psychiatric training for not teaching the difference between personal and scientifically based information. As evidence, he points to a recent survey which shows a lack of researchers in departments of psychiatry. "Where do psychiatrists," he questions, "learn to critically evaluate what they read and hear?" (p. 2016).

The purpose of this article was to raise the consciousness of professionals about the language used in talking about families of the mentally ill. Clues to alienation of families are common in the literature. The explanations for language barriers are several: difficulties in dealing with complexities in social science writing; inadequate attention to the effects of language on those it is describing; personal preferences and needs of professionals; and the lack of adequate theory to provide a language appropriate to new views of families.

References

Anderson, C.M. A psychoeducational program for families of patients with schizophrenia.

In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 99–116.

Anderson, C.M.; Hogarty, G.; and Reiss, D.J. The psychoeducational family treatment of schizophrenia. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics*. San Francisco: Jossey-Bass, 1981. pp. 79–94.

Arey, S., and Warheit, G. Psychological costs of living with psychologically disturbed family members. In: Robins, L.; Clayton, P.; and Wing, J.K., eds. *The Social Consequences of Psychiatric Illnesses*. New York: Brunner/Mazel, 1980. pp. 158–175.

Beels, C., and McFarlane, W. Family treatments of schizophrenia: Background and state of the art. *Hospital & Community Psychiatry*, 33:541–550, 1982.

Bell, J. The family in the hospital: Experiences in other countries. In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982. pp. 255–276.

Berkowitz, R.; Kuipers, L.; Eberlein-Frief, R.; and Leff, J. Lowering expressed emotion in relatives of schizophrenics. In: Goldstein, M., ed. *New Developments in Interventions With Families of Schizophrenics*. San Francisco: Jossey-Bass, Inc., 1981. pp. 27–48.

Bernheim, K. Supportive family counseling. *Schizophrenia Bulletin*, 8:634–640, 1982.

Bruggen, P., and O'Brien, C. An adolescent unit's focus on family admission decisions. In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982. pp. 27–47.

Chase, S. *The Tyranny of Words*. New York: Harcourt, Brace & World, Inc., 1966.

Clarkin, J.F., and Glick, I.D. Recent developments in family therapy: A review. *Hospital & Community Psychiatry*, 33:550–556, 1982.

Clausen, J., and Yarrow, M.R., eds. The impact of mental illness on the family. *Journal of Social Issues*, 11:3–64, 1955.

Coleman, W.E. The struggle for control in health care settings: Political implications of language usage. *Et Cetera*, 40:401–409, 1983.

Creer, C., and Wing, J. *Schizophrenia at Home*. London: Institute of Psychiatry, 1974.

Doll, W. Family coping with the mentally ill: An unanticipated problem of deinstitutionalization. *Hospital & Community Psychiatry*, 27:183–185, 1976.

Durin, C. The implications of general semantics for the diagnosis of "Mental Illness." *Et Cetera*, 40:67–73, 1983.

Falloon, I.R.; Boyd, J.L.; McGill, C.W.; Strang, J.S.; and Moss, H.B. Family management training in the community care of schizophrenia. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics*. San Francisco: Jossey-Bass, 1981. pp. 61–78.

Goldstein, M.J. Editor's notes. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics*. San Francisco: Jossey-Bass, 1981. pp. 1–4.

Goldstein, M.J., and Kopeiken, H.S. Short- and long-term effects of combining drug and family therapy. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics*. San Francisco: Jossey-Bass, Inc., 1981. pp. 5–26.

Goldstein, S.J., and Dyche, L. Family therapy and the schizophrenic poor. In: McFarlane, W., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 289–307.

Haley, J. The art of psychoanalysis. In: Hayakawa, S.I., ed. *The Use and Misuse of Language*. Greenwich, CT: Fawcett Publishers, 1962. pp. 207–218.

Harbin, H.T. The family changes the hospital? In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982*a*. pp. 143–154.

Harbin, H.T. Family treatment of the psychiatric in-patient. In: Harbin, H.T., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982*b*. pp. 3–25.

Hatfield, A.B. The family as partner in the treatment of mental illness. *Hospital & Community Psychiatry*, 30:338–340, 1979.

Hatfield, A.B. Psychological costs of schizophrenia to the family. *Social Work*, 23:355–359, 1978.

Hatfield, A.B. What families want of family therapy. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1981. pp. 41–65.

Hayakawa, S.I. Foreword. In: Hayakawa, S.I., ed. *The Use and Misuse of Language*. Greenwich, CT: Fawcett Publications, 1962. pp. vii–x.

Hayakawa, S.I. *Language in Thought and Action*. New York: Harcourt, Brace, & World, Inc., 1964.

Heinrichs, D.W., and Carpenter, W.T., Jr. The coordination of family therapy with other treatment modalities for schizophrenia. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New

York: Guilford Press, 1983. pp. 267-287.

Holden, D.H., and Lewine, R.J. How families evaluate mental health professionals, resources, and effects of illness. *Schizophrenia Bulletin*, 8:626-633, 1982.

Johnson, W. You can't write writing. In: Hayakawa, S.I., ed. *The Use and Misuse of Language*. Greenwich, CT: Fawcett Publishers, 1962.

Lee, I.J. Why discussions go astray. In: Hayakawa, S.I., ed. *The Use and Misuse of Language*. Greenwich, CT: Fawcett Publications, 1962. pp. 29-40.

Leff, J.P.; Kuipers, L.; and Berkowitz, R. Intervention in families of schizophrenics and its effect on relapse rate. In: McFarlane, W., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 173-187.

Madanes, C. Strategic family therapy in the prevention of hospitalization. In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982. pp. 49-77.

McFarlane, W. Multiple family therapy in the psychiatric hospital. In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982. pp. 103-130.

McFarlane, W. Systemic family therapy in schizophrenia. In: McFarlane, W., ed., *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 227-247.

McFarlane, W., and Beels, C. Family research in schizophrenia: A review and integration for clinicians. In: McFarlane, W., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 311-323.

Ogden, C.K., and Richards, I.A. *The Meaning of Meaning*. New York: Harcourt Brace Jovanovich, 1956.

Postman, N. *Crazy Talk. Stupid Talk*. New York: Delacorte Press, 1976.

Ryan, W. *Blaming the Victim*. New York: Vintage, 1973.

Scharfstein, B., and Libbey, M. Family orientation: Initiating patients and their families to psychiatric hospitalization. *Hospital & Community Psychiatry*, 33:560-563, 1982.

Snyder, K.S., and Liberman, R.P. Family assessment and intervention with schizophrenics at risk for relapse. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenia*. San Francisco: Jossey-Bass, Inc., 1981. pp. 49-60.

Sussman, M. Family-organizational linkages. In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum Publications, 1982. pp. 277-296.

Terkelsen, K.G. Schizophrenia and the family: Adverse effects of family therapy. *Family Process*, 22:191-200, 1983.

Weinstein, S.E. The family of the chronic mentally ill patient: Ally or adversary? In: Harbin, H., ed. *The Psychiatric Hospital and the Family*. New York: Spectrum, 1982. pp. 297-308.

Wynne, L.C. A phase-oriented approach to treatment with schizophrenics and their families. In: McFarlane, W., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983.

Wyatt, R.J. Science and psychiatry. In: Kaplan, H.I., and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry IV*. Vol. 2. Baltimore: Williams & Wilkins Company, 1985. pp. 2011-2032.

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Response to Agnes B. Hatfield

by Ian R.H. Falloon

Abstract

Although there remains an undercurrent of criticism toward families in the recent research literature on the family management of schizophrenia, the data offer overwhelming support for the benefits of family care. Professional assistance for family caregivers that is based upon the stated needs of individual families is likely to prove more effective than that based upon theories of family functioning, especially those that implicate the family as a negative influence in the course of schizophrenia.

The major thesis of Agnes Hatfield's essay is that the language employed in the recent scientific literature concerning new developments in family interventions with schizophrenia delivers a semantic message that is indicative of continuing negative attitudes, thoughts, and behaviors of mental health professionals. While acknowledging that recent attempts to assist families may satisfy family needs, Dr. Hatfield states that many families continue to experience considerable alienation from the mental health profession, and concludes that "there are fundamental problems that lie in the perceptions and attitudes of those who work with families."

Despite having some difficulty in fully comprehending the inferential conclusions drawn from the "data" presented in this essay, I would like to endorse some of the sentiments that are expressed. In my opinion, much of the current resurgence of interest in family interventions for schizophrenia undoubtedly continues the legacy of negative attitudes toward families where a member develops schizophrenia—at least in written reports. However, before one condemns these new developments, it is important to note the positive changes that have accrued.

First and foremost, semantics notwithstanding, the benefits of these approaches in reducing the clinical (Goldstein et al. 1978; Leff et al. 1982; Falloon et al. 1985; Hogarty et al., in press), social (Falloon 1985), and family (Falloon and Pederson 1985; Leff et al. 1985) burdens associated with schizophrenia may represent a major breakthrough, comparable with (and complementary to) the major advances associated with the introduction of neuroleptic drug therapy three decades earlier. The optimist now may foresee the day when schizophrenia will no longer be a feared disorder for most patients or their families. The realist will recognize that many years of patient, skillful research is now before us. The pessimist will remind us that if after 30 years even the relatively simple technology of providing optimal drug management remains a scarce commodity in clinical practice, what hope can the consumer feel that the delivery of a rather more complex psychosocial treatment will be readily available if and when sufficient scientific evaluation of its efficacy has been completed?

In the face of the professional optimism that has accompanied preliminary evidence of overwhelming benefits, why do so many families still mistrust us? A simple answer might be that few of us wrote articles in the social science literature that we expected would be read by the consumers. The semantics we have used may merely represent the social science jargon prevalent in the family literature of our generation.

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As Dr. Hatfield has concluded, these new developments lack a new theory, so we have fallen back on the old theories and the old language. The suggestion that these semantic slurs are merely regressive slips that reveal our theoretical heritage gains credence when one compares the recent family literature with that of the previous generation of family theorists. The negative connotation of family involvement with schizophrenia dominates earlier written reports. The recent protagonists appear to see the family in a much more positive light, and these semantic slips are much harder to tease out. For the sensitive family member, however, they undoubtedly leap out from the page.

But do these negative connotations of families really translate into negative behavior toward families? This surely is the crux of the thesis. The strongest evidence that Dr. Hatfield presents is from two sources. First, many of the new developments in this field have selected families for treatment in research studies on the basis of the "expressed emotion" index. The clear implications are that those deemed "high" on the index provide "bad," "noxious" living situations detrimental to the index patients and in need of "therapy"; while those rated "low" on the index provide "good," "benign," "supportive" living situations that benefit the index patient. Furthermore, the sole criterion for a "good" or "bad" outcome has been whether or not the patient experiences a "relapse" of florid symptoms at any time during a 9-month period. While the negative aspects of this concept are clear, perhaps the most disquieting feature of applying such oversimplified (yet excellent) scientific data to clinical practice is the assumption that those families who are deemed "low" on this index have no needs for specific help in caring for their often disabled relatives. In my experience, many families rated as "low" on this index are suffering enormous burdens, but tend to endure their suffering in silence. This behavior may keep patients out of hospitals, but how many of these family members develop their own health problems as a result of the benign neglect they experience so often at the hands of clinicians? The requirement to assess the needs of the *whole* family that result from one or more members who develop any form of major enduring health problems has not been emphasized in the recent literature.

The second source of evidence for negative behavior that is presented is that despite comments about collaboration between professionals and families in most programs, the goals of the interventions are set by the professionals with very limited input from the families. I believe that this approach is to be deplored in clinical practice, while accepting that for research purposes it is sometimes necessary to predetermine the goals of specific interventions. The behavioral family approach assists all family members, including the index patient, to set their own goals and to harness the resources of the family unit to assist them to achieve these goals. It is based on the assumption that at any time all family members are doing their utmost to cope with the situation in which they find themselves. Value judgments that certain family role structures, solutions to problems, or goals are better than others are avoided wherever possible. For example, it remains a *family* decision whether the index patient should continue to live at home or find alternative accommodation. Collaboration is further encouraged by assisting families in the comfort of their own homes where the therapist tends to develop the role of a trusted friend and advisor.

I was surprised that Dr. Hatfield did not question the continued use of the term "schizophrenic" in its adjectival form. Although the concept of the "schizophrenic family" has almost disappeared, persons who have experienced the symptoms of schizophrenia on at least one occasion are frequently described as "schizophrenic." The negative implications of this expression are clear for patients and their families. However, efforts to describe such individuals in such terms as "persons who have had schizophrenia," or "persons (currently) suffering (symptoms of) schizophrenia" seldom pass the eye of journal copy editors who inevitably insert the briefer term "schizophrenics."

I do not agree that a new theory will change the attitudes or behavior of the new breed of family therapists. Nor do I believe we need a new language to describe what we do. We do need to use the English language in a highly skilled manner to describe our methods as clearly as possible to as wide an audience as possible—not the least to our patients and their families. But perhaps the most important message I would like to impart is that I believe that the family is the greatest natural resource we have for the enhancement of the health of *all* its members, including those unfortunate enough to develop a major illness such as schizophrenia. It is a precious resource that deserves our utmost respect and support at all times. This positive attitude should not diminish when the family appears unable to cope at times of crisis, nor should our assistance vanish when the family ceases to plead for help. Our services must meet the changing needs of all families with the most effective strategies that we have developed. I trust that the effectiveness of our actions should far outweigh the inadequacy of our words!

References

Falloon, I.R.H. *Family Management of Schizophrenia: A Study of Clinical, Social, Family and Economic Benefits*. Baltimore: Johns Hopkins University Press, 1985.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Williamson, M.; Razani, J.; Moss, H.B.; Gilderman, A.M.; and Simpson, G.M. Family management in the prevention of morbidity of schizophrenia: Clinical outcome of a two-year longitudinal study. *Archives of General Psychiatry*, 42:887-896, 1985.

Falloon, I.R.H., and Pederson, J. Family management in the prevention of morbidity of schizophrenia: The adjustment of the family unit. *British Journal of Psychiatry*, 147:156-163, 1985.

Goldstein, M.J.; Rodnick, E.H.; Evans, J.R.; May, P.R.; and Sternberg, M. Drug and family therapy in the aftercare treatment of acute schizophrenia. *Archives of General Psychiatry*, 35:1169-1177, 1978.

Hogarty, G.E.; Anderson, C.M.; Reiss, D.J.; Kornblith, S.J.; Greenwald, D.P.; Javna, C.D.; and Madonia, M.J. Family psychoeducation, social skills training and maintenance chemotherapy in the aftercare treatment of schizophrenia: I. One year effects of a controlled study on relapse and expressed emotion. *Archives of General Psychiatry*, in press.

Leff, J.; Kuipers, L.; Berkowitz, R.; Eberlein-Vries, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, 141:121-134, 1982.

Leff, J.; Kuipers, L.; Berkowitz, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients: Two year follow up. *British Journal of Psychiatry*, 146:594-600, 1985.

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An Invitation to Readers

Providing a forum for a lively exchange of ideas ranks high among the *Schizophrenia Bulletin's* objectives. In the section **At Issue**, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. **At Issue** is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the *Bulletin* or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—*The Editors*

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Schizophrenia and Attentional Deficit Disorder (ADD): Reply to Kaffman

by Joseph Marcus

Abstract

Kaffman's critique (1986) of the National Institute of Mental Health-Israeli High-Risk Study pointed to the existence of children who were considered to be suffering from Attentional Deficit Disorder (ADD) among the offspring of the schizophrenic parents—who were considered adult ADD patients rather than schizophrenic patients. We feel this viewpoint represents a misunderstanding of the nature and genesis of ADD and schizophrenia. While a subgroup of the offspring of schizophrenic parents do show "ADD-like" behavior, we believe that their disorder represents a separate and different entity. This is based upon the epidemiological evidence of the differing family patterns on the one hand, and some findings of differing attentional deficits on the other hand. A literature review of these findings is presented as a basis for our view about the differences between ADD and schizophrenia.

Kaffman's critique (1986) of the Israeli High-Risk Study pointed to a very relevant issue which deserves some clarification. Having noted the many signs of central nervous system dysfunctioning that we found in the children born to schizophrenic parents, he suggested that these findings "point to the presence among the index children of a typical syndrome of attentional deficit disorder" (p. 155). Kaffman then hypothesized that there was a subgroup of Attentional Deficit Disorder (ADD) parents in the sample of schizophrenic patients who were included in the project.

It is our opinion that this represents a common misunderstanding which exists in the literature and among practicing psychiatrists. Therefore, we feel it is imperative to clarify this issue.

In the early literature on Minimal Brain Dysfunction (MBD) which has more recently been appropriately designated as Attentional Deficit Disorder (ADD), there were several reports regarding adults who were schizophrenic. Psychiatrists who took the histories of these patients found evidence of some behaviors in childhood that seemed typical of MBD as it was viewed at that time. Wender (1971), in one of the first complete descriptions of this syndrome, alluded to the possibility that some children with MBD might develop schizophrenia, but his present opinion (personal communication) is that this is clearly not so. The same view was put forward by Bellak (1979) based on his clinical experience with adults—which he tried to support with secondhand information regarding the initial reports of our findings in the Israeli High-Risk Study. In a retrospective "catchup" study, Menkes et al. (1967) also reported on four psychotic cases who had been considered to have had MBD during childhood. However, all of these earlier reports have now been supplanted by very different results from other studies, some retrospective, but more importantly, others that are longitudinal and prospective.

Presently, there is a large body of data from many studies that reveals a clear picture—ADD seems to run in families characterized by adults suffering from the behaviors of residual type ADD accompanied by antisocial personality disorders, alcoholism, and drug abuse who give birth to children who have ADD. A subgroup of these children later grow

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up to be normal adults while others develop psychopathology that inevitably presents as antisocial personality disorders, alcoholism, or drug abuse, but not schizophrenia. Some of the principal studies that have demonstrated this pattern include those of Cantwell (1975), Goodwin, Schulsinger, and Hermansen (1975), Blouin, Bornstein, and Trites (1978), Hopkins et al. (1979), Dykman and Ackerman (1980), Loney et al. (1981), Satterfield, Hoppe, and Schell (1982) and Weiss et al. (in press). This issue has been carefully reviewed by Weiss (1985). Bellak (1985) also recently revised his earlier observations and described ADD psychosis as a separate entity from schizophrenia.

At the same time, there is now a growing body of evidence that in many studies of the offspring of schizophrenic parents there exists a very clear subgroup of children who have neurobehavioral deficits, and that these children are similar and appear in similar frequency in different studies and in different countries. This finding became clear in the recent meeting of the Consortium of At-Risk Researchers in Schizophrenia in April 1985. Those results are also reported by Fish (1977), Erlenmeyer-Kimling et al. (1984), and Marcus et al. (1981, 1985).

Thus, from the epidemiological point of view, it would seem that these two disease entities are different and separate, even though the childhood behavior appears on the surface to be similar—both being characterized by attentional deficits, motor deficits, and perceptual deficits.

However, it is our belief that when we begin to investigate these external behaviors on a more basic neurophysiological level, we will discover that the disturbances are in fact different. Although there is not yet a great deal of evidence to support this hypothesis, there is a body of promising work by Nuechterlein (1983, in press) and Nuechterlein and Dawson (1984) that demonstrates distinctly different defects in sustained attention that are found in groups of attentionally deficient schizophrenic offspring and in children with a diagnosis of ADD.

Nuechterlein (1983) examined groups of children using his version of the continuous performance test (Rosvold et al. 1956) and analyzed the data using signal detection theory indices rather than the traditional raw error score. This procedure allowed the investigators to characterize the subjects in terms of the accuracy of their discrimination of target (signal) and nontarget (noise) stimuli, as well as by the response criteria levels—i.e., the amount of perceptual evidence that the subject demands before responding that a given stimulus is a signal. The results suggested that the schizophrenic offspring showed impaired signal/noise discrimination (i.e., a more perceptual-cognitive disturbance), while the ADD children had low response criterion levels consistent with an impulsive cognitive style. Subsequent studies (O'Dougherty, Nuechterlein, and Drew 1984; Nuechterlein, personal communication) have suggested that the lowered response criterion in ADD children may be the primary discriminator between these two groups, as some ADD children also show impaired signal/noise discrimination.

In summary, we would conclude that although a subgroup of offspring of schizophrenic parents do exhibit "ADD-like" behavior, they are not suffering from a disorder identical to that of other children from other families who have ADD. Furthermore, as will be presented in our upcoming article in *Schizophrenia Bulletin* (Marcus et al., in press), it is specifically these offspring who are truly at risk of becoming schizophrenic during their early adulthood.

References

Bellak, L. Psychiatric aspects of minimal brain dysfunction in adults: Their ego function assessment. In: Bellak, L., ed. *Psychiatric Aspects of Minimal Brain Dysfunction in Adults.* New York: Grune & Stratton, Inc., 1979. pp. 73-102.

Bellak, L. ADD psychosis as a separate entity. *Schizophrenia Bulletin,* 11:523-527, 1985.

Blouin, A.; Bornstein, R.; and Trites, R. Teenage alcohol use among hyperactive children. *Journal of Pediatric Psychology,* 3:188-194, 1978.

Cantwell, D.E. Familial-genetic reseach with hyperactive children. In: Cantwell, D.E., ed. *The Hyperactive Child.* New York: Spectrum Publications, Inc., 1975. pp. 93-108.

Dykman, R., and Ackerman, R. Long term follow-up studies of hyperactive children. In: Kamp, B.W., ed. *Advances in Behavioral Pediatrics.* Vol. 1. New York: Jai Press, 1980. pp. 97-128.

Erlenmeyer-Kimling, L.; Kestenbaum, C.; Bird, H.; and Hilldoff, U. Assessment of the New York High-Risk Project subjects in sample A who are now clinically deviant. In: Watt, N.F.; Anthony, E.J.; Wynne, L.; and Rolf, J.E., eds. *Children At-Risk for Schizophrenia.* Cambridge: Cambridge University Press, 1984. pp. 227-239.

Fish, B. Neurobiologic antecedents of schizophrenia in children: Evidence for an inherited, congenital neurointegrative defect. *Archives of*

General Psychiatry, 38:1297–1313, 1977.

Goodwin, D.; Schulsinger, F.; and Hermansen, L. Alcoholism and the hyperactive child syndrome. *Journal of Nervous and Mental Disease,* 160:349–353, 1975.

Hopkins, J.; Pearlman, T.; Hechtman, L.; and Weiss, G. Cognitive style in adults originally diagnosed as hyperactives. *Journal of Child Psychology and Psychiatry,* 20:209–216, 1979.

Kaffman, M. The Israeli High-Risk Study: Some critical remarks. *Schizophrenia Bulletin,* 12:151–157, 1986.

Loney, J.; Whaley-Klahn, M.; Kosier, T.; and Conboy, A. Paper presented at meeting of the Society of Life History Research, Monterey, California, November 1981.

Marcus, J.; Auerbach, J.; Wilkenson, L.; and Burack, C.H. Infants at-risk for schizophrenia: The Jerusalem Infant Development Study. *Archives of General Psychiatry,* 38:703–713, 1981.

Marcus, J.; Hans, S.L.; Mednick, S.A.; Schulsinger, F.; and Michelsen, J. Neurological dysfunctioning in offspring of schizophrenics in Israel and Denmark. *Archives of General Psychiatry,* 42:753–761, 1985.

Marcus, J.; Hans, S.L.; Nagler, S.; Auerbach, J.G.; Mirsky, A.F.; and Aubrey, A. A review of the NIMH Israeli High-Risk Study and the Jerusalem Infant Development Study. *Schizophrenia Bulletin,* in press.

Menkes, M.; Rowe, J.; and Menkes, J. A 25-year followup study on the hyperkinetic child with minimal brain dysfunction. *Pediatrics,* 38:383–399, 1967.

Nuechterlein, K.H. Signal detection in vigilance tasks and behavioral attributes among offspring of schizophrenic mothers and among hyperactive children. *Journal of Abnormal Psychology,* 92:4–28, 1983.

Nuechterlein, K.H. Converging evidence for vigilance deficit as a vulnerability indicator for schizophrenia. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies.* New York: Guilford Press, in press.

Nuechterlein, K.H., and Dawson, M.E. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin,* 10:160–202, 1984.

O'Dougherty, M.; Nuechterlein, K.H.; and Drew, B. Hyperactive and hypoxic children: Signal detection, sustained attention and behavior. *Journal of Abnormal Psychology,* 93:178–191, 1984.

Rosvold, H.E.; Mirsky, A.; Sarason, I.; Bransome, E.D., Jr.; and Beck, L.H. A continuous performance test of brain damage. *Journal of Consulting Psychology,* 20:343–350, 1956.

Satterfield, J.H.; Hoppe, C.M.; and Schell, A.M. A prospective study of delinquency in 110 adolescent boys with attention deficit disorder and 88 normal adolescent boys. *American Journal of Psychiatry,* 139:797–798, 1982.

Weiss, G. Follow-up studies on outcomes of hyperactive children. *Psychopharmacology Bulletin* 21:169–177, 1985.

Weiss, G.; Hechtman, L.; Perlman, T.; and Tuck, D. The hyperactive child as adult. A controlled prospective 15-year follow-up of 63 hyperactive children. *Journal of the American Academy of Child Psychiatry,* in press.

Wender, P. *Minimal Brain Dysfunction in Children.* New York: Wiley-Interscience, 1971.

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Letter to the Editor

The Subcortex, Frontal Lobes, and Psychosis

To the Editor:

Goldberg (1985) has argued that disruption of the mesolimbic/mesocortical dopamine system by long-term neuroleptic treatment may be responsible for producing the tardive dysmentia syndrome (Wilson et al. 1983), which can be seen as similar to the pseudopsychopathic presentation sometimes observed following damage to orbitomedial frontal cortex (Blumer and Benson 1975; Damasio 1979). Similarly, the akinesia syndrome of Rifkin, Quitkin, and Klein (1975) is viewed as analogous to the pseudodepressed variety of frontal syndrome that follows damage to the dorsolateral frontal convexity. These can be considered cognitive or "psychic" analogues to the more purely motoric tardive dyskinesia syndromes that result from disorders of the nigrostriatal dopamine system.

I would extend these observations to include the possibility that dysfunction in the nigrostriatal system, as well as in the mesolimbic/mesocortical system, may also play a role in the cognitive, affective, and behavioral manifestations of schizophrenia and allied disorders, and that the basal ganglia and associated structures may play important roles in broader aspects of human behavior.

The basal ganglia are frequently implicated in neurologic disease states that produce delusional and psychotic symptomatology (Bowman and Lewis 1980; Cummings 1985), and MacLean (1985) has suggested that the neostriatal portion of the basal ganglia may play a role in the coordination of behavioral subroutines that enable the organism to negotiate the demands of the external environment vis-à-vis its own needs and desires—a capacity that has been associated with frontal lobe functioning (Nauta 1971; Luria 1980) and with the psychotic disorders that may attend its dysfunction (Muller 1985).

In man, although the basal ganglia have traditionally been considered a component of the motor system, clinical evidence exists to implicate these structures in the modulation of recursive goal-directed behavior and thought, i.e., in the processes of "motivation" in a broad sense (Caine et al. 1978; Eccles 1982). Indeed, the akinesias ascribed by Goldberg (1985) to dysfunction of the mesolimbic/mesocortical-prefrontal system bear a striking resemblance to akinetic syndromes, psychic as well as motoric, that occur with neostriatal disease or damage (Caine et al. 1978; Laplane et al. 1984). Thus, the nigrostriatal system may possess points of functional as well as anatomic interface with the limbic-cortical systems regulating complex behavior.

Goldberg (1985) points out that

> the frontal lobes, more than any other system, are likely to be the point of summation of dysfunctions generated by many morphologically or chemically diverse systems due to a multifocal or diffuse CNS disease, or to reflect anatomically specific but remote CNS events or disturbances. [p. 259]

As neuropsychologists working in the field of psychiatry, we must resist the temptation to infer structure from function in a literal, correlational manner, based on reference to the brain-behavior rubrics traditionalized over time with neurological populations. Luria (1973) warned against this "narrow localizationism" with respect to brain-injured subjects; how much more cautious must we be when dealing with psychiatric groups where structure-function relationships are only beginning to be understood (Miller, in press).

Goldberg (1985) has made a valuable contribution to this effort by emphasizing the multidimensional nature of brain-behavior interactions with respect to "frontal lobe" deficits in schizophrenia. A similar point has been made with reference to the hemisphericity issue in schizophrenia (Miller 1984). It is to be hoped that this approach will diffuse through research and theory in psychiatric neuropsychology as a whole.

References

Blumer, D., and Benson, D.F. Personality changes with frontal and temporal lobe lesions. In: Benson, D.F., and Blumer, D., eds. *Psychiatric Aspects of Neurologic Disease.* New York: Grune & Stratton, 1975. pp. 151-170.

Bowman, M., and Lewis, M.S. Sites of subcortical damage in diseases which resemble schizophrenia. *Neuropsychologia,* 18:597-601, 1980.

Caine, E.D.; Hunt, R.D.; Weingartner, H.; and Ebert, M.H. Huntington's dementia: Clinical and neuropsychological features. *Archives of General Psychiatry,* 35:377-384, 1978.

Cummings, J.L. Organic delusions: Phenomenology, anatomical correlations and review. *British Journal of Psychiatry,* 146:184-197, 1985.

Damasio, A. The frontal lobes. In: Heilman, K.M., and Valenstein, E., eds. *Clinical Neuropsychology.* New York: Oxford University Press, 1979. pp. 360-412.

Eccles, J.C. How the self acts on the brain. *Psychoneuroendocrinology,* 18:597-601, 1982.

Goldberg, E. Akinesia, tardive dyskinesia and frontal lobe disorder in schizophrenia. *Schizophrenia Bulletin,* 11:255-263, 1985.

Laplane, D.; Baulac, M.; Widlocher, D.; and Dubois, B. Pure psychic akinesia with bilateral lesions of the basal ganglia. *Journal of Neurology, Neurosurgery and Psychiatry,* 47:377-385, 1984.

Luria, A.R. *The Working Brain.* New York: Basic Books, 1973.

Luria, A.R. *Higher Cortical Functions in Man.* 2nd ed. New York: Basic Books, 1980.

MacLean, P.D. Brain evolution relating to family, play, and the separation call. *Archives of General Psychiatry,* 42:405-417, 1985.

Miller, L. Hemispheric asymmetry of cognitive processing in schizophrenics. *Psychological Reports,* 55:932-934, 1984.

Miller, L. "Narrow localizationism" in psychiatric neuropsychology. *Psychological Medicine,* in press.

Muller, H.F. Prefrontal cortex dysfunction as a common factor in psychosis. *Acta Psychiatrica Scandinavica,* 71:431-440, 1985.

Nauta, W.J. The problem of the frontal lobe: A reinterpretation. *Journal of Psychiatric Research,* 8:167-187, 1971.

Rifkin, A.; Quitkin, F.; and Klein, D.F. Akinesia. *Archives of General Psychiatry,* 32:672-674, 1975.

Wilson, J.C.; Garbutt, J.C.; Lanier, C.F.; Moylan, J.; Nelson, W.; and Prange, A.J. Is there a tardive dysmentia? *Schizophrenia Bulletin,* 9:187-191, 1983.

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Thought Disorder in Schizophrenia: Editor's Introduction

by Philip S. Holzman

Investigations into the biology of schizophrenia and of the affective disorders have yielded valuable information about psychotic conditions. Two events have sparked this progress. The first was the introduction of the phenothiazines and the mood-altering drugs in the early 1950s, an event that produced testable biological theories about the drugs' action. The second was the set of two critical reviews of the biological studies of psychosis by Seymour S. Kety (1959), which exposed the many methodological errors that had marred biological studies of psychosis up to that time, and established the standard for acceptable experimentation in this area.

No advance comparable to the biological investigations of schizophrenia has captured the attention and interest of psychopathologists investigating psychological processes, even though it is the psychological aspects of psychosis that cry out most loudly for explanations. Fortunately, today conditions are ripe for a new understanding of psychological dysfunctions in the schizophrenias that is comparable to that brought earlier by advances from the biological sciences. The articles contained in this issue of the *Schizophrenia Bulletin* address in novel ways some relatively neglected aspects of the psychopathology of schizophrenia, namely the varieties of thinking disorders characteristic of schizophrenic patients and their possible underlying mechanisms.

Science advances in spurts. Many periodic eruptions of scientific progress appear to be tied to some advances in knowledge or in technology that then assume the role of scientific tools in other areas of exploration. Our expanding knowledge of brain chemistry surely can claim a decisive role in the extraordinary progress made in the biochemistry of psychosis. Significant conceptual and technological advances await comparable exploitation by the behavioral sciences. These advances include new methods of brain imaging, and the methods cognitive psychologists have been fashioning for the measurement of perceptual registration and the processing of ideas.

The theme of this issue is *formal thought disorder*, that is, disorders in the *form* of thinking and in the organization, control, and processing of thoughts. We are not concerned with the content of thoughts or certain pathological products like delusions. Formal thought disorder has long been considered to be a principal dysfunction of schizophrenic illness. Bleuler (1911/1950) believed that the basic symptom of schizophrenia is the "loosening of associations," his term for formal thought disorder. In his view, formal thought disorder was the basis for the other fundamental and secondary symptoms of the schizophrenias. Bleuler regarded the obvious pathological products of schizophrenic illness, like delusions, as attempts at recovery, as restitutive and reconstructive. As such, he argued, they represent accessory symptoms and are therefore merely epiphenomena. He had no doubt that the primary symptom, the disorder of thinking, was based on a brain dysfunction which, while still unknown and mysterious, would one day surely be known. But, since Bleuler, there has been no systematic effort to study thought disorder that is comparable

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to the generative effort that opened up the biology of mental disorders.

It is of interest that at times Bleuler considered disorders of thought and of attention as distinct fundamental symptoms of schizophrenia, and at other times he appeared to believe that the attentional dysfunction was secondary to the thought dysfunction. Bleuler's description of the schizophrenic disorder of attention, however, is of a special kind. It is not a disorder of *paying attention*, although that can obviously be disordered when motivation is impaired. Both Kraepelin (1896/1919) and Bleuler (1911/1950) believed that the attentional dysfunction in schizophrenia involved what the German language refers to as *passive* attention. Bleuler wrote that even though the patients seem to be absorbed in themselves and are apparently paying little attention to their surroundings, they do register an astonishing number of events that are of little or no significance to them. It is as if the selective function of attention, which regulates the input of myriads of sensory events, is severely curtailed, and thus permits almost anything that stimulates the sense organs to be registered. "Thus," Bleuler wrote, "both facilitating and inhibitory propensities of attention are disordered." Consequently, as far back as Bleuler, one can demonstrate that there has been at least a historical interest in coupling attentional and thought disorders. Today we label them the cognitive dysfunctions of schizophrenia.

It seems evident that to understand the pathogenesis and perhaps even the etiology of the schizophrenias requires that we understand the basic dysfunctions of this set of disorders. If, as Bleuler believed, the basic dysfunction appears in thought organization, can it be identified before the obvious secondary symptoms set in? Early identification of thought disorder, especially in its subtle forms, would permit the studies that are otherwise impeded by the behavioral disorganization and deterioration that characterize the chronic form of the illness.

This question reflects the implicit general orientation of this set of articles: to find in the *forms* of thinking the distinguishing features of various disorders. The writings of both Kraepelin and Bleuler hint at that goal. Kraepelin (1896/1919), who received much of his scientific training in Wundt's psychological laboratory in Leipzig, tried to understand the ways in which basic cognitive processes are organized in several disorders. He particularly concerned himself with the functions of attention, concentration, memory, and motility. Bleuler (1924) discerned several kinds of disordered thinking in schizophrenic patients; incoherence, bizarre associations, incorrect association, clang association, condensation, stereotypy, association by contiguity, and phrase completion are some of the types he described. Both theorists apparently assumed that changes in psychological functions express the organizing principles of the person who manifests those changes which are therefore diagnostic of different disorders. Diagnostic activity here is to be understood as not merely to name a disorder but to come to understand its nature and so to recognize and even to control it.

The focus on thought disorder asks what psychological functions are selectively impaired in different mental disorders. It tries to establish how to recognize the presence of the primary symptoms of mental disorder before the secondary symptoms become conspicuous. This is strategically necessary as well as desirable for research and eventually for prevention, because the secondary, reactive symptoms may be absent or only inconsistently present. When present they may appear only in the advanced forms of the disorders where they have shown themselves to be refractory to scientific scrutiny. Indeed, when present, they actually impede or cut off further communication with the patient and thus bar one of the principal routes for exploring them.

Two impediments have slowed the investigation of cognitive disorders. The first is the tendency of many investigators to search for a single quality of thinking that would encompass all of schizophrenic thinking. Thus, for example, disorders of concept formation, of logical inference ("paralogical thinking"), and of narrow or wide inclusiveness have been introduced as both the principal descriptor of all schizophrenic thinking and as the explanation for the thinking disorders, a rather interesting demonstration of circularity in logic. It is noteworthy that Bleuler considered that the "disorder of association" encompassed many different types of thought disorder, and he considered them all to be symptomatic of the basic defect, which, in his opinion, was a brain disorder.

The second impediment was the absence of appropriate tools for the measurement of thought disorder. Now, however, reliable scales for the measurement of thinking disorders have been constructed, three of which are used in the set of studies reported in this issue of the *Schizophrenia Bulletin*, and whose manuals are reprinted here. In addition, cognitive psychology, which may be considered a basic science for studies of the varieties of thinking processes, has made significant progress in fashioning increasingly precise and subtle methods, some of which are represented in studies reported in the articles that follow.

In the first article, Andreasen and Grove distinguish between "positive" and "negative" thought disorder. The former refers to such occurrences as disorganization, or loss of goal and incoherence. The latter refers principally to poverty of speech. Andreasen and Grove measured thought disorder by the Scale for the Assessment of Thought, Language, and Communication, which distinguishes 18 types of thought disorder. They report that manic patients showed disorganization, incoherence, and illogicality in a context of verbal fluency, which corresponds to Andreasen's category of "positive thought disorder." The hebephrenic schizophrenic patients were typically disorganized, incoherent, and illogical in a context of poverty of speech and content, an indication of "negative thought disorder." Paranoid schizophrenic patients showed similar kinds of thought disorder to hebephrenic patients, but with less poverty of content and less incoherence. Schizoaffective patients also resembled the schizophrenics. At followup, thought disorder in the manics tended to be significantly reduced, even to normal levels, while schizophrenics continued to show disorganized thinking. Andreasen and Grove suggest that the fluency dimension may be a critical one for distinguishing between affective psychosis and "core schizophrenia." They also note that while the thought disorder shown by the schizoaffective patients resembles that of the schizophrenics, at followup it appears to normalize, suggesting the more benign course of the manics. Andreasen and Grove suggest that the different qualities of thought disorder represent "different mechanisms" in the different psychoses. It is clear from their data, moreover, that the thought disorder of schizophrenic patients appears not only as impoverishment of content and speech, but as incoherence and illogicality, which suggests that the negative-positive dichotomy does not capture the richer yield of Andreasen's scale.

Holzman, Shenton, and Solovay argue that the disorders under consideration are properly considered thought disorders and not speech or language disorders. They used the Thought Disorder Index to examine the quality of thought disorder in a group of schizophrenics, schizoaffective patients (both manic and depressed subtypes), manics, and normals. Although the groups did not differ significantly with respect to total amount of thought disorder present, there were distinct differences in the kinds of thought disorder each of the psychotic groups displayed. In this respect, their results are congruent with those Andreasen and Grove reported. Manics showed loosely tied together ideas that were generally extravagantly combined, and a playful, breezy style of speaking. Schizophrenic thinking showed none of the manics' jocularity. Their thinking was fluid, unstable, overly concise, and confused, with a significant number of word and phrase peculiarities. Schizoaffective patients, for the most part, resembled the schizophrenic patients, although the depressed subtype tended to be very constricted and less productive than the manic subtype. The authors conclude that, from the vantage point of thought disorder, schizoaffective disorder should be considered a schizophrenic disorder, a conclusion that requires separate validation from other approaches.

Harrow and Marengo, using their thought disorder assessment techniques, report a longitudinal examination of thought disorder in schizophrenic, psychotic nonschizophrenic, and nonpsychotic patients. They observed that most of the thought disorder occurred in the schizophrenic and manic groups. The schizophrenic patients, however, generally showed the most severe kinds of thought disorder, a finding also reported by Holzman, Shenton, and Solovay. Interestingly, at followup 1½–2 years after discharge and again 2 years after the first followup, it was the schizophrenic patients who typically showed persistent or episodic thought disorder. The appearance of very severe thought disorder during hospitalization was associated with significantly poorer general functioning outside the hospital. Although thought disorder was measurably present in all psychotic groups, it appeared to be a "key symptom" in the schizophrenic patients.

Spohn et al. report the effects of neuroleptic treatment on the thought disorder of 100 chronic schizophrenic patients, all of whom showed a therapeutic response to drug therapy. Using the Thought Disorder Index as the measuring instrument, they found that compared to bipolar patients and normal controls, the schizophrenic and schizoaffective patients show significantly more thought disorder, a finding consistent with those in the first two articles. The total thought disorder of schizoaffective patients is similar to that of schizophrenic patients, and both groups resemble "Bleulerian thought disorder." In general, thought disorder in chronic schizophrenia is not reduced to normal levels by drugs, although the most severe levels of thought disorder are affected. Spohn et al. suggest that the mild and minimal levels are not significantly attenuated. The residual thought disorder may reflect a central nervous system cognitive deficit. They entertain two hypotheses for how the neuroleptic drugs affect thought disorder: the drugs either act on cognitive pathways or increase social awareness, resulting in inhibited expression of disordered thinking. The authors prefer the first hypothesis.

Nuechterlein et al. ask to what extent schizophrenic symptoms, particularly disorders of thinking, are related to impairments in attention and information processing. They thus raise the question Bleuler had earlier proposed. They use a span of apprehension test and a special version of the continuous performance test (CPT), which uses blurred figures to make the task more difficult. Both tasks require effortful information processing, in addition to their other performance requirements. The authors examined 40 schizophrenic patients at the time of their hospital stay and 32 of them after their hospital discharge to determine the stability of the information-processing performance and the relation of that performance to thought disorder, as measured by the Thought Disorder Index and by subscales of the Brief Psychiatric Rating Scale (BPRS).

Only moderate but significant correlations were obtained between the CPT and span of apprehension, on the one hand, and fluid and perhaps confused thinking, on the other. These modest correlations were obtained in the schizophrenic group but not in the manic group. Their results are interesting in light of the probability that any valid measure of thought disorder contains both trait and state factors. Both the CPT and the span of apprehension test are moderately stable over time, suggesting that they may contain more "trait" variance than do the thought disorder assessments. The correlations obtained by the Nuechterlein group are, therefore, quite consistent with an interpretation that suggests trait characteristics for both thought disorder and the attentional measures in schizophrenic but not in manic patients. Similar correlations were obtained between the information-processing tests and the anergia scale of the BPRS, which the authors call a "negative" symptom. With their measures, they identify a constellation in schizophrenic patients that consists of (1) impaired active, effortful attention and information processing, (2) affective flattening, (3) motor retardation, and (4) a quality of thought disorder characterized by fluid and perhaps confused thinking. This constellation strongly resembles part of the constellation Bleuler identified as the primary symptoms of schizophrenia.

Knight et al., like Harrow and Marengo, attempt to predict outcome from thought disorder. They did not use a special scale to measure thought disorder, but constructed one from an inventory of patient characteristics, the Psychotic Inpatient Profile (PIP). They report that a measure of attentional impairment, inferred from the PIP, is related to outcome, such that the greater the attentional impairment, the worse the clinical outcome. They found only very weak support for the classification of thought disorder into positive and negative types, inasmuch as poverty of speech, defined as a "negative" thought disorder symptom, was not related to outcome. They argue that a variable like attentional impairment is a very complex one, and probably reflects different dynamic processes in different psychotic conditions. That is an argument whose pursuit requires more finely honed measures than the authors employed.

The two articles that follow use some of the new techniques of cognitive psychology to explore the cognitive processes that underlie thought disorders. Saccuzzo and Braff, using the method of backward masking, ask how quickly information is introduced into and then processed by the central nervous system of schizophrenics. The technique of backward masking permits one to measure how long it takes for a target to register and then for that engram to move from the stage of registration to short-term memory. For example, flash the letter B on a screen long enough for the subject to recognize it. After that recognition threshold has been established, flash the B again for the duration of that established recognition time (called the "critical recognition threshold"). Now, 120 ms later, follow that presentation by flashing a pattern of X's. The subject now reports seeing the X's but not the B. If the X's follow the B by, say, 500 ms, the subject reports seeing both the B and the X's successively. The second target, the X's, has acted as a mask that interfered with the experience of having seen the first target, the B. The critical time period between the two targets varies between subjects, but for schizophrenic subjects, the mask is effective for as long as 500 ms, whereas it ceases to be effective at about 250 ms in normal subjects.

Saccuzzo and Braff are particularly interested in identifying the stage of information processing at which dysfunctions can be reliably seen—whether early or late in that process. They tested schizophrenic, schizoaffective, bipolar, and depressed patients. All psychotic patients showed a persistent effect of the backward mask, which indicates, the authors interpret, that processing is slower in psychotic patients than in normal subjects. Presumably information is registered, but only sluggishly transferred to the next stages of cognitive processing. Because of that sluggishness, the authors reason, less information becomes available for processing and for working over. This is an interesting interpretation, which the authors pit against an alternative: too much information is getting through, and both relevant and irrelevant information is being processed—a state of affairs that overloads the system. A program of studies can be instituted that will help to decide the issue. The authors do present cogent evidence for a time-linked impairment in the information-processing sequence, and they attempt to relate that impairment to the appearance of thought disorder in psychosis.

Patterson et al., in a preliminary report of work in progress, focus on the very early epochs of information processing in schizophrenic patients, those that occur less than 100 ms after the stimulus is received. The authors specifically want to ascertain whether there is a defect in sensory storage, slow information processing, failure to organize the responses, or in all of them. If the earliest stage of information processing is intact, the authors reason, it is likely that the defects occur later in the process. They, like Saccuzzo and Braff, used a backward masking paradigm. They recorded evoked potentials at the time of target presentation and at the time of presentation of the mask, and they used topographic mapping techniques to present a picture of the evoked potential at any one instant in the process. They tested 19 schizophrenic patients, 15 nonschizophrenic psychotic patients (consisting of both bipolar and schizoaffective patients), and 31 normal control subjects. They found clear differences between the normal group and both groups of psychotic patients, with great overlap between the two groups of patients. The differences occurred at 80 to 105 ms after the onset of the target, while the stimulus was still exposed. There is, thus, a difference between the normal and the psychotic groups in information registration that may translate into a dysfunction in expectancy and in the capacity to recognize incongruity. The specificity of this finding for schizophrenia is ambiguous, since the composition of the schizoaffective group, which the authors consider to be a nonschizophrenic group, may not, in fact, be different from that of the schizophrenic group. The purity of the nonschizophrenic group may therefore be compromised. The authors speculate that hippocampal and thalamic brain structures and processes may be involved.

Altogether, these empirical studies suggest that thought disorder in schizophrenic patients tends to have significant trait characteristics since it is present both during a psychotic episode and, in milder forms, after a psychotic episode. It is likely that thought disorder is a key symptom of schizophrenic conditions. Thought disorder has many manifestations, and schizophrenia can be distinguished from manic conditions on the basis of those qualities which can now be described with greater precision than heretofore. It is not clear whether schizophrenic thought disorder should be characterized as "negative," since many types of thought disorder do not easily fit into that category. It is probably more useful to describe the various qualities of thought disorder that can be reliably distinguished in schizophrenic conditions and then to look for their correlates. Severe thought disorder is responsive to drug treatment, but in schizophrenia there are residual thinking disorders. As a trait in schizophrenic conditions, thought disorders reflect as yet unidentified central nervous system pathology that involves very early stages of information processing and that implicates attentional grasp.

Three manuals of thought disorder assessment accompany these articles: the Assessment of Thought, Language, and Communication; the Thought Disorder Index; and the composite method used by Harrow and his associates. The editor expresses his hope that these articles will provide a departure point for psychopathologists to deepen, broaden, and clarify the cognitive dysfunctions in schizophrenic conditions. Some of the methods described here can be applied to populations at risk for psychotic conditions. They can also be used to help infer brain processes that may be involved in cognitive disorders, to aid in early detection of schizophrenic conditions, and to chart response to various therapeutic interventions.

References

Bleuler, E. *Textbook of Psychiatry*. Translated by A.A. Brill. New York: MacMillan Company, 1924.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias*. (1911) Translated by J. Zinkin. New York: International Universities Press, 1950.

Kety, S.S. Biochemical theories of schizophrenia. *Science*, 129 (Part 1):1528–1532; (Part 2):1590–1596, 1959.

Kraepelin, E. *Dementia Praecox and Paraphrenia*. (1896) Translated by R.M. Barclay. Chicago: Chicago Medical Books, 1919.

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Foundations' Fund Prize for Research in Psychiatry

The American Psychiatric Association takes pleasure in inviting submissions for the Tenth Annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for this prize should be citizens of the United States or Canada and should be nominated by a sponsor. Sponsors should be members of the American Psychiatric Association. Members of the Prize Board are excluded from submitting nominations.

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The *nominee* should submit:

- A book or paper, or a group of representative, and *thematically linked,* books or papers published (or accepted for publication) in English and dated within 10 years before the deadline of submission.
- A summary statement written by the nominee, emphasizing the *principal theme* (or themes) running through the work, its internal cohesiveness and consistency, and scientific implications.
- An up-to-date curriculum vitae.
- An up-to-date bibliography.

All entries must be submitted in six copies (including six copies each of the sponsor's letter, curriculum vitae, bibliography, and summary statement) and sent to Ira D. Glick, M.D., Chairman, Foundations' Fund Prize Board for Research in Psychiatry, American Psychiatric Association, 1400 K Street, N.W., Washington, DC 20005. Entries will be acknowledged, but cannot be returned. The prize is based on a yearly competition and resubmission is encouraged. The award will be presented at the Convocation of Fellows at the Association's Annual Meeting in May of 1987.

The deadline for submission is November 1, 1986.

Thought, Language, and Communication in Schizophrenia: Diagnosis and Prognosis

by Nancy C. Andreasen and William M. Grove

Abstract

Using the Scale for the Assessment of Thought, Language, and Communication (TLC), we examined the frequency of "thought disorder" in 94 normal volunteers and 100 psychiatric patients (25 each suffering from manic disorder, schizoaffective disorder, schizophrenic disorder, disorganized type, and schizophrenic disorder, paranoid type). We observed the manics to have a substantial amount of thought disorder and the normals to have a modest amount, suggesting that thought disorder is probably not pathognomonic of schizophrenia. The patients with affective illness did, however, show a somewhat different pattern of abnormality. In particular, patients with affective psychosis have more prominent positive thought disorder, while the schizophrenic patients tend to have more negative thought disorder. Evaluation of the patients 6 months later indicated that most types of thought disorder remit in the manics, while they persist in the schizophrenics; patients with schizoaffective disorder also tend to improve substantially. The strongest predictor of outcome was the presence of negative thought disorder.

Ever since the concept of thought disorder was given preeminence in Bleuler's (1950) conceptualization of schizophrenia, the study of this important symptom (or sign) has been plagued by the absence of a common ground of agreement concerning its definition or the best methods for assessing it. Bleuler's description of associative loosening is relatively vague, and until the recent neo-Kraepelinian revival, Kraepelin's (1919) early descriptions of thought and language abnormalities were rarely consulted.

The Scale for the Assessment of Thought, Language, and Communication (TLC) was originally developed in order to remedy this problem and to provide a consistent set of definitions that could be used clinically and that would have high reliability (Andreasen 1978, 1979*a*, 1979*b*). Because these definitions were designed for use in a clinical setting, they rely heavily on the naturalistic observation of language behavior. While it is also possible to obtain indices of cognitive performance or "thought" using formal tests to elicit disordered thinking, such as proverb interpretation or projective tests (Gorham 1956; Shimkunas, Gynther, and Smith 1967; Andreasen, Tsuang, and Canter 1974; Johnston and Holzman 1979), in a clinical setting we usually infer a person's thoughts directly from his speech. Consequently, the set of definitions in the TLC emphasizes the observation of language behavior as a way of evaluating thought disorder.

In our early work with these definitions, we studied a sample of 113 patients suffering from mania, depression, and schizophrenia (Andreasen 1979*a*, 1979*b*). We observed that the traditional concept of thought disorder, which emphasizes the importance of associative loosening, was not useful diagnostically, since many patients suffering from mania also exhibited associative loosening. On the other hand, we observed some differences between manic and schizophrenic patients in their overall pattern of subtypes of thought disorder. In particular, a distinction between positive and negative formal thought disorder seemed to be useful, since patients

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suffering from schizophrenia tended to have prominent poverty of speech and poverty of content while manic patients did not. We concluded that "thought disorder" is probably not pathognomonic of schizophrenia, as Bleuler originally suggested, although some patients with schizophrenia may show a type of thought disorder that is characterized by intellectual impoverishment and that is somewhat more characteristic of schizophrenia.

Since our original development of the TLC, a number of other investigators have explored its usefulness (Harvey 1983; Harvey, Earle-Boyer, and Wielgus 1984; Berenbaum, Oltmanns, and Gottesman 1985; Simpson and Davis 1985; Davis et al., in press). They have found it to be highly reliable in a variety of different settings and have supported the distinction between positive and negative formal thought disorder.

In our own work on thought and language in schizophrenia, we felt it was important to amplify our original studies of thought, language, and communication in several different ways. In particular, we were interested in observing the extent to which normal individuals exhibit the types of TLC abnormality observed in schizophrenic patients. Further, we were interested in examining the frequency of thought, language, and communication abnormalities in a transitional diagnostic group, patients with schizoaffective disorder, and in subtypes of schizophrenia. Finally, we were interested in exploring whether either severity or type of thought disorder is predictive of outcome when patients are followed longitudinally.

Methods

The patients in this study were stratified into four diagnostic groups: 25 each suffering from manic disorder; schizoaffective disorder; schizophrenic disorder, disorganized or hebephrenic subtype; and schizophrenic disorder, paranoid subtype. Patients for each of these groups were recruited from consecutive admissions to the University of Iowa Psychiatric Hospital. All diagnoses were made using the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978). RDC diagnoses were made based on data collected using the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978).

A comparison or control group consisted of 94 normal individuals recruited from Iowa City and nearby communities. Recruits were obtained through advertising in a local newspaper. In order to match the controls to the patients as closely as possible, normal individuals were included only if they were under age 40 and had less than 2 years' college education. The normals were screened for past history of major psychiatric illness (limited to those diagnoses defined by the RDC), and volunteers who had a current or past history of these illnesses were excluded.

All subjects in the study were evaluated using the Scale for the Assessment of Thought, Language, and Communication (TLC) (Andreasen 1978). This instrument contains definitions of 18 subtypes of "thought disorder" and has been found to have good interrater reliability. The scale contains both the definitions and instructions for rating severity on a 0–3 or 0–4 scale (depending on the item). To ensure good reliability, definitions are relatively specific, and judgment as to severity for each of the individual items usually depends on the frequency with which a particular phenomenon has been observed. Since such frequencies may vary with the length of the interview, we have used a standard 45-minute interview.

All subjects were evaluated with a standardized interview designed to avoid a discussion of the patient's psychopathology (available from N.C.A. on request). This interview begins by having each subject talk about himself for as long as possible (for at least 5 minutes) without interruption, in response to the prompt, "Could you tell me a little bit about what you're like, such as where you're from, what you're interested in, and things like that?" If the patient had difficulty in elaborating his response, nonspecific prompts such as "Could you tell me a little more about that?" were used. After the initial monologue, additional prespecified questions in the interview covered a variety of topics, such as politics, religious beliefs, and family life. Ratings of thought, language, and communication were done "live," and score sheets were filled out immediately after the interview, and after the patient had left the room. Because it defines 18 types of thought disorder, the TLC is a complicated scale; consequently, raters usually make notes during the interview about the frequency of individual phenomena and complete the score sheet immediately after the interview. Interviews were conducted either by one of the authors (W.M.G.) or by a research assistant with a bachelor's degree in psychology who had been trained in the use of the scale by one of the authors. All interviews were also tape-recorded and transcribed for later analysis, but all the data in this article are based on the live interviews.

The standardized interview covering neutral topics was used in order to keep the interviewer as blind to the patient's diagnosis as possible. Discussion of the patient's psychopathology was avoided. Nevertheless, in a "live" situation, some clues are inevitably provided. Quite simply, manics tend to talk more and more rapidly, and normals tend to appear relatively normal and unmedicated. Thus, the use of live ratings does introduce some potential bias which could be avoided by using either tapes or transcribed samples. For the present investigation, however, our primary goal was to obtain the most accurate ratings of thought disorder that would be possible, and we felt this goal was best achieved through the use of live interviews. We have not as yet completed ratings of thought disorder using live *versus* taped *versus* transcribed samples, but we anticipate that estimated severity is likely to increase as one becomes more removed from the live situation, since intonations, gestures, and interpersonal interaction tend to make speech appear more comprehensible. Thus, in this investigation, we used all possible safeguards to maximize blindness, but we recognize that blindness is probably not complete. Evaluation of transcripts might provide the most rigorous test of the frequency of abnormalities in normals *versus* various groups of patients, although with the possible hazard of overestimating the degree of abnormality in all groups.

To give some indication of general intelligence, all normals and patients were also evaluated with the Shipley Institute of Living Scale.

Patients were evaluated during the first week after admission, usually in the first 3 days. Nearly all were receiving medication at the time of evaluation, since medications are usually prescribed at Iowa Psychiatric Hospital within the first day or two after admission to reduce length of stay. Despite this, all were still severely symptomatic and manifested a full and typical manic, schizophrenic, or schizoaffective syndrome. In a few instances, patients who were admitted consecutively and met inclusion diagnostic criteria had responded to medication so quickly that they no longer manifested the full manic syndrome during the first week; these patients were therefore not interviewed with the TLC and not included in the study.

We conducted a followup evaluation with the patients 6 months after the index evaluation. At followup, all available patients were examined with an abridged version of the SADS (SADS-C) and with the Global Assessment Scale (GAS) (Endicott et al. 1976) and the TLC. We were able to obtain followup evaluations on approximately 70 percent of the original sample. These were divided evenly across the various diagnostic cells. No significant differences in diagnosis, sex ratio, age, or severity of illness were observed between those patients whom we were successful in following up and those who could not be interviewed at the followup evaluation.

Results

Demographic and other characteristics of the subjects are summarized in table 1. In general, all subjects are relatively similar in age, educational status, sex ratio, and intelligence (as assessed by the Shipley). As might be expected, the patients do differ substantially from one another in duration of the current episode and total amount of time spent in the hospital during previous episodes. In particular, the two schizophrenic groups had consid-

Table 1. Demographic and other characteristics of subjects

	Normals (*n* = 94)		Manics (*n* = 25)		Schizo-affectives (*n* = 25)		Hebephrenics (*n* = 25)		Paranoids (*n* = 25)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	28.92	9.13	35.04	14.01	28.16	9.50	26.68	7.41	27.75	6.21
n (%) female	56	(59)	13	(52)	14	(56)	12	(48)	8	(32)
Education, years	14.10	3.42	13.38	2.24	13.50	1.89	12.56	1.66	13.74	2.77
Shipley-Hartford	115.48	7.71	102.88	11.98	108.00	10.09	95.26	12.88	104.00	9.80
Duration of episode, weeks			9.88	9.63	64.48	164.44	246.96	239.55	256.58	262.24
Time in hospital, total weeks			28.46	30.84	41.72	76.27	66.71	160.85	79.71	139.76

erably greater chronicity, while the manics had the least chronicity.

Table 2 indicates the frequencies with which the various types of thought, language, and communication abnormalities were observed in the normal individuals and the four patient groups. It is interesting to note that some TLC "abnormalities" occur in the speech of normal individuals; in particular, the normals showed a relatively large amount of derailment and loss of goal. The patient groups differ significantly from one another on nearly all types of language abnormality that occur with any frequency. Manic speech tends to be fluent and disorganized, as manifested by a high rate of pressure of speech, derailment, loss of goal, circumstantiality, incoherence, and illogicality. The schizoaffectives show a less severe but similar type of abnormality, with a somewhat greater frequency of poverty of speech and a lesser frequency of pressure of speech. Hebephrenic speech tends to be empty (high ratings on poverty of speech and poverty of content), but also very disorganized (high frequency of derailment, incoherence, and illogicality). The paranoid patients show a similar but less severe pattern, particularly with less poverty of content and incoherence.

Following are illustrative examples of some of the abnormalities observed in these various groups:

Normal

I: Tell me about your work. What do you like about it the most, and why?

P: Oh, it's easy work for the pay. I guess that's what I like about it the best. It has good benefits. Uh, my hours are pretty good, except when I have to work 8-hour days. Sometimes I get stuck with the 10 to 7 shift. That's kind of a drag. Um, it's mostly older people that come into the grocery store, since it's up in the Polish neighborhood, I guess, of Iowa City. I think they come there mostly for somebody to talk to. They don't have anything else to do, most of them are retired. Uh, I like to sew. I went and got a new sewing machine this week, and I plan on making a dress for Noelle for Christmas. I like to swim, I haven't had the chance since I was pregnant

Table 2. Frequencies of thought, language, and communication abnormalities [1] among groups

	Normals		Manics		Schizo-affectives		Hebe-phrenics		Paranoids		Chi-square[2]	p
	n	%	n	%	n	%	n	%	n	%		
Poverty of speech	5	5	1	4	6	24	6	24	9	36	22.04	.01
Poverty of content	1	1	2	8	3	12	12	48	2	8	47.13	.01
Pressure of speech	6	6	22	88	8	32	6	24	4	16	73.94	.01
Distractible speech	3	3	5	20	3	12	3	12	0	0	11.91	.02
Tangentiality	2	2	1	4	2	8	6	24	4	16	16.14	.01
Derailment	30	32	20	80	14	56	17	68	14	56	24.63	.01
Incoherence	0	0	6	24	2	8	10	40	5	20	36.76	.01
Illogicality	0	0	9	36	3	12	12	48	3	12	49.39	.01
Clanging	0	0	1	4	0	0	1	4	1	4	4.80	.31
Neologisms	0	0	0	0	0	0	0	0	0	0	—	—
Word approximations	2	2	4	16	0	0	1	4	2	8	10.42	.03
Circumstantiality	6	6	11	44	4	16	4	16	4	16	21.85	.01
Loss of goal	17	18	13	52	5	20	8	32	7	28	12.68	.01
Perseveration	7	8	11	44	5	20	8	32	4	16	21.69	.01
Echolalia	0	0	0	0	0	0	0	0	0	0	—	—
Blocking	1	1	0	0	0	0	0	0	0	0	1.08	.90
Stilted speech	1	1	4	16	2	8	1	4	0	0	13.07	.01
Self-reference	1	1	1	4	0	0	0	0	0	0	2.93	.57
Global rating	6	6	19	76	15	60	20	80	14	56	81.61	.01

[1] Criterion: Rating > 1.

[2] Between patient groups, normals excluded.

and I haven't gone back yet. I like little kids. I hope I'll have some more, but I don't know whether I'll be able to afford it. Um, let me think. I voted today.

Manic

I: So, to begin with, could you tell me a little bit about yourself, things like where you're from—

P: I'm from Cedar Rapids, and I live with my parents. My parents are the holy family, part of it. My brother lives in Omaha, and he's part of the holy family. He's Jesus. I'm the daughter of God, and my sister's pregnant and she has a part in the holy family and God knows what I'm gonna do next. God has preplanned, has planned my life, and I'm not afraid to speak for the, for the unfortunate as for the fortunate, and for the stars and for Stella and for, and for, and for myself. I'm a big star. I just haven't grown enough, um, God and the Holy Spirit help me. And without, without my friends I would be nothing. Without my music I'm a vegetable. Without my, my nurses which have, heroes, the women are the heroes, the men are the heroes, but there's a champion that, that's been suffering. That all now?

Schizophrenic

I: Why don't you tell me a little bit about what you think about current political issues, like the energy crisis, for example.

P: They're destroying too many cattle and oil just to make soap. If we need soap when you can jump into a pool of water and then when you go to buy your gasoline, m-my folks always thought they should, get pop but the best thing to get, is motor oil, and money. May as well go there and trade in some, pop caps and, uh, tires, and tractors to grup, car garages, so they can pull cars away from wrecks, is what I believed in.

To obtain a perspective on how patients' speech differs from normal speech in its descriptive characteristics, we compared the mean scores for each of the patient groups to that of the normals. Because of the large number of comparisons, we used a somewhat conservative .01 significance level, rather than the more customary .05 level. At this level, the manics showed significant differences on pressure of speech, distractibility, derailment, incoherence, illogicality, circumstantiality, loss of goal, and perseveration. The schizoaffectives showed fewer differences, having only more pressured speech, derailment, and illogicality. The hebephrenic patients had significantly higher ratings on poverty of speech, poverty of content, tangentiality, derailment, incoherence, illogicality, word approximations, and perseveration. The paranoids had significantly higher ratings on poverty of speech, poverty of content, derailment, incoherence, and illogicality.

Tables 3-6 explore our ability to replicate our previous work. One major aspect was the division of TLC abnormalities into positive *versus* negative thought disorder. In our previous study, this concept was more useful in distinguishing manics from schizophrenics than was a description involving loose associations. Table 3 shows the mean scale scores of the four diagnostic groups for positive *versus* negative formal thought disorder and loose associations. The score for positive *versus* negative formal thought disorder was developed by subtracting two indices of negative formal thought disorder (poverty of speech and poverty of content) from the sum of pressure of speech, tangentiality, derailment, incoherence, and illogicality. The score for loose associations consists of a sum of five types of thought disorder often considered to be manifestations of loose associations: tangentiality, derailment, incoherence, illogicality, and clanging.

Table 3 also shows an analysis of variance (ANOVA) for positive *versus* negative formal thought disorder. The four groups differ significantly on this variable at the .01 level. Followup tests indicate that the significance is due to differences between manics and other patients, manics and schizoaffectives, and manics and schizophrenics. Thus, the results of this second study confirm the usefulness of the positive *versus* negative distinction in discriminating between manic formal thought disorder and various types of schizophrenic formal thought disorder.

Table 3 also shows the ANOVA for loose associations. This ANOVA is also significant, although to a somewhat lesser degree. The followup tests indicate that this construct may be more useful in discriminating between various traditional subtypes of schizophrenia. The followup tests indicate significant contrasts between the schizoaffectives and the schizophrenics, and between the hebephrenics and the paranoids. In our 1979 article (Andreasen 1979*a*, 1979*b*), we did not find significant differences between manics and schizophrenics when we used a similar measure of "loose associations." We did not, however, stratify by schizophrenic subtype or include schizoaffectives in the sample; therefore, we could not make subtype comparisons.

Table 4 compares the discriminant function weights generated from our earlier work and from the present investigation. The weights developed from the two samples are somewhat different. In our first sample, poverty of speech and derailment were somewhat more important as discriminators than they are in the

Table 3. Comparison of diagnostic groups on formal thought disorder ratings[1]

Variable	Manics ($n = 25$) Mean	Manics ($n = 25$) SD	Schizoaffectives ($n = 25$) Mean	Schizoaffectives ($n = 25$) SD	Hebephrenics ($n = 24$) Mean	Hebephrenics ($n = 24$) SD	Paranoids ($n = 25$) Mean	Paranoids ($n = 25$) SD
Positive-negative formal thought disorder[2]	169.55	30.32	141.13	28.36	151.05	40.11	138.27	38.17
Loose associations[3]	252.49	29.85	236.99	18.66	264.81	38.75	245.71	33.32

	ANOVA for positive-negative formal thought disorder					ANOVA for loose associations				
Source	*df*	Sums of squares	Mean square	*F*	*p*	*df*	Sum of squares	Mean square	*F*	*p*
Groups	3	14984.97	4994.99	4.17	.01	3	10329.46	3443.15	3.58	.02
Error	96	114945.60	1197.35			96	92284.18	961.29		
Total	99	129930.57				99	102613.64			
Contrasts	*df*			*t*	*p*	*df*			*t*	*p*
Manic vs. other patients	1			3.26	.01	1			.46	.65
Schizoaffective vs. schizophrenic	1			− .42	.68	1			− 2.41	.02
Hebephrenic vs. paranoid	1			1.31	.19	1			2.18	.04
Affective symptoms vs. nonaffective	1			1.54	.13	1			− 1.70	.10
Manic vs. schizoaffective	1			2.90	.01	1			1.77	.08
Manic vs. schizophrenic	1			2.94	.01	1			.37	.72

[1] Individual ratings were transformed to have a mean of 50 and a standard deviation of 10.
[2] Sum of pressure of speech, tangentiality, derailment, incoherence, and illogicality, less sum of poverty of speech and poverty of content.
[3] Sum of tangentiality, derailment, incoherence, illogicality, and clanging.

present sample, while in the current sample, illogicality has been included with a negative weighting. However, the 1979 discriminant function performed very successfully in classifying the second replication sample. Using the 1979 function on the 1979 sample, we correctly classified 84 percent of manics and schizophrenics. When applied to the present sample for class cross-validation, this function correctly classifies 80 percent of the cases. We also used jackknife procedures in order to apply the new function derived from the current sample and found that it correctly classified 83 percent of that sample. These results provide a relatively strong confirmation of our earlier work, suggesting that examining various subtypes of TLC abnormality may be quite useful in distinguishing manics from schizophrenics on the basis of their language behavior.

Tables 5 and 6 compare the frequency of TLC abnormalities in the 1979 and the current sample in order to explore what might be called replicability. The ratings on the current sample were completed by different interviewers from those who collected the 1979 sample. The patients were, of course, completely different, although the size of the diagnostic cells for mania and schizophrenia is quite similar. The frequency of ratings shows surprising stability from one study to another. The only significant differences between the two studies are the global rating for schizophrenics and the rating of tangentiality for manics.

Table 7 contains an orthogonal factor analysis of the TLC variables. The results of this analysis yielded three factors from Cattell's scree test. After rotation to simple structure by normalized Varimax rotation, the first factor might be termed a "fluent disorganization" factor, with high

Table 4. Comparison of discriminant functions between manics and schizophrenics

Variable	Standardized discriminant weight Function from 1979 sample (n = 77)	Function from replication sample (n = 75)
Poverty of speech	–.261	—
Poverty of content	–.351	–.457
Pressure of speech	.634	.898
Distractible speech	.422	.685
Derailment	–.406	—
Illogicality	—	–.390
Loss of goal	.223	—
% Correctly classified	80[1]	82.7[2]

[1] Cross-validation performance.

[2] Estimated performance by jackknife.

positive loadings on pressure of speech, derailment, incoherence, illogicality, loss of goal, and perseveration, and a high negative loading on poverty of speech. Factor 2 appears to be an "emptiness" factor, with high positive loadings on poverty of speech, poverty of content, and tangentiality and a negative loading on pressure of speech and circumstantiality. Factor 3 might be considered to be a "linguistic control" factor, with high positive loadings on stilted speech and persistent self-reference and negative loadings on clanging and neologisms. The components in this third factor occur so infrequently,

Table 5. Comparison of thought, language, and communication abnormalities[1] for schizophrenics: 1979 vs. replication sample

Variable	1979 sample (n = 45) n	%	Replication sample (n = 50) n	%	Chi-square[2]	p[1]
Poverty of speech	13	29	15	30	.04	.84
Poverty of content	18	40	14	28	1.53	.22
Pressure of speech	12	27	10	20	.59	.56
Distractible speech	1	2	3	6	—	.35
Tangentiality	16	36	10	20	2.88	.09
Derailment	25	56	31	62	.41	.52
Incoherence	7	16	15	30	2.78	.10
Illogicality	12	27	15	30	.13	.72
Clanging	0	0	2	4	—	.27
Neologisms	1	2	0	0	—	.47
Word approximations	0	0	3	6	—	.14
Circumstantiality	2	4	8	16	3.36	.07
Loss of goal	20	44	15	30	2.12	.15
Perseveration	11	24	12	24	2.41	.12
Echolalia	2	4	0	0	—	.22
Blocking	2	4	0	0	—	.22
Stilted speech	1	2	1	2	—	.77
Self-reference	6	13	0	0	—	.01
Global rating	41	91	34	68	7.61	.01

[1] Criterion: rating > 1.

[2] Where no statistic is reported, Fisher's exact test was used.

Table 6. Comparison of thought, language, and communication abnormalities[1] for manics: 1979 vs. replication sample

Variable	1979 sample (n = 32)		Replication sample (n = 25)			
	n	%	n	%	Chi-square[2]	p[1]
Poverty of speech	2	6	1	4	—	.59
Poverty of content	6	19	2	8	—	.22
Pressure of speech	23	72	22	88	2.20	.14
Distractible speech	10	31	5	2-	.92	.33
Tangentiality	11	31	1	4	7.79	.02
Derailment	18	56	20	80	3.56	.06
Incoherence	5	16	6	24	.63	.43
Illogicality	8	25	9	36	.81	.37
Clanging	3	9	1	4	—	.40
Neologisms	1	3	0	0	—	.56
Word approximations	1	3	4	16	—	.11
Circumstantiality	8	25	11	44	2.28	.13
Loss of goal	14	44	13	52	.38	.64
Perseveration	11	34	11	44	.55	.46
Echolalia	1	3	0	0	—	.56
Blocking	1	3	0	0	—	.56
Stilted speech	2	6	4	16	—	.22
Self-reference	7	22	1	4	—	.06
Global rating	28	88	19	76	1.28	.74

[1] Criterion: rating > 1.

[2] Where no statistic is reported, Fisher's exact test was used.

however, that it cannot be considered to be a very important one.

Table 8 shows the frequency of TLC abnormalities in the various diagnostic groups at the time of followup. We did not follow up the normal individuals, but their rates at the index evaluation are included in table 8 for comparison. As table 9 indicates, the manic patients have remitted significantly at followup, as have the schizoaffectives. The speech of the manics has essentially normalized. So, too, has that of the schizoaffectives, although they continue to have a somewhat high rate of poverty of speech. On the other hand, the schizophrenic patients continue to have relatively persistent disorganization. When mean ratings between index evaluation and followup evaluation are compared, the hebephrenics show a significant change only in pressure of speech and incoherence (in the direction of improvement), while the paranoids show no significant differences. Thus, the language abnormalities in the schizophrenics tend to persist at followup.

We were interested in determining whether the type and amount of thought disorder shown by these patients was in any sense predictive of the severity of their syndrome at followup. Therefore, we examined the correlation between outcome, as measured by the Global Assessment Scale, and positive *versus* negative thought disorder (as previously defined). Table 9 shows that negative formal thought disorder is moderately correlated with outcome, as indicated by absolute GAS rating and by the degree of change in the GAS from index evaluation to followup. Thus, the presence of negative formal thought at index evaluation predicts a poor outcome at followup. On the other hand, positive formal thought disorder has little prognostic significance when used to predict outcome among psychotic patients in general. The lack of correlation appears to occur because manic patients, who have a relatively high rate of positive formal

Table 7. Factor analysis of thought, language, and communication variables among patients

	Loading on factor			
Variable	1 Fluent disorganization	2 Emptiness	3 Linguistic control	Commonality
Poverty of speech	−.592	.416	.167	.551
Poverty of content	.236	.609	.055	.430
Pressure of speech	.648	−.462	−.078	.639
Distractible speech	.428	−.158	−.208	.251
Tangentiality	.251	.464	−.135	.297
Derailment	.775	.021	−.112	.614
Incoherence	.632	.365	−.089	.541
Illogicality	.547	.128	.265	.386
Clanging	.443	.323	−.466	.518
Neologisms	.227	.238	−.353	.233
Word approximations	.501	.256	.358	.445
Circumstantiality	.501	−.493	−.017	.494
Loss of goal	.740	.019	−.244	.607
Perseveration	.746	−.052	.137	.578
Echolalia	.099	−.277	−.223	.136
Blocking	.036	.102	.288	.095
Stilted speech	.531	.087	.531	.571
Self-reference	.269	−.149	.673	.547
% of variance	25.5	9.7	8.9	
Cumulative % of variance	25.5	35.2	44.1	

thought disorder, have a good outcome, but its predictive power washes out when manic patients are pooled with schizophrenic patients (who may have a high rate of positive formal thought disorder and a relatively poor outcome).

Discussion

This larger and more extensive study of thought, language, and communication in schizophrenia, schizoaffective disorder, and mania has replicated a number of our previous findings and has elaborated them further.

Taken together with our previous work, the current study suggests that the global concept of "thought disorder" should not be considered to be pathognomonic of schizophrenia or diagnostic of it. The present study indicates that mild abnormalities in language behavior even occur in normal individuals. Derailment, the closest single equivalent to Bleuler's "associative loosening," is the most common type of "abnormality" observed. Further, patients with mania and schizoaffective disorder also display prominent abnormalities in thought, language, and communication. If anything, the abnormalities are more pronounced in patients with mania than in those with schizophrenia.

The present study provides additional support for the utility of subdividing the global concept of thought disorder into a variety of subtypes. The TLC distinguishes between 18 different subtypes. Some of these, however, are relatively uncommon; some types of thought disorder that have frequently been widely described and discussed in the past are among those that are relatively uncommon, such as blocking or neologisms. When the rate of the various subtypes of thought disorder is observed across four diagnostic groups, interesting differences in quality and severity of formal thought disorder are observed. The abnormalities seen in mania appear to be qualitatively different from those observed in schizophrenia. In particular, patients with mania have a more prominent positive thought disorder, while patients with schizophrenia have a more prominent negative formal thought disorder.

The distinction between positive and negative formal thought disorder

Table 8. Frequencies of thought, language, and communication abnormalities[1] among diagnostic groups at followup

Variable	Normal (*n* = 94)		Manic (*n* = 18)		Schizo-affective (*n* = 19)		Hebe-phrenic (*n* = 17)				Paranoid (*n* = 15)	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	Chi-square[2]	*p*
Poverty of speech	5	5	2	11	6	32	5	29	5	33	18.53	.01
Poverty of content	1	1	1	5	1	5	11	55	6	40	66.23	.01
Pressure of speech	6	6	4	22	2	11	2	12	0	0	6.48	.17
Distractible speech	3	3	1	6	0	0	0	0	0	0	2.22	.69
Tangentiality	2	2	2	11	3	16	9	53	0	0	44.08	.01
Derailment	30	32	6	33	4	21	10	59	5	33	6.22	.18
Incoherence	0	0	1	6	2	11	2	11	1	7	9.70	.05
Illogicality	0	0	1	6	1	5	7	41	2	13	39.66	.01
Clanging	0	0	0	0	0	0	0	0	0	0	—	—
Neologisms	0	0	0	0	0	0	1	6	0	0	8.58	.07
Word approximations	2	2	1	6	1	5	1	6	1	7	1.53	.82
Circumstantiality	6	6	2	11	6	32	6	35	0	0	19.89	.01
Loss of goal	17	18	5	28	1	5	4	24	0	0	7.13	.13
Perseveration	7	8	3	17	3	16	7	41	1	7	15.31	.01
Echolalia	0	0	0	0	0	0	0	0	0	0	—	—
Blocking	1	0	0	0	0	0	0	0	0	0	.75	.95
Stilted speech	1	1	1	6	3	16	2	12	1	7	9.52	.05
Self-reference	1	1	1	6	0	0	2	12	0	0	8.42	.08
Global rating	6	6	6	33	5	26	14	82	5	33	51.49	.01

[1] Criterion: Rating > 1.

[2] Between patient groups, normals excluded.

may have more clinical utility than the traditional concept of associative loosening. Both approaches to conceptualizing thought disorder were examined in the four diagnostic groups. The distinction between positive *versus* negative thought disorder was useful in separating manics from all other patients, from schizoaffectives, and from schizophrenics. On the other hand, associative loosening is significantly more severe in schizophrenics than in schizoaffectives, and within the schizophrenic group more severe in the hebephrenics than in the paranoids. These results suggest that, within the schizophrenia spectrum, the degree of disorganization (as manifested by associative loosening) may be useful in identifying more severe illnesses (i.e., pure schizophrenia *versus* schizoaffective disorder or hebephrenic *versus* paranoid schizophrenia). On the other hand, within the broad spectrum of psychosis, the fluency and productivity of language behavior may be more important for distinguishing between affective psychosis and "core" schizophrenia.

Table 9. Correlation between index formal thought disorder and outcome as measured by Global Assessment Scale

	GAS		Change in GAS	
	r	*p*	*r*	*p*
Positive FTD	−.13	.279	−.03	.796
Negative FTD	−.38	.002	−.28	.02

Note.—GAS = Global Assessment Scale; FTD = formal thought disorder.

When language behavior is observed longitudinally in psychotic patients, the patients appear to differ substantially in course and outcome. Patients suffering from mania tend to improve markedly and to have essentially reversible abnormalities. On the other hand, the schizophrenic patients tend to have relatively persistent abnormalities. Patients with schizoaffective disorder resemble the manics and improve, but to a somewhat lesser degree. This finding is interesting since, at index evaluation, the schizoaffectives are more similar to the schizophrenics; at outcome, however, they more closely resemble the manics. This finding is probably indicative of some heterogeneity within the schizoaffective group, but it may also indicate that schizoaffective disorder is a transitional diagnosis and (as defined by the RDC) represents an illness that resembles schizophrenia phenomenologically but which has a relatively better outcome. The overall good outcome in patients with pure manic phenomenology or some tincture of manic phenomenology suggests that the language disorganization in mania is probably due to a different mechanism than that occurring in schizophrenia.

The type of thought disorder observed initially may provide some prognostic indication. Patients who manifest a prominent negative thought disorder are less likely to have global improvement or remission, as indicated by the Global Assessment Scale. Thus, negative thought disorder, when present, may be predictive of a poor outcome. On the other hand, positive thought disorder is not a useful prognostic indicator. Some patients with positive thought disorder will remit or improve, while others will not.

Based on these observations, the Scale for the Assessment of Thought, Language, and Communication (TLC) appears to be a useful tool for the clinical assessment of language behavior in the major psychoses. The results show good consistency from one study to another, with relatively stable rates of the various TLC abnormalities across two different samples and good cross-validation of a discriminant function. Thus, the definitions that it contains appear to have utility both in the real world of the clinic and as a research tool.

References

Andreasen, N.C. *The Scale for the Assessment of Thought, Language, and Communication (TLC).* Iowa City: The University of Iowa, 1978.

Andreasen, N.C. Thought, language, and communication disorders: I. Clinical assessment, definition of terms, and evaluation of their reliability. *Archives of General Psychiatry,* 36:1315–1321, 1979*a*.

Andreasen, N.C. Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry,* 36:1325–1330, 1979*b*.

Andreasen, N.C.; Tsuang, M.T.; and Canter, A. The significance of thought disorder in diagnostic evaluation. *Comprehensive Psychiatry,* 15:27–34, 1974.

Berenbaum, H.; Oltmanns, T.F.; and Gottesman, I.I. Formal thought disorder in schizophrenics and their twins. *Journal of Abnormal Psychology,* 94:3–16, 1985.

Bleuler, E. *Dementia Praecox, or the Group of Schizophrenias.* (1911) Translated by J. Zinkin. New York: International Universities Press, 1950.

Davis, G.C.; Simpson, D.M.; Foster, D.; Arison, Z.; and Post, M. Reliability of Andreasen's Thought, Language, and Communication Scale. *Journal of Clinical Psychiatry,* in press.

Endicott, J., and Spitzer, R.L. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia (SADS). *Archives of General Psychiatry,* 35:837–844, 1978.

Endicott, J.; Spitzer, R.L.; Fleiss, J.; and Cohen, J. The Global Assessment Scale: A procedure for measuring overall severity of psychiatric illness. *Archives of General Psychiatry,* 33:766–771, 1976.

Gorham, D.R. Use of the proverbs test for differentiating schizophrenics from normals. *Journal of Consulting Psychology,* 20:435–440, 1956.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking.* San Francisco: Jossey-Bass, Publishers, 1979.

Kraepelin, E. *Dementia Praecox and Paraphrenia.* Translated by R.M. Barclay. Edinburgh: E. & S. Livingstone, 1919.

Harvey, P.D. Speech competence in manic and schizophrenic psychoses: The association between clinically rated thought disorder and cohesion and reference performance. *Journal of Abnormal Psychology,* 92:368–377, 1983.

Harvey, P.D.; Earle-Boyer, E.A.; and Wielgus, M.S. The consistency of thought disorder in mania and schizophrenia: An assessment of acute psychotics. *Journal of Nervous and Mental Disease,* 172:458–463, 1984.

Shimkunas, A.M.; Gynther, M.D.; and Smith, K. Schizophrenic responses to the proverbs test: Abstract, concrete, or autistic? *Journal of Abnormal Psychology,* 72:128–133, 1967.

Simpson, D.M., and Davis, G.C. Measuring thought disorder with clinical rating scales in schizophrenic and nonschizophrenic patients. *Psychiatry Research,* 15:313–318, 1985.

Spitzer, R.L.; Endicott, J.; and Robins, E. Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, 35:773–782, 1978.

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Quality of Thought Disorder in Differential Diagnosis

by Philip S. Holzman, Martha E. Shenton, and Margie R. Solovay

Abstract

The authors used a reliable scale to assess quantity and quality of thought disorder in manic, schizophrenic, schizoaffective manic, and schizoaffective depressed patients. High levels of thought disorder occurred in all psychotic groups except in the schizoaffective depressed group. Manic patients produced thought disorders that revealed both prominent combinatory thinking and intrusions of irrelevant ideas into the stream of discourse, usually with flippancy and humor. The thinking of schizophrenic patients was confused and fluid, and usually peppered with many idiosyncratic and peculiar words and phrases. Although schizoaffective manic patients resembled manics in their tendency to show combinatory thinking, their productions lacked the jocularity of the manics. The schizoaffective manic patients more strongly resembled the schizophrenic patients in their production of idiosyncratic verbalizations and in confused thinking. Schizoaffective depressed patients showed a strikingly constricted output, but produced a few noteworthy absurd responses, and in that respect resembled the schizophrenic patients. The authors suggest that quality of thought disorder differs in schizophrenia and in mania, and that the thought disorder of schizoaffective conditions resembles that of schizophrenia.

For over a decade, our laboratory has been conducting studies of thought disorder in psychotic conditions. Our early work concentrated on developing a reliable measure of thought disorder that encompassed the various kinds of thought disorder encountered by clinicians. In the course of this work, we evolved a metric for identifying and scoring types of thinking disorders, the Thought Disorder Index (Johnston and Holzman 1979); a copy of the revised manual for its use appears in this issue of the *Schizophrenia Bulletin*. Subsequent work demonstrated that the TDI is sensitive to changes in clinical state and can therefore be used as a measure of the effectiveness of treatment modality (Hurt, Holzman, and Dans 1973). Recent work has extended the application of the TDI to the exploration of whether there are identifiable types of thought disorder that accompany schizophrenia, mania, and schizoaffective disorder (Shenton, Solovay, and Holzman, in press; Solovay, Shenton, and Holzman, in press). This article summarizes those studies and suggests directions for further probes into the nature of thought disorder.

Language, Thought, or Speech Disorder

We have chosen to label peculiarities in psychotic communication as *thought disorders* rather than speech or language disorders. We are aware of the controversy about this issue (e.g., Brown 1973; Chaika 1974; Fromkin 1975), and therefore we state at the outset our arguments for considering what seem to be linguistic disorders as essentially thinking disorders. For several reasons we are persuaded that it is quite unprofitable to treat the verbal productions of psychotic patients as language disorders.

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As a first theoretical argument, we regard language as a medium through which thought is communicated. This view is influenced by Heider's (1959) analysis of the perception of things, and the media through which they are conveyed. Sizes, shapes, textures, constancies, and colors are examples of *media* through which we perceive *things*. When we look at a book on a desk, we do not see the book's retinal size, its changes in shape as we approach it, or its changes in color as incandescent light rather than daylight bathes it. We see the *book*, which is either the one we were looking at, the one we had never seen before, or the one we put there yesterday. The perception of that book is mediated by its properties of size, shape, slant, and color. At any time, of course, we can voluntarily direct our attention to the retinal size, color, or slant, and in that process we transform those mediators into *things* or objects of perception.

The process is the same with language. From the early moments of our life, we are trained to look through the language to the thoughts that the language is conveying. Language is thus transparent, although it can be an object of examination, as when a linguist studies language usage, grammar, and syntax. As it is with the perception of objects, when we listen to a psychotic or a normal person speak, we grasp the thoughts of the person, as conveyed by the language.

The second argument that bolsters our position bases itself on the fact that psychotic patients do not speak a shared language or even a shared dialect. There is no such thing as a schizophrenic or a manic language. Schizophrenic persons have as much difficulty as nonschizophrenic persons in understanding the speech of a schizophrenic person (Hunt and Walker 1966).

The observation that language and thought disorders are not only conceptually but empirically separable constitutes a third argument for our position. Thought disorders exist without the presence of language disorders. The reverse is also true; language disorders exist without the presence of thought disorders. Cases of pure paranoia exemplify instances of obvious thinking disorders with no trace of any language disorder. The delusional elements verbalized by such patients occur in a setting of language that is syntactically and grammatically unexceptional. As psychotic people become more disorganized, however, language deviations become conspicuous along with the thought disorders. There is, thus, an area of overlap in which the thought disorder occurs in a setting of deviant language.

There are, of course, conditions that manifest serious language disturbances without any thought disorders, as in the case of the congenitally deaf. These people are generally able to read written English at no better than a fifth grade level, and their spoken speech is burdened with syntactic and grammatical errors. Yet, when they communicate in sign language, their thinking is as well organized as is that of persons with no impaired hearing. In addition, there are several examples of thinking disorders that occur in behavior without the use of language, and that are tapped by the use of nonverbal tests such as block designs, digital symbol substitution, and maze tracing.

In personal communications as well as in some of his published work, our colleague, Roger W. Brown (e.g., Brown 1973), has further clarified for us the separability and the overlap of language and thought. Professor Brown has called our attention to linguistic factors that are independent of thought; these factors include phonotactics, morphology, and syntactics. Phonotactics refers to the sound clusters that are more or less acceptable within a particular language, such as the "ph" sound in Greek, the "ø" sound in Norwegian, the click in some African languages, and the gutteral "ch" in Hebrew. In English there are such tolerable combinations of sounds as "pr," "dr," and "cr," but not "sr." It is noteworthy that as the exemplar group of psychotic patients, schizophrenic patients do not violate these phonotactic rules.

The second factor, morphology, refers to the rules for linguistic inflection, such as "walk, walking, walked" or "pen, pens," or "trauma, traumatize," in English. The third factor, syntactics, refers to the rules for meaningful word order in a particular language. No studies have shown that psychotic patients violate the tolerance limits of their own language in either morphology or syntax.

As for speech, there are central nervous system lesions that create speech impairments, as in Broca's aphasia. These, too, are separable from thought impairments, and the appearance of expressive or receptive aphasias is also quite different from the disorders of schizophrenic communication.

It seems to us, therefore, that until there is convincing demonstration of a purely linguistic disorder in psychosis, it is more fruitful to regard the verbal productions of psychotic patients as the outcome of disordered thought processes. We therefore refer to the productions of psychotic patients as reflecting thought disorders.

Measurement of Thought Disorder

During the second half of the 19th century, when schizophrenic conditions were first being systematically described, clinicians emphasized the derangements of thought that accompanied those conditions. By the time Kraepelin's (1896/1919) unifying diagnostic theory of dementia praecox was published, there had already been general acceptance that thought disturbances were present in those psychoses. Kraepelin referred to them as *derailments in speech*. Bleuler (1911/1950) referred to them as *disorders of association*. Bleuler, in particular, noted the variety of thought disorders that occur in the schizophrenias, and he pointedly distinguished those disorders from the associative flights and looseness that occur in mania. Since that time, there have been many attempts to specify the nature of thought disorder that occurs in schizophrenic conditions, but these efforts have not been particularly successful.

One reason for their failure lies in their implicit faith that a single kind of thought disorder characterizes schizophrenic thinking. Thus, concreteness, paralogical thinking, regressive or primary process thinking, disturbed reality testing, and overinclusive thinking are some of the rubrics that investigators have used to classify schizophrenic thinking. None of these categories can be selected as the decisive one for characterizing schizophrenic thinking. Therefore, they fail in their claim to be a generalized description of schizophrenic thinking. Although all of these rubrics can claim some empirical justification, they are all correct only for some schizophrenic patients, but not correct for all schizophrenic patients or even for some schizophrenic patients all of the time. Schizophrenic thought disorder takes many forms, and it appears that scales that emphasize only one of those forms cannot be universally accurate in the measurement of the range of schizophrenic thinking. Further, clinical experience has shown that there is a range of severity in thought disorder from mild to severe; individuals can be placed at different points on this range, and one can compare severity of thought disorder both within a single patient over time and between patients. A focus on only single categories of thought disorder neglects the severity dimension of thought disorder.

If one agrees that thought disorder is multifaceted and varies in severity, it becomes apparent that any measuring device must incorporate those features. The Thought Disorder Index (TDI) bases itself on those assumptions. This index is briefly described below, although the reader is referred to the monograph published by Johnston and Holzman (1979) for a more complete description and rationale.

TDI

The TDI assesses a range of thought disorder that occurs during verbal discourse. The index identifies 22 different categories of thinking disturbances at four levels of severity. Most of these categories had been described by Rapaport, Gill, and Schafer (1946/1968), and later presented as an index, called the Delta Index by Watkins and Stauffacher (1952). The Delta Index was subsequently refined by Johnston and Holzman (1979) as the TDI. The categories listed in the TDI represent most of the deviant verbalizations encountered in the course of verbal interactions with psychotic patients. We do not claim that the TDI is a complete list of thought deviations, but it does include those thought disorders of a formal nature that occur with some frequency in psychotic patients. Investigators will revise the TDI periodically as new categories are incorporated, probably on the basis of the testing of new patient groups. Three such categories—*Flippant Verbalization*, *Playful Confabulation*, and *Fragmentation*—appear in the current revision, which is published in this issue of the *Schizophrenia Bulletin*. The revised manual actually contains 23 categories, reflecting the addition of the category *Fragmentation*, which had not been considered at the time of the present studies.

Typically, verbal protocols from the Rorschach Test provide the material to which the TDI is applied. In principle, however, any unpracticed, extended verbal production is suitable, and the verbal subscales of the Wechsler Adult Intelligence Scale (WAIS) have also been used. In our experience, the WAIS is not as rich a source of deviant verbalizations as is the Rorschach Test, because the former tends to restrict the length of responses and also because many of the WAIS questions elicit more or less stereotyped and overlearned answers.

In the work to be described in this article, the Rorschach Test was administered to each subject individually and the entire session was tape recorded and then transcribed verbatim. Two teams, each consisting of two trained scorers, applied the TDI to verbatim records. Response productivity was controlled by dividing the number of thought disorder scores by the total number of Rorschach responses. The total number of words spoken was also considered as a correction for verbal productivity. The correlation between the total thought disorder and the total number of words spoken was about the same as the correlation between the total thought disorder and the total number of responses given, with manic and schizophrenic patients as the subject groups (.59 and .52, respectively). Since response count is far simpler to obtain than a word count, response count has been preferred as the correction for verbal productivity. The total TDI score always reflects the ratio of the sum of weighted scores to the number of responses, multiplied by 100.

Earlier work has shown that trained scorers show high interrater reliability for the TDI—.90 in one study (Johnston and Holzman 1979) and .89 in another (Solovay, Shenton, and Holzman, in press). The latter study reported reliabilities that range between .79 and .89 with the Spearman Brown correction for the individual categories. Scoring of the TDI, however, requires intensive training and continued practice.

The total TDI and the category scores are not normally distributed. Most normal people produce no thought disorder; therefore, the use of statistics that depend on a Gaussian distribution is problematic. Category scores must be transformed statistically if parametric statistics are to be used. One way that we have addressed this problem is to transform and standardize the category scores. Since each category had a high frequency of zero usage, it was not possible to employ any one transformation that would both include the zero category and yield a normal distribution. Therefore, we considered only the non-zero frequencies of each of these categories in attempts at normalization. The transformations included negative reciprocal, square root, logarithmic, and natural logarithmic conversions. There are populations in which the incidence of thought disorder scores in particular categories is infrequent enough to defy even these conversions. In our studies, therefore, we have used a simple dichotomy of "present" or "absent" for such categories. In the studies to be described in this article, for example, the category of neologisms was too skewed to be normalized by transformation and it has therefore been dichotomized.

Previous Work Using the TDI. Our earliest study (Johnston and Holzman 1979) reported that the TDI was highly elevated in all psychotic patients, which included chronic schizophrenic patients (hospitalized at least 5 years), recently hospitalized schizophrenic patients (hospitalized less than 1 year), and nonschizophrenic patients (including atypical psychoses and manics). Nonpsychotic hospitalized patients (personality disorders, for example) and normals (those without a history of psychiatric treatment or illness) had the lowest TDI levels. Although the normal control group had the lowest TDI score of any group, individuals within that group did show minimal to mild elevations. Sex differences, ethnicity, social class, and intellectual level did not account for the differences among the groups (Haimo and Holzman 1979).

Hurt, Holzman, and Davis (1983) used the TDI to chart changes in patients who were administered a butyrophenone (haloperidol) after a flexible washout period that ranged from 1 to 3 weeks. The TDI showed a rapid decrease in total thought disorder after the introduction of the drug. The reduction in thought disorder was dramatic after 3 days of treatment, and continued improvement until discharge was documented. The pattern of thought disorder reduction over time was congruent with the pattern of change in the subscales of the Brief Psychiatric Rating Scale (BPRS) that tap schizophrenic symptoms (thought disorder and hostility). In contrast, the BPRS subscales that reflect general psychopathology (activity, anergia, and anxiety-depression) showed a very different pattern, one that was almost random in its fluctuations.

The TDI has been used to test the thought slippage of psychotic children and children deemed at risk for psychosis by virtue of having a psychotic mother (Arboleda and Holzman 1985). To assess the effects of age on the presence of thought disorder, we first tested 79 normal children; it was against those records that we evaluated the psychotic and at risk children. Normal children showed a clear tendency for thought disorder to decline with age. The level of thought disorder in psychotic children and high-risk children was about three times higher than that of the normal children and children hospitalized for nonpsychotic psychiatric conditions. The occurrences of thought disorder that distinguished the normal from the high-risk and psychotic children were present in the protocols of only 10 percent or less of the normal children.

The aforementioned studies have used, for the most part, the total TDI scores, and no claim has been made for diagnostic specificity of those total scores because psychotic schizophrenic and manic patients did not differ significantly from each other. Nevertheless, there are indications, both from clinicians and from the Johnston and Holzman (1979) study, that qualitative distinctions exist between manic and schizophrenic patients. The remainder of this article reports two studies (Shenton, Solovay, and Holzman, in press;

Solovay, Shenton, and Holzman, in press) that explored those differences, and comments on their implications for diagnostic work, for the theory of psychotic disorganization, and for the status of schizoaffective disorder.

Method

Subjects. The subjects were drawn from a pool of 125 patients at two psychiatric hospitals, one a private active treatment center and one a State hospital for chronically ill patients. All patients were interviewed by one of four diagnosticians who used a standardized inventory that combined questions from the Present State Examination (PSE) (Wing et al. 1967) and the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L) (Spitzer and Endicott 1978). The information from those interviews was supplemented by systematic chart reviews to permit diagnostic classification of the patients on the basis of three diagnostic schemes: *DSM-III* (American Psychiatric Association 1980), the St. Louis Criteria (Feighner et al. 1972), and the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978). To minimize ambiguous clinical diagnoses and erroneous diagnostic assignments, we only accepted subjects who met criteria for all three of the classification systems. In addition, we included all of those who met criteria for the schizoaffective disorder classification of the RDC. The resulting groups consisted of 43 schizophrenic patients (16 from the State hospital), 20 manic patients (all from the private hospital), and 22 schizoaffective patients. There were 12 schizoaffective manic and 10 schizoaffective depressed patients. We made no attempt at further subdivision of the schizoaffective patients because the sample was not large enough.

We tested 22 normal subjects who were screened for the absence of mental illness in themselves and in their first-degree relatives.

Drug Effects. Several studies have shown that antipsychotic medication reduces thought disorder (e.g., Chapman and Knowles 1964; Orzack, Kornetsky, and Freeman 1967; Spohn et al. 1977; Hurt, Holzman, and Davis 1983). We were unable to use drug-free patients in this study for reasons discussed elsewhere (see Shenton, Solovay, and Holzman, in press; Solovay, Shenton, and Holzman, in press). Briefly, there is no satisfactory way to test a drug-free population of actively psychotic patients and have them remain representative of the main body of such patients; statistical controls are also unsatisfactory (but see Spohn et al., 1977, for studies of the medication issue). Our solution, clearly a compromise, was to test subjects as close to the beginning of their hospital stay as possible; by that timing, we hoped to minimize the effects on thought disorder of the ameliorative and masking influences of drug treatment.

All but two schizophrenic patients were receiving neuroleptic drugs and all but three manic patients were receiving either lithium salts alone or in combination with a neuroleptic. Two manic patients were receiving only neuroleptics and one was receiving only an anxiolytic agent. Only one schizoaffective patient was unmedicated; eight were receiving neuroleptics alone, and 12 were receiving lithium in combination with either a neuroleptic or a nonneuroleptic medication.

Procedure. After informed consent had been obtained, all subjects were administered the Rorschach Test as described by Rapaport, Gill, and Schafer (1946/1968) and in the TDI manual. All peculiar, deviant, idiosyncratic, strange, irregular, and unconventional verbalizations were inquired into, in the manner described in the TDI manual (published in this issue) and in the Johnston and Holzman (1979) monograph. All testing sessions were audio-recorded and labeled only with an identification number. These tapes were transcribed by an experienced transcriber, and typed protocols were then scored for thought disorder by the trained judges, without knowledge of diagnosis.

Full accounts of descriptive variables such as IQ, clinical condition, and socioeconomic status are contained in the original reports (Shenton, Solovay, and Holzman, in press; Solovay, Shenton, and Holzman, in press). Table 1 contains a summary of those variables.

Results

Total TDI. As Johnston and Holzman (1979) had reported, high levels of thought disorder occurred in all psychotic groups, compared with the level obtained in the normal control group.

The levels of thought disorder produced by the schizoaffective sample are of interest. As a group, they produced approximately the same quantity of thought-disordered responses as did the manic patients, but when the schizoaffective depressed and schizoaffective manic patients were considered separately, the former subdivision showed a low level of thought disorder, whereas the latter subgroup showed a level of thought disorder that was almost

Table 1. Demographic and clinical characteristics of the subject groups

	Manics (n = 20)	Schizo-phrenics (n = 43)	Schizo-affectives (n = 22)	Normals (n = 22)
Age (years)	24.1 (SD 8.0)	25.0 (SD 5.35)	23.5 (SD 4.7)	20.5 (SD 3.2)
Education (years)	14.0 (SD 2.7)	12.7 (SD 2.2)	13.7 (SD 1.62)	13.9 (SD 1.8)
Mean WAIS Verbal IQ	110.9 (SD 14.9)	103.3 (SD 15.3)	106.7 (SD 15.8)	111.2 (SD 13.2)
Sex (% male)	66.7	81.6	53.4	36.4
Social class:[1]				
Class 1-111	16 (80%)	32 (74.4%)	20 (91%)	9 (45.5%)
Class IV-V	4 (20%)	11 (25.6%)	2 (9%)	13 (54.5%)
Number of months in illness state	4.2 (SD 4.1)	28.7 (SD 35.6)	14.4 (SD 15.4)	
Global Assessment Scale	43.0 (SD 16.8)	31.8 (SD 10.0)	34.9 (SD 9.8)	
Medication				
No drugs	0 (0%)	2 (4.7%)	1 (4.5%)	
Neuroleptics only	2 (10%)	28 (65.1%)	8 (36%)	
Neuroleptics with antidepressants	0 (0%)	6 (13.9%)	0 (0%)	
Lithium	6 (30%)	0 (0%)	1 (4.5%)	
Lithium with neuroleptics	7 (35%)	7 (16.3%)	9 (41%)	
Lithium with other medications	4 (20%)	0 (0%)	3 (13.6%)	
Anxiolytics	1 (5%)	0 (0%)	0 (0%)	

[1] Hollingshead & Redlich (1958) 2-factor index of social class.

equivalent to that of the schizophrenic patients. When a priori contrasts for simple effects were conducted, however, the only group differences that were significant were those between the normals and all other groups, and between the schizoaffective depressed and schizophrenic patients from both hospitals (see table 2).

Qualitative Distinctions. The abovementioned studies focused mainly on the quantitative differences in thought disorder among the groups. It had already been sufficiently documented that all psychotic groups produced disordered thinking, and the quantitative levels of thought disorder in the present studies simply added confirmation. The remainder of this report examines whether these groups of psychotic patients produce different profiles of thought disorder—that is, whether they differ in the kinds of thought disorder they display. To explore that question, we applied the statistical techniques of factor analysis and discriminant function analysis.

Factor-Analytic Procedures. We investigated the yield and power of four differently derived sets of factors. The first was derived from a principal components analysis with varimax rotation. The second compared the subject groups with respect to factors that had been proposed on an a priori basis by Johnston and Holzman (1979). The third examined the distinctions among the groups on the basis of a

Table 2. Mean total scores on the Thought Disorder Index for all patient groups and normal control subjects

Group	n	Mean (SD)
Schizophrenic	43	34.6 (38.8)
State hospital	16	37.8 (33.0)
Private hospital	27	32.6 (42.3)
Manic	20	25.0 (16.2)
Schizoaffective	22	22.8 (21.4)
Manic	12	31.0 (25.6)
Depressed	10	13.0 (8.6)
Normal	22	5.9 (4.5)

Note.—Analysis of variance (ANOVA) for 4 principal groups: $F = 6.72$, $df = 3/103$, $p < .0003$, $\eta = .405$; ANOVA for all groupings: $F = 4.85$, df $= 5/101$, $p < .0005$; $\eta = .440$.

post hoc analysis of thought disorder category differences. The fourth was derived from the levels of severity, a 4-point scale already built into the TDI.

Principal Components Analysis With Varimax Rotation. To approximate the optimal ratio of subjects to measures, all 97 psychotic patients (28 of the original 125 patients were eliminated because they were not psychotic) were used to generate the factors. We used unit weighting rather than factor loadings to obtain individual scores that minimized the effects of sample fluctuations in this exploratory analysis. We found six interpretable factors with Eigen values above 1 (see table 3): *Combinatory Thinking, Idiosyncratic Verbalizations, Autistic Thinking, Fluid Thinking, Absurdity,* and *Confusion.* The subject groups were then compared by analysis of variance on the extent to which they made use of these six general categories.

Table 3. Thought disorder factors derived from a principal components factor analysis

Factor	Categories	Loading
1. Idiosyncratic Verbalization	9. Peculiar responses	.83
	11. Queer responses	.69
Eigen value = 4.06		
2. Combinatory Thinking	15. Playful confabulation	.83
	8. Incongruous combinations	.60
	2. Flippant	.58
	14. Fabulized combinations	.53
Eigen value = 2.11		
3. Autistic Thinking	20. Autistic logic	.79
	22. Incoherence	.72
Eigen value = 2.04		
4. Fluid Thinking	17. Fluidity	.71
	21. Contamination	.69
Eigen value = 1.53		
5. Absurdity	22. Neologisms	.86
	17. Absurd	.49
Eigen value = 1.24		
6. Confusion	3. Vagueness	.76
	12. Confusion	.73
Eigen value = 1.18		

The *Combinatory Thinking* factor showed statistically significant group differences and emerged as a clear manic factor ($F = 10.53$, $df = 3/103$, $p < .001$). An a priori contrast of simple effects shows that the manic patients are higher on this factor than all other groups, including the schizoaffective manic patients. The schizoaffective manic patients, however, produced more of these responses than did the schizophrenic patients ($F = 7.68$, $df = 5/101$, $p < .0001$). The schizoaffective depressed patients produced no more of these responses than did the normals. *Combinatory Thinking* embraces categories that indicate tendencies to merge percepts and ideas incongruously. At times the result may be humorous and flippant as in manic patients; but schizoaffective manic patients lacked that playful quality, and their combinatory thinking had a different quality from that produced by the manic patients.

The *Idiosyncratic Verbalizations* factor reflects peculiar language constructions and usage that can be awkward, stilted, and strange. There were significant group differences on this factor ($F = 3.18$, $df = 3/103$, $p < .027$). A priori contrast of simple effects shows that the schizophrenic and schizoaffective manic patients produce more of these responses than do the other psychotic patients and the normals.

The schizophrenic and the schizoaffective manic patients had higher scores on the *Autistic Thinking* factor than did all the other groups, but these trends did not reach conventional levels of statistical significance. *Autistic Thinking* includes TDI categories that tap arbitrary and disorganized thinking.

Marginally statistically significant group differences emerge on the *Fluidity* factor ($F = 2.19$, $df = 3/103$, $p < .094$). This factor reflects a tendency to blur boundaries between categories, ideas, or objects in such a way as to disregard the distinctive features of the objects or ideas. Schizophrenic patients gave the highest number of these responses.

The *Absurdity* factor reflects blatant disorganization of thought. It includes neologisms and absurd responses. Schizophrenic patients are, to a statistically significant degree, the high producers of responses on this factor, and it is even more characteristic of the State hospital schizophrenic patients than of the private hospital schizophrenic

Table 4. Thought disorder factors derived from a priori classification

Factor	Categories
1. Associative Looseness	1. Inappropriate distance 2. Flippant 6. Clangs 7. Perseveration 9. Relationship verbalization 13. Looseness 17. Fluidity
2. Combinatory Thinking	8. Incongruous combination 10. Idiosyncratic symbolism 14. Fabulized combination 19. Confabulation 15. Playful confabulations 20. Autistic logic 21. Contamination
3. Disorganization	3. Vagueness 5. Word-finding difficulty 12. Confusion 22. Incoherence
4. Idiosyncratic Verbalization	4. Peculiar 11. Queer 18. Absurd responses 23. Neologism

patients. There is a trend, which falls just beyond conventional levels of statistical significance, for both the schizoaffective depressed and the schizoaffective manic patients to give elevated numbers of responses in the *Absurdity* factor.

On the *Confusion* factor, schizophrenic and schizoaffective patients gave more responses than did manic patients or normals, but the trends did not reach statistical significance. Vagueness and blatant confusion are included in this factor.

It is noteworthy, then, that the schizophrenic and the manic patients are clearly different from each other on these six empirically derived factors. The schizoaffective manic patients were more like the schizophrenic patients on the five schizophrenic factors, but they were also like the manics on the one manic factor: *Combinatory Thinking*. The schizoaffective depressed patients were low producers of thought disorder on all factors except for *Absurdity*, where they resembled the schizophrenics.

A Priori Factors. The four a priori factors (table 4) show a similar pattern of differences among the diagnostic groups.

Associative Looseness, which encompasses tendencies to be led by internal sets rather than by task requirements, showed no differences among the psychotic groups, with the exception of the schizoaffective depressed patients, who gave the lowest number of these responses of any psychotic group. We regard this factor as a "general psychotic thinking factor," which has no specific distinguishing power among groups of psychotic patients.

Combinatory Thinking resembles the *Combinatory Thinking* factor of the empirical factors. Here, statistically significant group differences were found ($F = 5.29$, $df = 5/101$, $p < .001$). The manic and schizoaffective manic patients produced the largest number of responses, and the schizoaffective depressed patients and the normals produced the fewest.

Disorganized Responses represents thinking that is unconnected logically, confused, and quite difficult to understand. Group differences of borderline significance were obtained ($F = 1.89$, $df = 5/101$, $p < .103$). Schizophrenic patients and schizoaffective depressed patients, respectively, gave the highest and lowest number of these responses of the psychotic patients. The schizoaffective manic patients resembled the schizophrenic patients.

The category of *Unconventional Verbalizations* resembles the *Idiosyncratic Verbalizations* of the empirical factors. This factor reflects disruptions of word usage that are not under the speaker's voluntary control. Statistically significant group differences emerged on this factor ($F = 3.93$, $df = 5/101$, $p < .003$). Both schizophrenic and schizoaffective manic patients produced the highest number of responses.

In summary, on the a priori factors, *Associative Looseness* emerged as a nonspecific thought disorder factor that was characteristic of all psychotic patients, while *Combinatory Responses* characterized manic and schizoaffective manic patients. On the *Disorganized Responses* and *Unconventional Verbalizations*, the schizophrenic and schizoaffective manic patients produced the highest number of responses.

Post Hoc Factors. Inasmuch as this study is an exploratory one and does not claim to confirm previous results, we constructed a third set of factors from the TDI categories that best discriminated the schizophrenic and manic patients, as shown in the empirical and a priori analyses. We were able to construct five factors that embraced 14 of the 22 TDI categories (table 5). *Combinatory Thinking* and *Irrelevant Intrusions* emerge as manic factors. The three other factors, *Fluid Thinking*, *Confusion*, and *Idiosyncratic Verbalizations*, can be called schizophrenic factors. These post hoc factors did indeed yield significant group differences. It is particularly noteworthy that the schizoaffective manic patients resembled the manic patients on the *Irrelevant Intrusions* factor but not on the *Combinatory Thinking* factor; they resembled the schizophrenic patients on the *Confusion* and *Idiosyncratic Verbalization* factors. The schizoaffective depressed patients, in contrast, were low producers on all factors.

Table 5. Thought disorder factors derived from post hoc analysis

Factor	Categories
1. Irrelevant Intrusions	2. Flippant 13. Looseness
2. Combinatory Thinking	8. Incongruous combinations 14. Fabulized combination 15. Playful confabulation
3. Fluid Thinking	9. Relationship verbalization 17. Fluidity 21. Contamination
4. Confusion	5. Word finding difficulty 12. Confusion 18. Absurd responses 22. Incoherence 23. Neologism
5. Idiosyncratic Verbalization	4. Peculiar verbalization

Discriminant Function Analysis. We conducted a discriminant function analysis to test the effectiveness of the thought disorder factors in separating the patients into their constituent diagnostic groups. The test was accomplished by obtaining classification functions that sort subjects into manic, schizophrenic, or schizoaffective diagnostic classes on the basis of their scores on the thought disorder factors. To avoid overestimating the power that would result from applying classification functions on a sample of subjects that had been derived from that same sample, we randomly divided the sample and derived the functions from one half and applied them to the other half. Cutting the sample in half, of course, diminished the precision of the estimate, and therefore we repeated the technique several times, and took the average of all the estimates from each division as the final unbiased estimate. We report herein the discriminant function analyses only for the post hoc factors. The analyses for the a priori and empirical factors are reported in Solovay, Shenton, and Holzman (in press), and in Shenton, Solovay, and Holzman (in press).

The discriminant function analysis yielded one function that statistically significantly differentiated the groups ($\chi^2 = 45.31$, $df = 15$, $p < .0001$; canonical correlation $= -.603$). In this analysis, *Combinatory Thinking* and *Irrelevant Intrusions* had positive weightings (.625 and .616, respectively), and *Confusion*, *Idiosyncratic Verbalizations*, and *Fluid Thinking* had negative weightings ($-.55$, $-.26$, and $-.40$, respectively). Manic patients gave many responses in the *Combinatory Responses* and *Irrelevant Intrusions* factors, and gave very few responses in the other three factors. On the other hand, schizophrenic patients gave significant numbers of responses in the *Confusion*, *Fluid*, and *Idiosyncratic Verbalization* factors, but few responses in the *Combinatory Thinking* and *Irrelevant Intrusions* factors. Schizoaffective manic patients resembled the manic patients in the number of responses in the *Irrelevant Intrusions* factor, but they also resembled the schizophrenic patients in the number of responses given in the *Idiosyncratic Verbalizations* and the *Confusion* factors. It is striking that the schizoaffective manic patients did not resemble the manic patients on the *Combinatory*

Thinking factor, and their verbalizations were devoid of the humor and playfulness so characteristic of the manic patients. The schizoaffective depressed patients gave only a small number of thought-disordered responses. Their general productivity was constricted, although a few striking responses in the *Absurd* factor (of the empirical factor structure) did emerge.

When a three group classification function analysis (schizophrenic, manic, and schizoaffective) was performed, 57.7 percent of the patients were correctively classified—well above the chance expectation of 33.3 percent. It is of interest that the principal misclassifications are in the schizoaffective category. More schizoaffective patients are misclassified as schizophrenic (31.8 percent) than as manic (22.7 percent).

Levels of Severity. The TDI arrays the 22 thought disorder categories along a 4-point scale of severity, with the weights of .25, .50, .75, and 1.0 assigned to those points. An analysis showed that there were statistically significant group differences at all four levels of severity. The normals, of course, produced significantly fewer responses at all four levels. All psychotic groups, except the schizoaffective depressed group, produced roughly similar quantities of thought disorder at the .25 and .50 levels. At the 1.0 level, however, only the schizophrenic and the schizoaffective manic patients gave significant numbers of responses.

Discussion

We have presented evidence that manic and schizophrenic patients can be distinguished by their distinctive types of thought disorder. This distinction is obtained independently of the behavioral symptoms that the patients present and independently of the content of their delusions, or other disorganized communications. Therefore, on the dimension of formal thought disorder, distinctions do assert themselves through the medium of the TDI. Manic thought disorder manifests itself as loosely tied together ideas that are excessively and immoderately combined and elaborated. Often, there is a playful, mirthful, and breezy quality to their productions. Intrusions of incongruous ideas into social discourse is one consequence of this propensity.

Schizophrenic thought disorder shows very little, if any, of the exuberant, jocular, frivolous elaborations of the manic patients. Fluid thinking, interpenetrations of one idea by another, unstable verbal referents, and overly concise and contracted communications give the impression of inner turmoil and confusion. The listener, too, is often confused by these communications from the schizophrenic patients.

There is, moreover, a set of thinking disorders that appears to be nonspecific, to occur in all of the groups in this study. This set includes vagueness, loss of set, and inappropriate distance. These findings clarify the position taken in a number of publications, that thought disorder is not specific to schizophrenia (e.g., Harrow and Quinlan 1977; Andreasen 1979; Johnston and Holzman 1979). Thought disorder does indeed occur in all of the psychoses investigated in this report. But thought disorder is not unitary, and only certain qualities of thought disorder accompany certain psychoses. Even from this preliminary study, it is possible to conclude that it is no longer sufficient to state in a case report that "thought disorder is present." One should try to specify the type of thought disorder that is present.

With respect to the schizoaffective patients, our data show a similarity between the thought disorder of schizoaffective and schizophrenic patients. Although there were some similarities between the thought disorder of manic and schizoaffective patients (for example, combinatory thinking), there were striking qualitative differences between these two groups. These comparisons connote that schizoaffective disorder is more like schizophrenia than like manic psychosis. Our data also demonstrate that within the schizoaffective category, there is sufficient evidence of heterogeneity to warrant the retention of the schizoaffective depressed and schizoaffective manic subclassifications. Because of the small numbers of subjects within the schizoaffective group, we did not further scrutinize the classes schizoaffective, mainly schizophrenic, and schizoaffective, mainly affective, which are considered by the RDC to be subclasses of both schizoaffective mania and schizoaffective depressed. Within the two categories of schizoaffective disorder in the present study, there were only superficial resemblances to the thought disorder of manic patients. Although schizoaffective patients gave a number of *Combinatory Responses* that quantitatively lie midway between those of the manic and schizophrenic patients, those responses were devoid of the flippancy and humor of the manic patients. The resemblance between the schizoaffective manic and schizophrenic patients were quite strong on the factors that include *Idiosyncratic Verbalization* and *Confusion*.

On the basis of these data, we argued that schizoaffective disorder is not a variant of affective disorder but of schizophrenia (Shenton, Solovay, and Holzman, in press). In this respect, our evidence supports the position of Bleuler (1924). In discussing the many demarcations among the psychoses, Bleuler asserted that those divisions, including the so-called atypical psychoses, are illusory. The boundaries, he asserted, reflect changing clinical states but not different diseases. Manic and depressive symptoms surely appear in schizophrenic conditions, but these symptoms reflect a host of incidental or extraneous events. He considered the primary symptoms to be singularly diagnostic. Although he left no precise measurement guidelines for them, he was confident that thought disorders, which he called disorders of association, formed the principal primary symptom of schizophrenia. In this regard, his descriptions of schizophrenic thought disorder are remarkably like those we have turned up in this study: ideas that have "lost their continuity," "thinking [that] becomes illogical and often bizarre . . . two ideas, fortuitously encountered, are combined into one thought . . . two or more ideas are condensed into a single one . . . a high degree of associational disturbance usually results in states of confusion" (Bleuler 1911/1950, p. 14).

The qualities of thought disorder in the schizoaffective manic patients indicate that although there are some similarities to the irrelevant intrusions of the manic patients, those intrusions do not have the buoyancy, energy, and mirthfulness characteristic of mania. Nor do they have the clarity and understandable formulations of the manic productions. In their tendency to confusion and disorganization, they are very much like the schizophrenic patients.

As for the schizoaffective depressed patients, there are noteworthy tendencies to produce absurd responses. But since those absurd responses occur in a setting of general constriction, they have a greater weight than if they occurred in dilated records. The occurrence of those absurd responses also distinguishes the thinking of schizoaffective depressed patients from the thinking produced by constricted normals and by patients with a primary depression.

The major diagnostic classifications, such as the ICD-9 and the *DSM-III*, are based principally on symptom assessments. Inasmuch as psychiatry lacks tests for basic processes of psychopathology, analogous to serum enzymes for the diagnosis of a myocardial infarction, symptom assessment has had to suffice. It appears, however, that the qualitative assessment of thought disorder does show syndrome specificity, and if the data reported herein are replicated, they could enter into more refined, process-oriented diagnosis.

The kinds of thought disorder that characterize the manic and the schizophrenic patients suggest that different cognitive processes underlie them. A program of investigation into their underlying dynamics—psychological, neuropsychological, and neuropathological—should be undertaken. We have earlier argued in this article that language and speech are not impaired in the major psychoses, and that the capacity to use and to understand language are intact. Further, Cohen and Camhi (1967) have shown that the associative hierarchies of schizophrenic cognitive organization are also unimpaired. The TDI collects instances of impaired language performance rather than of language competence (cf. Chomsky 1969). Such impairments reflect the influence of cognitive factors that are different from those involved in some of the expressive and receptive aphasias. In the manias and schizophrenias, it is the integration of thought and language that displays impairment. This conclusion would point to pathophysiological investigations that perhaps do not include the speech and language centers of the brain. Further, it would seem that different central nervous system processes are involved in the unrestrained combinatory activity of the manics and in the unpredictable fluidity and confusion of the schizophrenics. This is the junction where neuropsychological, psychological, and neuropathological methods must link with the psychopathological. The linkage would study the sensory, conceptual, imaginal, ideational, linguistic, and motor aspects of the thought disorders. We believe that such linkages will encounter issues of the inhibitory and disinhibitory control processes that are involved in the regulation of speech, language, and thought, and which other studies have implicated in the pathophysiology of psychoses (e.g., Holzman and Levy 1977; Levin 1984*a*, 1984*b*).

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Andreasen, N.C. The clinical assessment of thought: Language and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1330, 1979.

Arboleda, C., and Holzman, P.S. Thought disorder in children at risk for psychosis. *Archives of General Psychiatry,* 42:1004–1013, 1985.

Bleuler, E. *Textbook of Psychiatry.* Translated by A.A. Brill. New York: MacMillan Company, 1924.

Bleuler, E. *Dementia Praecox or The Group of Schizophrenias.* (1911) Translated by J. Zinkin. New York: International Universities Press, 1950.

Brown, R.W. Schizophrenia, language, and reality. *American Psychologist,* 28:395–403, 1973.

Chaika, E. A linguist looks at "schizophrenic" language. *Brain and Language,* 1:257–276, 1974.

Chapman, L.J., and Knowles, R.R. The effects of phenothiazines on disordered thought in schizophrenia. *Journal of Consulting Psychology,* 28:165–169, 1964.

Chomsky, N. *The Acquisition of Syntax in Children From 5 to 10.* Cambridge, MA: MIT Press, 1969.

Cohen, B.D., and Camhi, J. Schizophrenic performance in word-communication task. *Journal of Abnormal Psychology,* 71:240–246, 1967.

Feighner, J.P.; Robins, E.; Guze, S.; Woodruff, R.A.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry,* 86:57–63, 1972.

Fromkin, V.A. A linguist looks at "A Linguist Looks at 'Schizophrenic' Language." *Brain and Language,* 2:498–503, 1975.

Haimo, S., and Holzman, P.S. Thought disorder in schizophrenics and normal controls: Social class and race differences. *Journal of Consulting and Clinical Psychology,* 47:963–967, 1979.

Harrow, M., and Quinlan, D. Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry,* 34:15–21, 1977.

Heider, F. On perception, event structure, and the psychological environment. In: *Psychological Issues.* No. 3. New York: International Universities Press, 1959.

Holzman, P.S., and Levy, D.L. Smooth-pursuit eye movements and functional psychoses: A review. *Schizophrenia Bulletin,* 3:15–27, 1977.

Hollingshead, A.B., and Redlich, F.C. *Social Class and Mental Illness.* New York: John Wiley & Sons, Inc., 1958.

Hunt, W.A., and Walker, R.E. Schizophrenics' judgments of schizophrenic test responses. *Journal of Clinical Psychology,* 22:118–120, 1966.

Hurt, S.S.; Holzman, P.S.; and Davis, J.M. Thought disorder: The measurement of its changes. *Archives of General Psychiatry,* 40:1281–1285, 1983.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking.* San Francisco: Jossey-Bass, 1979.

Kraepelin, E. *Dementia Praecox and Paraphrenia.* (1896) Translated by R.M. Barclay. Chicago: Chicago Medical Books, 1919.

Levin, S. Frontal lobe dysfunctions in schizophrenia—I. Eye movement impairments. *Journal of Psychiatric Research,* 18:27–55, 1984*a*.

Levin S. Frontal lobe dysfunctions in schizophrenia—II. Impairments of psychological and brain functions. *Journal of Psychiatric Research,* 18:57–72, 1984*b*.

Orzack, M.H.; Kornetsky, C.; and Freeman, H. The effects of daily administration of carphenazine on attention in the schizophrenic patient. *Psychopharmacologia,* 11:31–38, 1967.

Rapaport, D.; Gill, M.M.; and Schafer, R. *Diagnostic Psychological Testing* (1946). Edited by R. Holt. New York: International Universities Press, 1968.

Shenton, M.E.; Solovay, M.R.; and Holzman, P.S. Comparative studies of thought disorder: II. Schizoaffective disorder. *Archives of General Psychiatry,* in press.

Solovay, M.R.; Shenton, M.E.; and Holzman, P.S. Comparative studies of thought disorder: I. Mania and schizophrenia. *Archives of General Psychiatry,* in press.

Spitzer, R.L., and Endicott, J. *Schedule for Affective Disorders and Schizophrenia—Lifetime Version.* New York: New York State Psychiatric Institute, 1978.

Spitzer, R.L.; Endicott, J.; and Robins, E. *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders.* New York: New York State Psychiatric Institute, 1978.

Spohn, H.; Lacoursiere, R.; Thompson, K.; and Coyne, L. Phenothiazine effects on psychological and psychophysiological dysfunction in chronic schizophrenics. *Archives of General Psychiatry,* 34:633–644, 1977.

Watkins, J.G., and Stauffacher, J.C. An index of pathological thinking in the Rorschach. *Journal of Projective Techniques,* 16:276–286, 1952.

Wing, J.K.; Birley, J.L.T.; Cooper, J.E.; Graham, P.; and Isaacs, A.D. Reliability of a procedure for measuring and classifying 'Present Psychiatric State.' *British Journal of Psychiatry,* 113:499–515, 1967.

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Family Care of Schizophrenia

Family Care of Schizophrenia, authored by Ian R.H. Falloon, Jeffrey L. Boyd, and Christine W. McGill, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). In an ongoing search for the cause (causes) of schizophrenia, the family has often been identified as a prime candidate. Focus on the harmful effects of critical or rejecting family members and deviant communication patterns has obscured the potentially beneficial role of many families in providing support for their schizophrenic members. Though available evidence suggests that intolerance and emotional overinvolvement do heighten the risk of relapse, the authors' thorough review of the literature reveals that only half of the families studied exhibit such attitudes.

Family Care of Schizophrenia focuses on a model developed by the authors for the broad-based community treatment of schizophrenia and other severe forms of mental illness that taps this underutilized potential. Based on the hypothesis that environmental stress is a major factor in the onset and severity of schizophrenic episodes, the model incorporates well-established behavioral techniques to enhance the coping mechanisms and problem-solving abilities of the family. The goal of the program is not merely the reduction of stress that can trigger florid episodes, but also the restoration of the patient to a level of effective social functioning that permits employment and socialization with persons outside the family. Following a thoughtful and highly readable discussion of the rationale behind their approach, the authors present a detailed description of their behavioral family model, buttressed by illustrative transcripts from actual therapy sessions. Central to their strategy is the development of problem-solving skills and social supports for the patient and his or her family—including education about the illness and the effects of neuroleptics—that will enable them to cope not just with potentially threatening behavioral disturbances and other traumatic life events, but also with the stressors of daily life.

As the authors persuasively demonstrate, families can, with proper guidance, be taught to modulate the level of intrafamilial stress, regardless of whether it derives from family tensions or external life events. Their careful exposition of the family care model, coupled with case studies and results from a controlled outcome study, reveal the family to be an important resource in the community management of mental illness. A major contribution to the treatment of schizophrenia, this unique synthesis of systems theory and behavioral techniques will be of interest to psychiatrists, who are familiar with the limitations of present treatment strategies, as well as to family and behavior therapists concerned with the problems posed by major mental illness.

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Schizophrenic Thought Disorder at Followup: Its Persistence and Prognostic Significance

by Martin Harrow and Joanne T. Marengo

Abstract

To assess the prominence, persistence, and prognostic significance of positive thought disorder in schizophrenia, we studied a large sample of psychiatric inpatients at the acute phase and then followed them up twice—1½–2 years and 4 years after hospital discharge. Positive thought disorder was more prominent at the acute phase in schizophrenic and manic patients than in other types of psychotic and nonpsychotic disorders. At followup, positive thought disorder did not persist for all schizophrenics, but a significantly larger percentage of schizophrenic than other psychotic and nonpsychotic patients followed a course in which positive thought disorder was either persistently or episodically present. Positive thought disorder, when seen after the acute phase, was related to concurrent functioning, and predicted subsequent poorer outcome and functioning for both schizophrenic and nonschizophrenic patients. Positive thought disorder was one of several central features of the active schizophrenic disorder. Among schizophrenics, it was related to other major types of psychopathology, especially delusions, another positive symptom with cognitive-ideational elements. Severe positive thought disorder after the acute phase indicated a sustained episode, and suggested a more severe type of schizophrenia with a poorer prognosis.

Thought disorder has long been viewed as a major feature of schizophrenia. It has been studied extensively by both early (Bleuler 1911/1950; Cameron 1939; Kasanin 1944; Rapaport, Gill, and Shafer 1946; Arieti 1974) and more current theorists (Chapman and Chapman 1973; Holzman 1978; Andreasen 1979; Johnston and Holzman 1979; Harrow and Quinlan 1985; Oltmanns et al. 1985). Recent research has shown that positive thought disorder is not unique to schizophrenia (Andreasen and Powers 1974; Harrow and Quinlan 1977; Braff, Glick, and Griffin 1983) and raises questions about the relationship between positive symptoms (such as thought disorder and delusions) and outcome (Andreasen and Olsen 1982; Strauss, Carpenter, and Bartko, 1974). More specifically, some have questioned whether positive symptoms are important in diagnosis and prognosis (Pope and Lipinski 1978; Knight et al. 1979; Abrams and Taylor 1981).

This article is a report from the Chicago Followup Study, a research program designed to assess the importance of positive thought disorder in schizophrenia. We report the results of a series of studies pertaining to the frequency and prominence of thought disorder in schizophrenia at the acute phase, and to the persistence and prognostic significance of positive thought disorder over time.

Much of the previous research on the relationship between thought disorder and later outcome has focused on thought disorder during acute inpatient phases (Piotrowski and Bricklin 1961; Payne 1968; Cancro 1969; Harrow, Bromet, and Quinlan 1974; Knight et al. 1979). More recent longitudinal investigations are beginning to detect differences among schizophrenic patients in the severity and persistence of

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cognitive disorders over time (Asarnow and MacCrimmon 1978; Chapman 1979; Harrow, Silverstein, and Marengo 1983; Nuechterlein and Dawson 1984; Marengo and Harrow, in press). Our prognostic approach, that of subclassification on the basis of longitudinal characteristics, stems from the view that continuities or shifts of state may in themselves reflect relevant prognostic indicators (Strauss and Docherty 1979). A longitudinal study of schizophrenic cognition may be all the more important since the original concepts of dementia praecox, and of schizophrenia, were based on theoretical views about long-term course.

Our prospective, followup study has assessed previously hospitalized, young adult schizophrenic patients for signs of disturbed thinking 2 and 4 years after hospitalization. We hypothesize a continuum model of psychopathology in which the severity of thought disorder is expected to relate to the severity of outcome dysfunction over time. Formulations about unique subtypes of disturbance are explored by: (1) identifying three patient groups exhibiting (a) no thought disorder, (b) signs of abnormal thinking, and (c) severe thought disorder at the first followup, and (2) examining differences among these groups in symptoms and life adjustment at subsequent followup. Individuals demonstrating different *courses* of thought disturbance over the two followup assessments also are examined for similar variations in short-term outcome over the longitudinal course of disorder. Since thought disorder is found across a spectrum of psychotic and nonpsychotic patient groups during acute and postacute phases of disorder, the current research investigates the relationship between thought pathology and subsequent adjustment in schizophrenic, other psychotic, and nonpsychotic patient groups.

Specific research questions are:

- Is positive thought disorder at the acute phase prominent primarily in schizophrenic patients, or is it prominent in all types of psychotic patients?
- Does positive thought disorder persist after the acute phase in schizophrenic and other patient groups?
- Is thought disorder related to other aspects of functioning and psychopathology, and does it predict outcome in schizophrenia?

This article analyzes the first two questions by reviewing our recent data on the frequency and persistence of thought disorder over time. The article also presents new data we have collected on the prognostic significance of positive thought disorder, and on the efficacy of positive thought disorder in separating out a subgroup of schizophrenic patients with very poor adjustment and outcome.

Method

The Chicago Followup Study, from which 191 patients from Michael Reese Hospital and the Illinois State Psychiatric Institute were derived, examined the longitudinal course of positive symptoms (e.g., positive thought disorder and psychosis), negative symptoms (e.g., flat affect, and impoverished speech and thinking), prognosis and outcome over different phases of schizophrenia (Harrow, Silverstein, and Marengo 1983; Harrow, Carone, and Westermeyer 1985; Pogue-Geile and Harrow 1984, 1985; Grinker and Harrow, in press; Marengo and Harrow, in press). It also explores mechanisms that may be involved in thought pathology and psychosis (Harrow and Miller 1980; Harrow et al. 1983; Harrow and Quinlan 1985).

Patient Sample. The sample included 44 schizophrenic patients, 67 other psychotic patients, and 80 nonpsychotic patients who were assessed at the acute phase of hospitalization and then followed up twice, 2 and 4 years after hospital discharge (see table 1).

Diagnoses were made using the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978) and and *DSM-III* (American Psychiatric Association 1980). A detailed admission interview summarized in the patient's chart and the Schizophrenia State Interview (SSI), a semistructured, tape-recorded diagnostic research interview (Grinker and Holzman 1973), provided information for RDC and *DSM-III* ratings. The Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978) structured interview was also administered.

All 191 patients completed an assessment battery which evaluated thought disorder, psychosis, and adjustment at the first followup. Five patients were lost to suicide or death between the first and second followups (three RDC schizophrenic patients, one RDC schizoaffective patient, and one psychotic depressive). The thought disorder data at the second followup and examinations of the course of disordered thinking between the first and second followups are based on the remaining 186 patients.

Attrition did not appear particularly selective between the index and followup assessments, and patients who participated in the current study are representative of initial groups. Neither χ^2 nor t-test analyses showed any significant

Table 1. Research Diagnostic Criteria diagnoses of the patient sample

Schizophrenic	*n*	Psychotic nonschizophrenic	*n*	Nonpsychotic	*n*
Paranoid	21	Major depressive disorder	13	Major depressive disorder	49
Disorganized	1	Manic disorder	16	Minor depressive disorder	5
Undifferentiated	21	Schizoaffective depressed	21	Manic disorder	4
Catatonic	1	Schizoaffective manic	5	Other (e.g., panic disorder, anorexia, borderline)	22
		Other psychotic disorders (e.g., unspecified functional psychosis, drug abuse)	12		
Total	44		67		80

differences in initial diagnosis, sex, marital status, social class education, previous hospitalization, or index thought disorder between patients in the followup and those lost to followup.

Sampling Criteria. The sample comprised young psychiatric patients who were in early stages of disorder. Fifty-one percent were first admissions. The patients were homogeneous in the following respects: (1) *Age*—between 18 and 30 years old at index hospitalization; (2) *Previous Hospitalization*—either no more than two previous psychiatric hospitalizations during the entire period preceding index assessment, or no more than three hospitalizations within the 2 years preceding index assessment; (3) *Educational Levels*—at least 2 years of high school (91 percent completed high school or attended college).

This sampling procedure ensured that patients would be comparatively free from longstanding sociocultural deprivation, long-term hospitalization, and other factors that may obscure the characteristics of psychiatric disorders in the early stages. Chronicity has been limited in this sample.

Demographic Characteristics. Patients had a mean age of 23 ± 3.5 years, and 48 percent were male. Mean educational levels were 13.2 ± 2.1 years of education, and 61 percent were from social classes 1, 2, or 3 in the Hollingshead-Redlich (1958) system.

Table 2 presents data on the demographic characteristics of the three main diagnostic groups and for each of the major types of other psychotic patients. Among the three

Table 2*a*. Demographic characteristics of the 3 major patient groups

	Schizophrenic (*n* = 44)	Psychotic nonschizophrenic (*n* = 67)	Nonpsychotic (*n* = 80)
Sex (% male)	61	52	39
Mean age at index (years)	23	23.1	23.1
Race			
White (%)	74	77	90
Black (%)	26	21	10
Other (%)		2	
Head of household's social class (Hollingshead Index)			
I, II, III (%)	54	63	64
IV, V (%)	46	37	36
Mean education at index (years)	12.5	13.6	13.3

Table 2*b*. Demographic characteristics of the 4 psychotic nonschizophrenic groups

	Major depression, psychotic ($n = 13$)	Manic, psychotic ($n = 16$)	Schizoaffective ($n = 26$)	Other psychotic disorders ($n = 12$)
Sex (% male)	39	50	52	75
Mean age at index (years)	23.4	22.9	23.1	22.3
Race				
White (%)	83	73	88	67
Black (%)	17	20	12	33
Other (%)		7		
Head of household's social class (Hollingshead Index)				
I, II, III (%)	54	42	74	60
IV, V (%)	46	58	26	40
Mean education at index (years)	14	12.5	14.1	13.3

major diagnostic groups, differences in sociodemographic characteristics emerged in sex ($\chi^2 = 6.33$, $df = 2$, $p < .04$), educational achievement ($F = 3.89$, $df = 2/186$, $p < .02$), and the presence of hospitalizations before index admission ($\chi^2 = 8.24$, $df = 2$, $p < .02$) (table 2*a*). Nonpsychotic patients (primarily depressives) were more often female and had fewer hospitalizations than psychotic and schizophrenic patients. Nonpsychotic and psychotic nonschizophrenic patients also had attained a slightly higher educational level than schizophrenic patients. Demographic factors were not major contributors to between-group differences in thought disorder, and there were no significant variations among these features and thought disorder symptoms over time.

Medication Status. Fifty-five percent of the schizophrenic patients, 33 percent of the psychotic nonschizophrenic patients, and 13 percent of the nonpsychotic patients were receiving neuroleptic medication at the first followup. Fifty-nine percent of the schizophrenic patients, 36 percent of the psychotic nonschizophrenics, and 8 percent of the nonpsychotic patients were receiving neuroleptic medication at the second followup. There were no significant differences in the severity of positive thought disorder between medicated and unmedicated schizophrenic or nonschizophrenic patients at followup. It should be borne in mind that patients were treated with medication partly on the basis of their clinical condition and were not randomly assigned to receive medication.

Measures of Thought Disorder. A comprehensive measure of bizarre-idiosyncratic thinking was used to evaluate positive types of thought disorder (Fish 1962; Marengo et al. 1985). Scores for this measure are derived from the Goldstein-Scheerer (1941) Object Sorting Test, the Gorham (1956) Proverbs Test, and the Social Comprehension Subtest of the Wechsler Adult Intelligence Scale (WAIS) (Wechsler 1955). The construct of bizarre-idiosyncratic thinking encompasses the classic qualities of positive thought disorder proposed by diverse theorists. It also encompasses almost all the types of thought disorder described in the RDC and *DSM-III* manuals as symptoms that can be used as partial criteria for a diagnosis of schizophrenia, including incoherence, marked loosening of associations or derailment, illogical thinking, and other unusual or bizarre behavior.

Various types of disordered thinking were combined into an index that emphasized the severity of disordered thought. Proverb and Comprehension test responses were scored for positive thought disorder along 4-point scales that assign scores according to whether the response contains *no* bizarre or idiosyncratic features, *mild* cognitive slippage, *clear* positive thought disorder, or very *severe* positive thought disorder. The individual Object Sorting Test responses were similarly scored for positive thought disorder along a 3-point scale ranging from absent to severe thought disorder (Harrow et al. 1985). Scoring criteria and response examples of bizarre-idiosyncratic thinking have been published in previous reports (Marengo and Harrow 1985) and in detailed manuals (Harrow et al. 1985; Marengo et al. 1985). Satisfactory interrater reliabilities have been obtained for these scoring systems ($r = .75-.95$).

An example of a response with clear idiosyncratic features follows:

> Q: The grass is always greener in the other fellow's yard.
>
> A: Don't trouble trouble 'til trouble troubles you. (The question was repeated.) People are always—it's like you're always getting a raw shake and you're always trying to keep up with the Jones. My response is don't touch that question of comparison and competition. It's killing the world.

A response example with very severe bizarre-idiosyncratic features is given below:

> Q: Why are people who are born deaf usually unable to talk?
>
> A: When you swallow in your throat like a key it comes out, but not a scissors. A robin, too, it means spring.

Response scores from each test were summed for each patient. The raw summed scores were then classified into one of five qualitative categories according to cutoff points described in Marengo et al. (1985). The five qualitative levels of thought pathology were scored as: 1 = none; 2 = minimal to mild; 3 = definite signs of abnormal thinking; 4 = severe; and 5 = very severe. Patients were then assigned a composite score for bizarre-idiosyncratic thinking or positive thought disorder, on the basis of the most severe level obtained on any of the three tests. Groupings of patients at levels 3–5 provide the percentage of thought-disordered patients. The composite score encompasses a broad sample of patients' behavior and is more comprehensive than indexes that include only one or two tests.

To evaluate associations between the course of thought disorder and outcome in other areas, patients were divided into four groups: (1) those showing no thought disorder symptoms at either followup; (2) those showing thought disorder at the first but not the second followup; (3) those showing thought disorder at the second but not the first followup; and (4) those showing thought disorder at both followups. Groups 2 and 3 were conceived as having episodic courses of thought disorder. However, patients in group 3 were similar to those in group 4 in experiencing thought disorder symptoms concurrent with the functioning assessment at second followup. This patient classification scheme allows us to examine different aspects of the relationship between thought disorder and outcome, including the association of prior, concurrent, and persistent evidence of thought disorder with other aspects of functioning.

Assessment of Instrumental Adjustment at Followup. Information on instrumental adjustment during the year before followup was obtained through a structured interview on functioning (Westermeyer and Harrow 1984) and a modified SADS. The followup interviews involved detailed evaluations of: (1) social, work, and family functioning; (2) use of medication, psychotherapy, and other posthospital treatment; (3) neurotic, depressive, and psychotic symptoms; and (4) rehospitalization.

Two scales of adjustment were used. The first, that of Levenstein, Klein, and Pollack (1966) (LKP Scale), is an overall scale based on work functioning, life disruptions, self-support, symptomatology, potential suicide, relapses, and rehospitalization. It rates patients according to whether they had *very favorable outcomes* (i.e., scores of "1" or "2" based on good functioning in all areas), *equivocal outcomes* (i.e., scores of "3" to "6" based on good functioning in one or two areas with difficulties in other areas), or *poor outcome* (i.e., scores of "7" or "8" based on poor functioning in almost all areas in the year before followup. The second scale, designed by Strauss and Carpenter (1972), includes four individual indexes covering work functioning, social functioning, rehospitalization, and symptoms.

Finally, the presence or absence of psychosis at each followup was determined on the basis of patients' reports of delusions or hallucinations during the month preceding followup.

Procedure. All patients were interviewed and assessed extensively during their period of hospitalization. Patients were then followed up and interviewed 1½–2 years after discharge and then again approximately 2 years after the first followup. To assess thought disorder, the Proverbs test, the WAIS Comprehension Subtest, and the Object Sorting Test were administered in a systematically varied order. Each patient was also interviewed extensively about work, interpersonal relationships, symptoms, and rehospitalization. These interviews and tests were then scored by trained raters without knowledge of the patients' diagnoses.

Results

Prominence of Positive Thought Disorder at the Acute Phase of Schizophrenia. A number of earlier theorists regarded severe thought disorder as unique to schizophrenia. More recent evidence has suggested, however, that thought disorder also occurs in most acute manic patients, and possibly in other types of psychotic and nonpsychotic patients. This raises the issue of whether thought disorder is most characteristic of diagnostic groups such as schizophrenia and mania, or instead is ubiquitous to all psychotic patients, and possibly even to all acutely disturbed psychiatric patients.

We have recently addressed this issue (Marengo and Harrow 1985) in a study of 234 acute, hospitalized patients, including 85 RDC schizophrenic and 38 manic patients. In our analysis, the manic and schizophrenic disorders showed significantly more severe positive thought disorder than the combined sample of other psychotic patients at the acute inpatient phase (Marengo and Harrow 1985). The other psychotic patients comprised (1) schizoaffective patients, (2) RDC other functional psychotic patients, and (3) psychotic-depressive patients. Our results showed near significant trends for schizoaffective patients to manifest less positive thought disorder than schizophrenic patients ($p < .06$) and more thought disorder than psychotic-depressed patients ($p < .12$).

While fewer of the other psychotic patients showed positive thought disorder than the schizophrenic and manic patients, psychotic patients tended to show more positive thought disorder than nonpsychotic patients did. The presence of psychosis (e.g., delusions) appears to be related to the presence of severe thought disorder. However, other diagnostic features may be even more important as factors associated with the severity of thought disorder, since both schizophrenic and manic patients showed more positive thought disorder than did other types of psychotic patients at acute phases of disturbance.

The fact that schizophrenic and manic patients show more thought disorder than other types of psychotic patients is one indicator of the importance of thought disorder in schizophrenia. Its importance can also be supported by an index of the absolute number of schizophrenic patients with severe positive thought disorder or signs of abnormal thinking. Our data on the above sample of 85 RDC schizophrenic patients showed 52 percent with severe thought disorder at the acute phase, and an additional 23 percent with signs of abnormal thinking. In this research and in other research with partially overlapping samples, we have found a range of 75 to 80 percent of schizophrenic patients who show some signs of thought disorder at the acute phase (Harrow et al. 1982; Marengo and Harrow 1985). Not *all* schizophrenic patients show positive thought disorder at the acute phase, but the great majority do. Thus, our research suggests that thought disorder is a prominent factor in schizophrenic patients who are studied during an early, acute phase of illness. This prominence of positive thought disorder in schizophrenia has also been apparent in samples of acute schizophrenic patients which included both medicated and *unmedicated* schizophrenic patients (Harrow et al. 1982; Hurt, Holzman, and Davis 1983).

Thought Disorder After the Acute Phase. Table 3*a* shows the distribution of thought disorder for each of the three major patient groups at the first followup. As has been discussed in recent reports (Harrow, Silverstein, and Marengo 1983; Harrow, Marengo, and McDonald 1986; Marengo and Harrow, in press), almost half of the schizophrenic patients show signs of thought disorder at the first followup. However, a subgroup of psychotic nonschizophrenic patients also show thought disorder.

Patients showing signs of either abnormal thinking or severe thought disorder were found in each diagnostic group at the first followup. There were no differences in severity of thought disorder between the schizophrenic patients and other psychotic patients at the first followup, although both schizophrenic and other psychotic patients showed more severe thought disorder than nonpsychotic patients. Early analysis of our second followup results suggests larger differences between the schizophrenic and other psychotic patients at the second followup in the severity of thought disorder, and our analysis of the course of thought disorder suggests a more severe course for the schizophrenic than for the other psychotic patients.

Table 3*b* presents data on the distribution of the *course* of thought disorder between the first and second followups across diagnostic groups. These data, discussed in greater detail elsewhere (Marengo and Harrow, in press), suggest that schizophrenic patients more frequently experience episodic or persistent positive thought disorder than do nonschizophrenic patients early in the longitudinal course.

Overall, these data indicate that positive thought disorder can be found in a number of schizophrenic patients at followup, and that it may

Table 3*a*. Frequency of thought disorder in each patient group

Thought disorder group	Thought disorder at FU1[1]	Schizophrenic patients ($n = 45$)	Psychotic nonschizophrenic patients ($n = 66$)	Nonpsychotic patients ($n = 80$)
No thought disorder	None	11%	18%	19%
	Minimal-mild	44%	36%	49%
Abnormal thinking	Moderate	24%	21%	20%
Severe thought disorder	Severe	4%	17%	11%
	Very severe	17%	8%	1%

[1] FU1 = first followup.

persist over time in a subgroup. The thought disorder of a number of patients diminishes after the acute phase and is not observed in as large a percentage of schizophrenic or other psychotic patients at followup. About half of patients with early schizophrenia show no evidence of positive thought disorder at followup, and another 10–25 percent of schizophrenic patients only show moderate levels of thought disorder at followup. While positive thought disorder is less frequent in schizophrenia after the acute phase, it is still more common in schizophrenic than in nonpsychotic patients, and more schizophrenic patients than other psychotic and nonpsychotic patients show either persistent or episodic thought disorder courses (see table 3*b*). In this respect, it is still a factor in schizophrenia after the acute phase, although both positive thought disorder and other types of schizophrenic-like symptoms, such as delusions, show a reduction in frequency and in intensity after the most acute phase (Harrow, Carone, and Westermeyer 1985).

Is Thought Disorder Related to Other Aspects of Life Adjustment, and to Poorer Subsequent Functioning and Outcomes? Recently reported data bear on the issue of whether positive thought disorder in schizophrenia relates to other aspects of the patients' disorders, and in particular

Table 3*b*. Course of thought disorder in each patient group

Thought disorder course	Thought disorder at FU1 & FU2[1]	Schizophrenic patients ($n = 41$)[2]	Psychotic nonschizophrenic patients ($n = 65$)[2]	Nonpsychotic patients ($n = 80$)
No thought disorder	No TD[3] at either FU	24%	43%	56%
	TD at FU1 but not at FU2	13%	18%	20%
Episodic	TD at FU2 but not at FU1	24%	14%	11%
Persistent	TD at both FU1 and FU2	39%	25%	13%

[1] FU1 = first followup; FU2 = second followup.

[2] Suicides were excluded.

[3] TD = scores at moderate or severe levels of thought disorder.

to their life functioning (Harrow, Silverstein, and Marengo 1983; Harrow, Marengo, and McDonald 1986; Marengo and Harrow, in press). These data, based on followup studies, show a significant relationship between positive thought disorder in schizophrenia and overall functioning and adjustment. They also show a strong relationship between the presence of positive thought disorder and delusions.

In addition, we have focused on the relationship between the *course* of illness and positive thought disorder. In particular, we have studied whether positive thought disorder at followup in schizophrenia is associated with a new acute episode or with continued dysfunctioning. Our data have suggested that, for most schizophrenics, when positive thought disorder is found at followup, it is associated with continuing dysfunctioning and continuing psychopathology in other areas (Harrow, Marengo, and McDonald 1986).

To explore further the relationship between thought pathology and life adjustment, we analyzed thought disorder as a predictor of subsequent functioning. Table 4 presents Pearson correlations between the severity of first followup thought disorder and life adjustment at the second followup in the *total patient population*. For the total or combined patient sample, the severity of thought disorder at the first followup was significantly correlated with overall and specific areas of functioning at the second followup. Within the total followup group, patients with signs of thought disorder at the first followup showed greater dysfunctioning in all areas of life adjustment at the second followup.

When the total sample was categorized according to the extent of thought disorder at the first followup (no or only minimal thought disorder, abnormal thinking, and severe thought disorder), the three groups differed significantly in outcome at the second followup. One-way analyses of variance (ANOVAs) for the extent of thought disorder at the first followup revealed significant adjustment differences in overall outcome at the second followup, on the basis of both the LKP Scale ($F = 11.15$, $df = 2/189$, $p < .001$) and the Strauss-Carpenter Scale ($F = 12.81$, $df = 2/189$, $p < .001$). Work functioning ($F = 11.77$, $df = 2/189$, $p < .001$) and rehospitalization rates ($F = 5.90$, $df = 2/189$, $p < .01$) also showed significant thought disorder effects. Post hoc comparisons (Newman-Keuls) demonstrated that *severely thought-disordered patients* exhibited significantly poorer subsequent overall functioning and work adjustment than either non-thought-disordered patients or patients showing abnormal thinking ($p < .05$).

Table 4. Correlations between 1st followup thought disorder and 2nd followup functioning

Area of functioning at 2nd followup	Total sample ($n = 191$) Pearson r with thought disorder at 1st followup	p
Overall outcome (Levenstein, Klein, & Pollack 1966)	.35	< .001
Overall outcome[1] (Strauss & Carpenter 1972)	.38	< .001
Work functioning[1]	.35	< .001
Social functioning	.11	< .05
Rehospitalization rates[1]	.22	< .01
Delusions	.42	< .001
Hallucinations	.22	< .01

[1] Scales were adjusted so that positive correlations indicate that more severely thought-disordered patients demonstrate more severe impairment.

Only 17 percent of the patients with severe thought disorder at the first followup were fully functional at the second followup. Most notably, subsequent high rates of unemployment (82 percent), rehospitalization (59 percent), and psychosis (68 percent) characterized the severely thought-disordered patient group.

Non-thought-disordered patients were significantly less likely than thought-disordered patients to show delusions ($\chi^2 = 4.25$, $df = 2$, $p < .001$) or hallucinations ($\chi^2 = 7.28$, $df = 2$, $p < .05$) at the subsequent followup assessment (table 5). Thus, the presence of a posthospital thought disorder also was associated with psychotic symptoms at 4-year followup.

It should be noted in relation to the presence or absence of psychosis at hospital admission that although most of the initially nonpsychotic patients were not psychotic at followup, a subgroup of them did show psychosis. A number of patients from this small subgroup

Table 5. Relation between level of thought disorder at 1st followup and psychosis at 2nd followup[1]

Psychotic symptom	Patient group	Level of thought disorder (TD) at 1st followup					
		No thought disorder		Abnormal thinking		Severe thought disorder	
		% of patients with psychosis at 2nd followup					
		n	%	*n*	%	*n*	%
Delusions	Schizophrenic	20	30	10	60	9	78
	Psychotic nonschizophrenic	36	25	13	15	15	47
	Nonpsychotic	54	6	16	19	10	60
	Total group	110	16	39	28	34	59
Hallucinations	Schizophrenic	20	30	11	36	9	55
	Psychotic nonschizophrenic	36	17	13	8	15	27
	Nonpsychotic	54	9	16	6	10	30
	Total group	110	15	40	15	34	35

[1] Data on either delusions or hallucinations were missing for 1 or 2 subjects

were initially affectively disordered patients, with some showing mood-congruent psychotic symptoms at followup, and others showing mood-incongruent psychotic symptoms at followup.

Positive Thought Disorder as a Predictor of Outcome in Schizophrenia and Other Psychiatric Disorders. One major issue is whether the presence of positive thought disorder predicts subsequent psychopathology and functioning when diagnosis is controlled, or when the schizophrenic and the other patient groups are analyzed separately. Separate analysis of the relationship between thought disorder and subsequent outcome in schizophrenia becomes all the more important since thought disorder, as well as poorer posthospital adjustment and outcome, is more common in schizophrenic than in nonschizophrenic groups. Therefore, schizophrenic diagnosis may be a powerful intervening variable in the relationships found between thought disorder and life adjustment over time.

Two different types of analyses were conducted to determine whether the severity of positive thought disorder predicts subsequent functioning and outcome when the effect of diagnosis is controlled. The first analysis used partial correlations between thought disorder at the first followup and each outcome measure at the second followup, and controlled for diagnostic effects. In this analysis, patients were categorized according to whether they were schizophrenic (a score of "2"), or not schizophrenic (a score of "1"), and the influence of the diagnostic variable was partialled out or eliminated.

After partialling out the variance due to diagnosis, we found the severity of posthospital disordered thinking was significantly related to subsequent overall adjustment as assessed by both the LKP Scale ($r = .36$) and the Strauss-Carpenter Scale ($r = .36$). Thought disorder also was related significantly to subsequent work functioning ($r = .30$), rehospitalization ($r = .21$), delusions ($r = .40$), and hallucinations ($r = .19$). Separate analysis indicated that the correlations between thought disorder and each outcome area before diagnosis was controlled for (presented in table 4) did not differ significantly from the parallel correlations in which diagnosis was controlled.

The second type of analysis which controls for diagnosis involves separate correlations between positive thought disorder and subsequent adjustment for each patient group (see table 6). Significant positive relationships between thought disorder and subsequent outcome emerged across each diagnostic group in most areas. In particular, positive thought disorder is a significant predictor of subse-

Table 6. Correlations between 1st followup thought disorder and 2nd followup functioning

Area of functioning at 2nd followup	Pearson r with thought disorder at 1st followup Schizophrenics (n = 45)	Psychotic nonschizophrenics (n = 66)	Nonpsychotics (n = 80)
Overall outcome (Levenstein, Klein, & Pollack 1966)	.25	.32[3]	.34[4]
Overall outcome[1] (Strauss & Carpenter 1972)	.47[4]	.25[2]	.35[4]
Work functioning[1]	.38[3]	.33[3]	.21[2]
Social functioning[1]	.17	.14	.20[2]
Rehospitalization rates[1]	.32[2]	.10	.23[2]
Delusions	.51[4]	.28[2]	.40[4]
Hallucinations	.26	.15	.13

[1] Scales were adjusted so that positive correlations indicate that more severely thought-disordered subjects demonstrate more severe impairment.

[2] $p < .05$.

[3] $p < .01$.

[4] $p < .001$.

quent overall outcome, work functioning, rehospitalization, and delusions across the three types of patient samples.

Despite a significant positive relationship between thought disorder and subsequent hallucinations for the total patient sample (see table 4), thought disorder did not predict subsequent hallucinations at significant levels, and only predicted subsequent social relationships for one of the three patient groups.

The relationship between severe positive thought disorder at the first followup and delusions at the second followup is of special interest, since both have been categorized as positive symptoms. The correlations presented in table 6 between thought disorder and subsequent delusions were relatively high for the schizophrenic (r = .51) and for the nonpsychotic patients (r = .40). Analysis of the detailed data in table 6 shows that severely thought-disordered patients from each patient group experienced significantly more subsequent delusions.

In addition to the correlations for the three major patient groups, separate correlational analyses were conducted for each of the four other psychotic groups (outlined in table 2b) to determine whether thought disorder at the first followup predicted outcome at the second followup in these different types of psychotic patients. In general, correlations in these four groups were similar to those of the schizophrenic patients, although the relationships were not always quite so strong. For example, the predictive correlation for the schizoaffective sample (n = 26) showed that thought disorder at the first followup significantly predicted overall outcome ($p < .05$), work functioning ($p < .05$), and presence of hallucinations ($p < .05$) at the second followup, despite the fact that this patient group was smaller in size than the schizophrenic group. Similarly, for the psychotic-depressive patients, thought disorder at the first followup significantly predicted subsequent overall outcome ($p < .05$), social functioning ($p < .05$), and delusions ($p < .01$) at the next followup. The predictive relationships for the other two psychotic patient groups showed some similarity to those of the schizoaffective and psychotic-depressed patients, but the correlations for these very small samples were lower and were not significant.

Subsequent Life Adjustment in Severely Thought-Disordered Schizophrenics. The correlations presented in table 6 provide evidence for positive relationships between posthospital thought disorder and subsequent life functioning for the various patient groups. A related issue is whether the presence of *severe* thought disorder at followup identifies a group of patients with an extremely negative clinical course. To analyze this issue, we categorized positive thought disorder into three levels, according to whether patients showed severe thought disorder (the focus of this analysis), signs of abnormal thinking, or no thought disorder. A 3 × 3 ANOVA (diagnostic group × level of thought disorder at first followup) of second followup life adjustment showed significant main effects for thought disorder in each outcome area, with the exception of social functioning (table 7). Levels of life adjustment showed increasingly greater impairment with more severe thought disorder across diagnostic groups.

Within schizophrenia, one-way ANOVAs showed significant thought disorder differences in overall outcome (the Strauss-Carpenter Scale) ($F = 6.09$, $df = 2/40$, $p < .01$), instrumental work functioning ($F = 7.05$, $df = 2/40$, $p < .01$), rehospitalization ($F = 3.93$, $df = 2/40$, $p < .03$), and delusions ($\chi^2 = 6.36$, $df = 2$, $p < .05$) at the second followup.

Post hoc comparisons of functioning (Newman-Keuls Test) among schizophrenic patients showing no thought disorder, abnormal thinking, and severe thought disorder indicated that *severely thought-disordered patients* showed significantly poorer overall outcome (Strauss-Carpenter Scale), poorer work functioning ($p < .05$), and rehospitalization ($p < .05$) 2 years after the cognitive assessment than did non-thought-disordered schizophrenics or those showing abnormal thinking. *Severely thought-disordered schizophrenic patients* also were significantly more likely to show persistent signs of delusions than were non-thought-disordered schizophrenic patients ($\chi^2 = 4.37$, $df = 1$, $p < .04$).

A look at the absolute scores of the subgroup of 10 schizophrenics who were *severely thought disordered* at the *first* followup shows that 8 of these 10 schizophrenic patients subsequently had very poor overall outcomes at the second followup (using the LKP Scale), and the other two schizophrenic patients showed equivocal outcomes. In addition, seven of these nine schizophrenic patients were delusional at the second followup (the 10th schizophrenic had committed suicide).

In considering the relationship between the presence of positive thought disorder in schizophrenia and poor subsequent life adjustment, we should emphasize that many schizophrenic patients show difficulty in subsequent life functioning and behavioral deficits. Only a small to moderate percentage of schizophrenics show very favorable outcomes (Harrow et al. 1978; Harrow, Carone, and Westermeyer 1985). Within this general background of difficulty in life functioning, however, the above data indicate that the great majority of schizophrenic patients who showed severe thought disorder after the acute phase tended to show particularly poor subsequent outcomes.

Subsequent Life Functioning of Non-Thought-Disordered Schizophrenics. While none of the schizophrenic patients who were severely thought disordered at the first followup were fully functional at the second followup, 45 percent of the non-thought-disordered schizophrenic patients also demonstrated poor outcomes. Analyses of the differences between non-thought-disordered schizophrenic patients who showed successful ($n = 12$) and impaired ($n = 12$) adjustments at the second followup suggested that non-thought-disordered schizophrenic patients with poor outcome showed trends toward the following poor prognostic signs at the first followup: (1) more severe negative and deficit symptoms as indicated by psychomotor retardation ($t = 1.72$, $df = 22$, $p < .10$), concrete thinking ($t = 1.44$, $df = 16$, $p < .20$), and ratings of a blank, expressionless face ($\chi^2 = 2.37$, $df = 1$, $p < .20$; (2) more severe anxiety ($t = 1.95$, $df = 22$, $p < .07$); and (3) more severe levels of depression ($t = 2.12$, $df = 22$, $p < .05$). Lower levels of premorbid educational achievement also characterized the non-thought-disordered patients with poor outcome ($t = 2.25$, $df = 22$, $p < .05$). These factors, present at the first followup or earlier, were associated with at least a moderate proportion of later adjustment difficulties and poor outcome in the non-thought-disordered group of schizophrenic patients.

Thought Disorder in Nonschizophrenic Patients as a Predictor of Subsequent Life Functioning. Our data indicate a clear relationship between posthospital thought disorder and subsequent clinical course and life adjustment in schizophrenia. The question then arises as to whether this relationship is specific to schizophrenia or applies to all types of patients with disordered thinking.

For the current samples of patients, the severity of positive thought disorder was also predictive of adjustment in nonschizophrenic patients (table 6), although the relationship was not uniform for all types of nonschizophrenic patients.

Analysis of the subgroup of 10 *nonpsychotic* patients with severe thought disorder at first followup showed them with generally poor outcome at second followup (see table 7). One-way ANOVAs demonstrated that severely thought-disordered nonpsychotic patients showed significantly worse subse-

Table 7. Means and standard deviations of patient adjustment in each diagnostic and thought disorder group

		Level of thought disorder (TD) at 1st followup											
		No thought disorder			Abnormal thinking			Severe thought disorder					
		Life adjustment means at 2nd followup											
Area of life adjustment	Group	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	Diagnosis *F*	TD *F*	Diagnosis × TD *F*
Overall outcome[1] (Levenstein, Klein, & Pollack 1966)	Schizophrenic	24	5.08	2.69	11	4.82	2.23	10	6.80	1.48			
	Psychotic nonschizophrenic	36	3.42	2.50	14	4.57	2.68	16	4.75	2.72	11.68	11.23	—
	Nonpsychotic	54	2.41	1.92	16	2.25	1.81	10	5.40	2.27	($p < .001$)	($p < .001$)	
Overall outcome[2] (Strauss & Carpenter 1972)	Schizophrenic	21	10.60	3.03	11	10.18	3.40	10	6.33	3.46			
	Psychotic nonschizophrenic	36	12.00	3.64	13	11.46	3.23	15	10.60	3.92	9.40	11.01	—
	Nonpsychotic	54	13.41	2.81	16	12.31	3.63	10	9.20	3.11	($p < .001$)	($p < .001$)	
Work functioning[3]	Schizophrenic	21	2.10	1.65	11	2.45	1.63	10	.30	.48			
	Psychotic nonschizophrenic	36	3.06	1.37	13	2.23	1.74	15	2.27	1.58	10.58	10.32	—
	Nonpsychotic	54	3.26	1.26	16	3.56	1.09	10	1.90	1.73	($p < .001$)	($p < .001$)	
Social functioning[3]	Schizophrenic	21	2.75	1.45	11	3.00	1.41	10	2.20	1.75			
	Psychotic nonschizophrenic	36	3.03	1.23	13	3.62	.77	15	3.40	1.06	3.37	—	2.54
	Nonpsychotic	54	3.56	.90	16	2.75	1.73	10	2.90	1.45	($p < .05$)		($p < .05$)
Rehospitalization[3]	Schizophrenic	21	3.60	.60	11	3.73	.47	10	2.90	1.20			
	Psychotic nonschizophrenic	36	3.56	.94	13	3.31	1.18	15	3.33	.90	—	4.76	—
	Nonpsychotic	54	3.83	.61	16	3.88	.50	10	3.20	1.03		($p < .01$)	

[1] Scores range from adequate (1) to poor (9) adjustments.
[2] Scores range from adequate (16) to poor (0) adjustments.
[3] Scores range from adequate (4) to poor (0) adjustments.

quent overall adjustment than non-thought-disordered nonpsychotic patients ($F = 10.68$, $df = 2/78$, $p < .001$). The thought-disordered nonpsychotic patients also showed significantly higher rates of rehospitalization ($F = 4.26$, $df = 2/78$, $p < .02$), more impaired social functioning ($F = 3.57$, $df = 2/78$, $p < .04$), and greater problems in instrumental work performance ($F = 5.66$, $df = 2/78$, $p < .01$) than their non-thought-disordered counterparts. A significantly greater frequency of severely thought-disordered nonpsychotic patients vs. non-thought-disordered and moderately thought-disordered nonpsychotic patients showed delusions at the second followup ($\chi^2 = 19.84$, $df = 2$, $p < .001$) (see table 5). In contrast to the schizophrenic patients, the majority of nonpsychotic patients who showed no signs of thought disorder at the first followup had returned to full functioning by the second longitudinal assessment.

Longitudinal outcome differences were not significant across the thought disorder levels within the *psychotic* nonschizophrenic group. When one-way ANOVAs were conducted, however, even these other psychotic patients showed minor trends in the expected direction for overall adjustment at the second followup ($F = 1.92$, $df = 2/61$ $p < .16$), work functioning ($F = 2.28$, $df = 2/61$, $p < .12$), and delusions ($\chi^2 = 3.77$, $df = 2$, $p < .16$). In general, psychotic nonschizophrenic patients, the most heterogeneous diagnostic group, exhibited somewhat weaker relationships between thought disorder and outcome than did schizophrenics. Yet, the four significant predictive correlations in table 6 for the psychotic nonschizophrenic patients indicate that even among these other psychotic patients, there was some trend for positive thought disorder at the first followup to predict outcome at the second followup.

Relationships Between the Longitudinal Course of Thought Disorder and Life Functioning at the Second Followup. The outcome status of schizophrenic patients who differed in the *course* of thought disorder between the first and second followup was also examined. One of the goals of the current research program was to explore subtypes of psychopathology based on longitudinal symptom patterns. The primary research question here is: Are longitudinal patterns of thought disorder associated with other characteristics of outcome functioning over time?

Table 8 presents the means and standard deviations of adjustment at the second followup, and table 9 presents the rate of psychosis at the second followup for patients who experienced four different courses in the presence and persistence of thought pathology between the first and second followups. The focus of the subsequent analysis and discussion is on the schizophrenic group.

The data indicate that the longitudinal course of thought disorder was strongly associated with outcome. Within schizophrenia, one-way ANOVAs showed that patients who were thought disordered at both followups exhibited significantly more severe impairment than did non-thought-disordered patients ($F = 4.17$, $df = 3/39$, $p < .02$). The potential importance of positive thought disorder in schizophrenia and other psychiatric disorders is underscored by these data indicating poor outcome for patients with a clinical course characterized by thought disorder.

Mean outcome differences between schizophrenic patients showing thought disorder at the first but not the second followup (improved) and those showing thought disorder at the second but not the first followup (exacerbated or worsened) were not significant, although schizophrenic patients with increased thought disorder at the second followup showed minor trends toward poorer functioning in some other areas. Schizophrenic patients defined by episodic patterns of thought disorder exhibited outcomes that fell midway between the relatively better non-thought-disordered schizophrenic patients and the poorer adjustment and functioning of patients who were thought disordered at both followups. Consistent with other data in this report, the course of positive thought disorder in these patients with early schizophrenia was not significantly associated with social functioning at the second followup.

Assessment of the longitudinal *course* of positive thought disorder allowed us to focus on the relative prognostic strength of previously assessed thought disorder versus concurrent thought disorder as predictors of second followup outcomes. As reported, no significant differences in second followup outcome emerged between the two groups of schizophrenic patients with episodic thought disorder (i.e., those showing thought disorder at the first but not at the second followup versus those showing thought disorder at the second but not at the first followup). In the current sample of "episodic" schizophrenic patients, thought disorder present at the first followup was almost as effective in predicting outcome at the second followup as it was for "episodic"

Table 8. Relation between course of thought disorder and life adjustment at 2nd followup

		Thought disorder (TD) course from 1st (FU1) to 2nd followup (FU2)														
		No TD at FU1 or FU2			TD at FU1 but not at FU2			TD at FU2 but not at FU1			TD at both FU1 and FU2					
		Life adjustment means at FU2														
Area of life adjustment	Group	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	Diagnosis *F*	TD course *F*	Diagnosis × TD course *F*
Overall outcome[1] (Levenstein, Klein, & Pollack 1966)	Schizophrenic	10	3.70	1.57	5	4.20	2.17	10	4.90	2.64	16	6.25	1.91			
	Psychotic nonschizophrenic	27	3.00	2.24	12	3.50	2.11	9	4.67	2.96	16	5.00	2.58	6.86 ($p < .001$)	7.95 ($p < .001$)	—
	Nonpsychotic	45	2.27	1.97	16	3.06	2.35	9	3.11	1.54	10	4.10	2.72			
Overall outcome[2] (Strauss & Carpenter 1972)	Schizophrenic	10	10.90	2.51	4	9.75	3.77	10	10.30	3.59	16	8.12	3.95			
	Psychotic nonschizophrenic	26	12.54	2.98	12	11.92	3.50	8	10.25	5.12	16	10.31	3.59	5.43 ($p < .01$)	7.06 ($p < .001$)	—
	Nonpsychotic	45	13.47	2.95	16	12.38	3.22	9	13.11	2.09	10	9.10	3.69			
Employment[3]	Schizophrenic	10	2.60	1.51	4	2.40	1.82	10	1.60	1.71	16	1.12	1.50			
	Psychotic nonschizophrenic	26	3.11	1.28	12	2.33	1.61	8	2.89	1.69	16	2.19	1.68	6.27 ($p < .01$)	3.52 ($p < .05$)	—
	Nonpsychotic	45	3.29	1.25	16	3.06	1.53	9	3.11	1.36	10	2.70	1.70			
Social function[3]	Schizophrenic	10	2.70	1.57	4	2.40	1.82	10	2.80	1.40	16	2.69	1.60			
	Psychotic nonschizophrenic	26	3.07	1.27	12	3.75	.62	8	2.89	1.17	16	3.31	1.08	—	—	3.61 ($p < .01$)
	Nonpsychotic	45	3.56	.94	16	3.56	1.03	9	3.56	.73	10	1.60	1.65			
Rehospitalization[3]	Schizophrenic	10	3.70	.48	4	3.80	.45	10	3.50	.71	16	3.19	1.05			
	Psychotic nonschizophrenic	26	3.67	.73	12	3.50	.67	8	3.22	1.39	16	3.19	1.22	—	3.53 ($p < .05$)	—
	Nonpsychotic	45	3.84	.64	16	3.75	.77	9	3.78	.44	10	3.40	.84			

[1] Scores range from adequate (1) to poor (9) adjustments.
[2] Scores range from adequate (16) to poor (0) adjustments.
[3] Scores range from adequate (4) to poor (0) adjustments.

schizophrenic patients whose thought disorder was present concurrently with the evaluation of their second followup outcomes. In some cases, signs of thought disorder were found at the second followup in schizophrenic patients who did not show other signs of psychosis concurrent with their cognitive assessment. Some patients who demonstrated an episodic course of disturbance with signs of positive thought disorder at the second followup did not show as much psychopathology or poor functioning in other areas of adjustment as expected. As we complete the third wave of followups, there will be a greater focus on the levels of functioning and psychosis of the subgroup of schizophrenic patients with an episodic course of thought disorder.

Discussion

Is Thought Disorder an Important Feature of Schizophrenia? The present research was designed to analyze two issues pertaining to whether positive thought disorder is a major feature of schizophrenia. The first involves the broad issue of the frequency and importance of positive thought disorder at various phases of schizophrenia. Is thought disorder a frequent characteristic of acute schizophrenia? Does it persist in some or many schizophrenics after the acute phase? And is it linked to other aspects of psychopathology? The second issue is whether the presence of thought disorder after the acute phase predicts subsequent clinical course and outcome.

These analyses stem, in part, from questions that recently have been raised about the presence and importance of positive symptoms in schizophrenia. Positive thought disorder, when it occurs, has always been a dramatic feature of schizophrenia, but recent research by others, and by our own group, has found that it is also present in manic patients and in other types of psychotic patients, and not just in schizophrenia (Andreasen and Powers 1974; Harrow and Quinlan 1977; Andreasen 1979; Johnston and Holzman 1979; Harrow et al. 1982; Harvey 1983; Oltmanns et al. 1985).

In general, discussions of the role of positive thought disorder have ranged from earlier views that it is *the* primary or essential feature of schizophrenia (Bleuler 1911/1950; Kasanin 1944; Rapaport, Gill, and Shafer 1946) to some recent views that positive thought disorder and positive symptoms in general are not important to diagnosis or prognosis (Pope and Lipinski 1978; Abrams and Taylor 1981). In earlier research, we explored whether positive thought disorder is a major symptom in schizophrenia, is one of several key symptoms, or is a minor accompanying feature of schizophrenia that has little or no significance. We tentatively concluded that positive thought pathology is one of several key symptoms in schizophrenia, but not the only major symptom of this disorder (Harrow, Silverstein, and Marengo 1983; Harrow and Quinlan 1985).

The data we have collected and analyzed does support the thesis that positive thought disorder is one of the central symptoms of schizophrenia at the acute phase. Thought disorder is present in a very high percentage of schizophrenic patients at the height of an acute episode, and it is more common in schizophrenic and manic patients than in other types of psychotic patients (Marengo and Harrow 1985). However, thought disorder is not present in all schizophrenic patients, as measured by our instruments. Schizophrenia is a disjunctive concept, and it is probable that no single symptom is characteristic of, or dominant in, all schizophrenics. Viewed from within this context, the current evidence supports the thesis that positive thought disorder is one of several key symptoms in acute schizophrenia, along with other positive symptoms such as delusions.

At the time of our earlier reports, we had begun to obtain data indicating that positive thought disorder is present after the acute phase in some or many schizophrenic patients and had begun to obtain evidence linking positive thought disorder to other major aspects of psychopathology (Harrow, Silverstein, and Marengo 1983). The presence of cognitive pathology at followup is supported by other more recent evidence that positive symptoms such as delusions and thought disorder are present after the acute phase in some but not all schizophrenic patients (Harrow, Carone, and Westermeyer 1985; Harrow, Marengo, and McDonald 1986; Marengo and Harrow, in press). The importance of thought disorder in schizophrenia is underscored by the current data indicating that when positive thought disorder is present after the acute phase in patients with early schizophrenia, it predicts subsequent poor outcome, at least for the next few years. Thus, when thought disorder is present after the acute phase, it is linked to other types of psychopathology (Harrow, Silverstein, and Marengo 1983; Harrow et al., in press; Harrow, Marengo, and McDonald, in press; Marengo and Harrow, in press) and tends to predict poor outcome in schizophrenia.

Positive thought disorder seems to be one of several key symptoms at the most acute phase. *After* the acute

Table 9. Relation between course of thought disorder and psychosis at 2nd followup

		Thought disorder (TD) course from 1st (FU1) to 2nd (FU2) followup							
		No TD at FU1 or FU2		TD at FU1 but not at FU2		TD at FU2 but not at FU1		TD at both FU1 and FU2	
		% of patients with psychosis at FU2							
Psychotic symptom	Group	*n*	%	*n*	%	*n*	%	*n*	%
Delusions	Schizophrenic	10	40	4	75	10	20	16	67
	Psychotic nonschizophrenic	26	19	12	16	8	50	16	44
	Nonpsychotic	45	4	16	25	9	11	10	50
Hallucinations	Schizophrenic	10	30	4	75	10	30	16	38
	Psychotic nonschizophrenic	26	11	12	25	8	38	16	12
	Nonpsychotic	45	9	16	6	9	11	10	20

TD = scores at moderate or severe level of thought disorder.

phase, when major psychopathology of almost all types begins to show a reduction, positive thought disorder and positive symptoms are still a factor, but at reduced levels of intensity for many, and in a smaller percentage of schizophrenic patients.

Finally, vis-à-vis the role of positive thought disorder as (1) a potential indicator of future schizophrenic psychopathology or (2) a part of an episode or an "illness," we view positive thought disorder primarily as an integral part of the active schizophrenic disorder. The ability of severe positive thought disorder to predict subsequent outcome and to identify a subgroup of poor outcome schizophrenics may stem from the fact that its presence at followup suggests the continuation of an *active* disorder after the most acute phase. The continuation of the active schizophrenic disorder in a very severe form after the acute phase could be a sign that the patient possesses a more severe form of the schizophrenic disorder, with a consequently poorer outcome.

Thought Disorder as a Predictor of Schizophrenic Outcome. Our framework for evaluating the prognostic importance of thought pathology hinges on two related assumptions: (1) that the presence of positive thought disorder after the most acute phase is linked to other basic aspects of schizophrenic psychopathology, and (2) that a continuum of thought disorder severity may be associated with a continuum of adjustment in other areas. Specifically, we have hypothesized that a hierarchy of thought disorder severity relates to a similar continuum in outcome in other areas and accounts for some of the heterogeneity in adjustment found in schizophrenia.

In our study, more severe forms of disordered thinking 2 years after hospitalization were linked to poorer overall functioning in a number of specific areas at a subsequent outcome assessment. The severity of thought disorder in postacute stages emerged as a behavioral referent that, combined with other important features of disorder, can increase predictive power.

Why Does Thought Disorder at the Posthospital Phase Predict Subsequent Outcome? It is possible that schizophrenic patients who show positive thought pathology or possibly other positive symptoms, such as delusions, after the acute phase have a more severe disorder than other schizophrenic patients. If so, then the presence of thought disorder after the acute phase is one index of the severity of the schizophrenic disorder, and possibly of the severity of underlying factors involved in schizophrenic pathology. Thus, the link between thought pathology—one of the most prominent symptoms in schizophrenia—and the severity of the schizophrenic disorder is not surprising. When the potentially disruptive effects of pathology and disorganization in the acute phase are removed (as at followup), and the schizophrenic patient still shows severe thought pathology, this is one index of a more severe and sustained schizophrenic disorder.

Do Schizophrenics Who Are Not Thought Disordered Show Poor Outcomes? While the presence of a severe thought disorder in schizophrenia at the first followup predicted sustained functional impairments, the absence of severely disturbed thinking did not preclude episodic or consistent difficulties in adjustment over time. Schizophrenic patients who showed no thought disorder or moderate (though not severe) cognitive symptoms exhibited a much less predictable course of disturbance. In particular, some non-thought-disordered schizophrenic patients demonstrated good subsequent posthospital adjustment and others showed major problems in work and psychotic symptomatology over time. On the basis of the current data and other data we have reported, we believe various factors, including thought pathology and other positive symptoms, are linked to poor outcome in schizophrenia.

Severe thought disorder which persists into postacute stages seems to be one indicator of later psychopathology and poor clinical course. Other data showing later dysfunctioning in some *non*-thought-disordered schizophrenic patients suggest multiple determinants of schizophrenic outcome. A number of factors must be invoked to account for (1) the poor overall functioning of a number of non-thought-disordered schizophrenic patients and (2) the relatively good overall adjustment of some severely thought-disordered psychotic *non*schizophrenic patients. Other data of ours indicate that the level of future adaptation is associated with a constellation of features of young psychiatric patients. These include negative symptoms, premorbid work and social competence, premorbid educational levels, and acute vs. gradual onset of disturbance (Westermeyer and Harrow 1984; Pogue-Geile and Harrow 1985). Research by Strauss and Carpenter (1974, 1977), our own research (Westermeyer and Harrow 1984), and that of others suggest that future work, social adjustment, and symptomatology are best predicted by earlier measures in each domain. In addition, the diagnosis of schizophrenia itself is a powerful correlate of poor overall adjustment later in the course of disturbance (Harrow et al. 1978; Grossman et al. 1984). Such results conform to a prognostic model emphasizing that features of the disorder, the person's history, and the social milieu to which the patient returns may all influence outcome (Huffine and Clausen 1979).

Subgroups of Schizophrenics Based on the Course of Thought Disorder. Analysis of the *course* of disordered thinking also allowed the schizophrenic sample to be divided into subgroups of patients with different short-term outcomes. As expected, subjects who were free of cognitive symptoms over both followups exhibited the fewest adjustment problems. By contrast, persistent cognitive symptoms were associated with a very poor short-term outcome in schizophrenic (and nonschizophrenic) patients. Individuals who demonstrated sustained thought pathology over both followups were least likely to remit over the short-term course.

While the most extreme differences in longitudinal functioning were between non-thought-disordered and severely thought-disordered groups, the mere presence of pathological thinking during followup at any point over the course of disturbance predicted subsequent decrements in life functioning. In general, episodic thought-disordered subgroups exhibited more benign outcomes than continuously thought-disordered subgroups. Patients of the former type may show differences in the severity of other aspects of psychopathology as they are followed over a longer period.

Positive Thought Disorder as a Predictor of Delusions. The potential relationship, or lack of a relationship, between various types of positive symptoms bears directly on theory about major psychopathology. Our recent data have indicated that, for schizophrenics, severe positive thought disorder at a first followup is strongly associated with delusional thinking at the same time period (Harrow, Silverstein, and Marengo 1983; Harrow, Marengo, and McDonald 1986). The current data indicate that severe positive thought disorder at the first followup predicts the extent of delusional thinking at the second followup. In addition, as we have noted in the first section of the **Results**, other recent research of ours with an *acute* phase hospitalized sample of patients provides data indicating that a large percentage of severely thought-disordered patients show some delusional activity (Marengo and Harrow 1985).

The current research supports a closer association between thought disorder and delusions than between thought disorder and hallucinations during the early course of schizophrenia. These data provide evidence for a stronger link between features of disorder that are more clearly ideational in nature (i.e., thought disorders and delusions) than between purely ideational features and perceptual symptoms.

Although the current investigation does not present data bearing on the directional link between thought disorder and delusions, the results suggest that associations between the severity of disordered thought and the presence of delusions may be stronger than between thought disorder and other outcome features. A stronger relationship exists between positive thought disorder and delusions for schizophrenia than for other psychiatric diagnoses, but this positive relationship also cuts across diagnostic lines. In addition, other results of ours indicate that the positive relationship between thought disorder and delusions occurred at both followups.

Relationship Between Severe Positive Thought Disorder and Subsequent Work Functioning. In the current sample of patients with early schizophrenia, the presence of severe thought disorder was also associated with work adjustment early in the course of disorder. Effective performance in work and thought is facilitated by adequate functioning in the ego-oriented areas of problem-solving, reasoning, planning, and organization. Good work performance also can be facilitated by a reasonable level of initiative, energy, and ability to apply oneself on a *sustained* basis. Severely thought-disordered patients in our sample exhibited significantly poorer subsequent work functioning than did non-thought-disordered patients and patients showing moderate thought disorder. Other analyses of the data on the present sample indicated that the best predictor of instrumental work performance in the posthospital period was work competence before the initial hospitalization.

Overall, the presence of severe thought disorder at the first followup successfully predicted subsequent poor work functioning at the second followup, but other factors also were effective predictors of posthospital work adjustment—for example, a schizophrenic diagnosis, preindex hospital history, and prior work functioning.

Conclusions

A series of investigations support the thesis that positive thought disorder is one of several key symptoms in schizophrenia. Thus, positive thought disorder is prominent and important for the great majority of schizophrenic patients at the acute phase, and is more common in schizophrenic and manic patients at that phase of disorder than in other psychotic and nonpsychotic patients.

After the acute phase, when major psychopathology of all types begins to show a reduction, positive thought disorder and other positive symptoms such as delusions are still important features, but at reduced levels of intensity for many and in a smaller percentage of patients. However, at followup, positive thought disorder is still more frequent in schizophrenic than in nonschizophrenic patients, is linked to other types of psychopathology and dysfunctioning, and emerges as a predictor of subsequent outcome in schizophrenia.

Positive thought disorder showed a slightly stronger relationship to subsequent functioning and psychopathology for the schizophrenic group, but it was generally significant for the nonschizophrenic patients as well. In addition, severe thought disorder at followup predicted subsequent poor work functioning and subsequent delusions. The relationship between severe positive thought disorder and both concurrent delusions and subsequent delusions in *schizophrenia* provides a link between two types of positive symptoms, both with strong cognitive-ideational components.

In general, when thought disorder occurs in schizophrenia, it should be viewed as an integral part of the schizophrenic disorder, rather than solely as an indicator of future psychopathology. At followup, when positive thought disorder is present, it forms part of a larger picture of residual psychopathology after the acute phase. The continuation or emergence of an active schizophrenic disorder at followup suggests a more severe and sustained illness, with a poorer subsequent prognosis.

References

Abrams, R., and Taylor, M.A. Importance of schizophrenic symptoms in the diagnosis of mania. *American Journal of Psychiatry*, 138:658–661, 1981.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

Andreasen, N.C. Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1331, 1979.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia. *Archives of General Psychiatry*, 39:789–794, 1982.

Andreasen, N.C., and Powers, P.S. Overinclusive thinking in mania and schizophrenia. *British Journal of Psychiatry*, 125:452–456, 1974.

Arieti, S. *Interpretation of Schizophrenia*. New York: Basic Books, 1974.

Asarnow, R.F., and MacCrimmon D.J. Residual performance deficit in clinically remitted schizophrenics: A marker of schizophrenia? *Journal of Abnormal Psychology*, 87:597–608, 1978.

Bleuler, E. *Dementia Praecox or The Group of Schizophrenias*. (1911) Translated by J. Zinkin. New York: International Universities Press, 1950.

Braff, D.L.; Glick, I.D.; and Griffin, P. Thought disorder and depression in psychiatric patients. *Comprehensive Psychiatry*, 24:57–64, 1983.

Cameron, N. Schizophrenic thinking in a problem-solving situation. *Journal of Mental Science*, 85:1012–1035, 1939.

Cancro, R. Clinical prediction of outcome in schizophrenia. *Comprehensive Psychiatry*, 10:349–354, 1969.

Chapman, L.J. Recent advances in the study of schizophrenic cognition. *Schizophrenia Bulletin*, 5:568–580, 1979.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia*. New York: Appleton Century Crofts, 1973.

Endicott, J., and Spitzer, R.L. Diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35:837–844, 1978.

Fish, F.J. *Schizophrenia*. Baltimore: Williams & Wilkins Co., 1962.

Goldstein, K., and Scheerer, M. Abstract and concrete behavior: An experimental study with special tests. *Psychological Monographs*, 53:Whole No. 2, 1941.

Gorham, D. Use of the proverbs test for differentiating schizophrenics from normals. *Journal of Consulting and Clinical Psychology*, 20:434–440, 1956.

Grinker, R.R., Sr., and Harrow, M. *A Multidimensional Approach to Clinical Research in Schizophrenia*. Springfield, IL: Charles C Thomas, Publisher, in press.

Grinker, R.R., Sr., and Holzman, P.S. Schizophrenic pathology of young adults: A clinical study. *Archives of General Psychiatry*, 28:168–175, 1973.

Grossman, L.S.; Harrow, M.; Fudala, J.L.; and Meltzer, H.Y. The longitudinal course of schizoaffective disorders: A prospective follow-up study. *Journal of Nervous and Mental Disease*, 172:140–149, 1984.

Harrow, M.; Bromet, E.; and Quinlan, D. Predictors of posthospital adjustment in schizophrenia: Thought disorders and schizophrenic diagnosis. *Journal of Nervous and Mental Disease*, 158:25–36, 1974.

Harrow, M.; Carone, B.J.; and Westermeyer, J.F. The course of psychosis in early phases of schizophrenia. *American Journal of Psychiatry*, 142:702–707, 1985.

Harrow, M.; Grinker, R.R., Sr.; Silverstein, M.L.; and Holzman, P.S. Is modern-day schizophrenic outcome still negative? *American Journal of Psychiatry*, 135:1156–1162, 1978.

Harrow, M.; Grossman, L.S.; Silverstein, M.L.; and Meltzer, H.Y. Thought pathology in manic and schizophrenic patients: Its occurrence at hospital admission and seven weeks later. *Archives of General Psychiatry*, 39:665–671, 1982.

Harrow, M.; Grossman, L.S.; Silverstein, M.L.; Meltzer, H.Y.; and Kettering, R.L. A longitudinal study of thought disorder in manic patients. *Archives of General Psychiatry*, in press.

Harrow, M.; King, G.; Marengo, J.; Rattenbury, F.; and Silverstein, M. "A Manual to Assess Positive Thought Disorder, Using the Object Sorting Test." Unpublished manual, 1985.

Harrow, M.; Lanin-Kettering, I.; Prosen, M.; and Miller, J.G. Disordered thinking in schizophrenia: Intermingling and loss of set. *Schizophrenia Bulletin*, 9:354–367, 1983.

Harrow, M.; Marengo, J.; and McDonald, C. The early course of schizophrenic thought disorder. *Schizophrenia Bulletin*, 12:208–224, 1986.

Harrow, M., and Miller, J.G. Schizophrenic thought disorders and impaired perspective. *Journal of Abnormal Psychology*, 89:717–727, 1980.

Harrow, M., and Quinlan, D. Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry*, 34:15–21, 1977.

Harrow, M., and Quinlan, D. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, 1985.

Harrow, M.; Silverstein, M.L.; and Marengo, J. Disordered thinking: Does it identify nuclear schizophrenia? *Archives of General Psychiatry*, 40:765–771, 1983.

Harvey, P. Speech competence in manic and schizophrenic psychoses: The association between clinically rated thought disorder and cohesion and reference performance. *Journal of Abnormal Psychology*, 92: 368–377, 1983.

Hollingshead, A.B., and Redlich, R.C. *Social Class and Mental Illness*.

New York: John Wiley & Sons, 1958.

Holzman, P.S. Cognitive impairment and cognitive stability: Toward a theory of thought disorder. In: Serban, G., ed. *Cognitive Defects in the Development of Mental Illness.* New York: Brunner/Mazel, 1978. pp. 361–376.

Huffine, C.L., and Clausen, J.A. Madness and work: Short- and long-term effects of mental illness on occupational careers. *Social Forces,* 57:1049–1062, 1979.

Hurt, S.W.; Holzman, P.S.; and Davis, J.M. Thought disorder: The measure of its changes. *Archives of General Psychiatry,* 40:1281–1285, 1983.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking.* San Francisco: Jossey-Bass, 1979.

Kasanin, J.W., ed. *Language and Thought in Schizophrenia.* New York: W.W. Norton and Co., 1944.

Knight, R.A.; Roff, J.D.; Barrnett, J.; and Moss, J.L. Concurrent and predictive validity of thought disorder and affectivity: A 22-year follow-up of acute schizophrenics. *Journal of Abnormal Psychology,* 88:1–12, 1979.

Levenstein, S.; Klein, D.F.; and Pollack, M. Followup study of formerly hospitalized voluntary psychiatric patients: The first two years. *American Journal of Psychiatry,* 10:1102–1109, 1966.

Marengo, J., and Harrow, M. Thought disorder: A function of schizophrenia, mania, or psychosis? *Journal of Nervous and Mental Disease,* 173:35–41, 1985.

Marengo, J., and Harrow, M. Schizophrenic thought disorder at followup: A persistent or episodic course? *Archives of General Psychiatry,* in press.

Marengo, J.; Harrow, M.; Lanin-Kettering, I.; and Wilson, A. A manual for assessing aspects of bizarre-idiosyncratic thinking. In: Harrow, M., and Quinlan, D., eds. *Disordered Thinking and Schizophrenic Psychopathology.* New York: Gardner Press, 1985. pp. 394–449.

Nuechterlein, K.H., and Dawson, M.E. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin,* 10:300–312, 1984.

Oltmanns, T.F.; Murphy, R.; Berenbaum, H.; and Dunlop, S.R. Rating verbal communication impairment in schizophrenia and affective disorders. *Schizophrenia Bulletin,* 11:292–299, 1985.

Payne, R. The long-term prognostic implications of overinclusive thinking in mental patients: A follow-up study using objective tests. *Proceedings of the IV World Congress of Psychiatry, Madrid, 1966.* New York: Excerpta Medica Foundation, 1968. pp. 2657–2659.

Piotrowski, Z.A., and Bricklin, B.A. A second validation of a long-term Rorschach prognostic index for schizophrenic patients. *Journal of Consulting Psychology,* 25:123–128, 1961.

Pogue-Geile, M.F., and Harrow, M. Negative and positive symptoms in schizophrenia and depression: A followup. *Schizophrenia Bulletin,* 10:371–387, 1984.

Pogue-Geile, M.F., and Harrow, M. Negative symptoms in schizophrenia: Their longitudinal course and prognostic value. *Schizophrenia Bulletin,* 11:427–439, 1985.

Pope, H., and Lipinski, J. Diagnosis in schizophrenia and manic-depressive illness. *Archives of General Psychiatry,* 35:811–828, 1978.

Rapaport, D.; Gill, M.; and Schafer, R. *Diagnostic Psychological Testing.* Chicago: Yearbook Publishers, 1946.

Spitzer, R.L.; Endicott, J.; and Robins, E. *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders.* 3rd ed. New York: New York State Psychiatric Institute, 1978.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: I. Characteristics of outcome. *Archives of General Psychiatry,* 27:739–746, 1972.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: II. Relationships between predictors and outcome variables. *Archives of General Psychiatry,* 31:37–42, 1974.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia: III. Five-year outcome and its predictors. *Archives of General Psychiatry,* 34:159–163, 1977.

Strauss, J.S.; Carpenter, W.T., Jr.; and Bartko, J.J. An approach to the diagnosis and understanding of schizophrenia: Part III. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin* 1 (Experimental Issue No. 11):61–69, 1974.

Strauss, J.S., and Docherty, J.P. Subtypes of schizophrenia: Descriptive models. *Schizophrenia Bulletin,* 5:447–452, 1979.

Wechsler, D. *Wechsler Adult Intelligence Scale Manual.* New York: Psychological Corporation, 1955.

Westermeyer, J.F., and Harrow, M. Prognosis and outcome using broad (*DSM-II*) and narrow (*DSM-III*) concepts of schizophrenia. *Schizophrenia Bulletin,* 10:624–637, 1984.

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Episodic and Residual Thought Pathology in Chronic Schizophrenics: Effect of Neuroleptics

by Herbert E. Spohn, Lolafaye Coyne, Jan Larson, Frederick Mittleman, Judy Spray, and Kenneth Hayes

Abstract

We sought to determine the effect of neuroleptic treatment on thought disorder in chronic schizophrenics, using the Thought Disorder Index (TDI). We were able to demonstrate that high levels of thought disorder are found in chronic schizophrenic and schizoaffective patients, while in both bipolar patients and normal controls thought pathology is minimal. Moreover, the TDI appears to be particularly well suited to the characterization of thought disorder in chronic schizophrenics. For total scores on the TDI, we were unable to demonstrate a significant reduction in thought pathology, although it was evident that thought disorder as reflected in the Conceptual Disorganization and Unusual Thought Content subscales of the Brief Psychiatric Rating Scale is significantly reduced by neuroleptics. In analyses of subscores of the TDI reflecting four levels of severity, we were able to show that more severe forms of thought pathology are normalized by neuroleptics, while less severe pathology is largely unaffected. Our results suggest that neuroleptic treatment reduces thought pathology associated with acute episodes, but that somewhat milder, though still disabling, residual thought pathology persists after clinical remission has been attained.

In this report we describe a study designed to assess the relation of neuroleptic treatment to thought disorder or, more precisely, to a measure of thought disorder, the Thought Disorder Index (TDI) (Johnston and Holzman 1979) in chronic schizophrenic inpatients. The rationale for this study is multifold.

Notwithstanding recent critiques of thought disorder as a pathognomonic symptom of schizophrenia (Pope and Lipinski 1978; Andreasen 1979*b*), the phenomenon continues to play an important role in the diagnosis of schizophrenia. If neuroleptic treatment normalizes thought disorder in schizophrenics, or modifies it in some other fashion, the validity of diagnosis in medicated patients is jeopardized. From the vantage point of descriptive psychiatry, therefore, it is of some interest to determine the effects of neuroleptic treatment on thought disorder. The absence of thought disorder symptoms reduced or removed by neuroleptic treatment may "bias" diagnostic judgment. Similarly, treaters of schizophrenic patients, be they behaviorists or psychotherapists, have an interest in knowing to what extent neuroleptics reduce thought pathology that may interfere with treatment. In untreated psychotic patients, acute thought pathology may divert attention from therapeutic interventions.

Studies of thought disorder continue to interest investigators concerned with the unraveling of the specific nature of cognitive dysfunction in schizophrenia (Chapman and Chapman 1973). Although the focus of interest has shifted in recent years to experimental studies of impairment in the earlier phases of information processing, there continues to be concern with disorders of central processing and with the response or performance side of information processing, i.e., language and communication (Rochester and Martin 1979; Maher 1983). Accordingly, an assessment of neuroleptic

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effects on thought disorder in medicated schizophrenics can play a clarifying role, both for studies of information processing as well as studies of central processing and expressive behavior.

There is a growing conviction on the part of investigators concerned with causation that the identification of biological markers (Holzman and Levy 1977) and markers of vulnerability (Nuechterlein and Dawson 1984) is a major step in the direction of the elucidation of etiology in schizophrenia. Quite typically, marker studies seek to determine the state/trait characteristics of behavior identified as having marker status potential. One widely accepted criterion for marker status is its persistence in patients whose clinical remission has resulted from neuroleptic treatment. Thus, another major aim of the present study, and perhaps its theoretically and clinically most interesting one, was to determine whether thought disorder or aspects of thought disorder merit trait (i.e., marker) status by being manifest as residual symptoms after a clinical remission has been achieved by neuroleptic treatment.

There is a rather small research literature that bears on neuroleptic effects on thought disorder. We review this literature here in order to show some of the conceptual and methodological problems that must be addressed in any effort to examine neuroleptic treatment-thought disorder relationships. This research literature extends over a period in which major changes in the conceptualization and measurement of thought disorder have taken place. Accordingly, we shall also be concerned with such important recent developments as Andreasen and Olsen's (1982) negative/positive symptom typology.

In an early and influential study, Orzack, Kornetsky, and Freeman (1967) found that while a phenothiazine (carphenazine) normalized attentional impairment (vigilance), this neuroleptic failed to affect "cognitive" impairment on the Wechsler Bellevue Digit Symbol Substitution Subtest (Wechsler 1944). This finding was in keeping with a then rather widely held view that phenothiazines were less effective in modifying cognitive functions in schizophrenics than attentional functions. This hypothesis has since been disconfirmed. Moreover, the Digit Symbol Substitution Test can hardly be considered a valid measure of thought disorder. Nonetheless, there is a much greater volume of research addressed to neuroleptic treatment effects on attention/information processing than to drug effects on disorders of central processing and response such as thought disorder, language, and communication.

Among the few studies in the latter area is one by Saretsky (1966), who, in a placebo-controlled design that employed measures of "primary process" thinking applied to Rorschach protocols, found that recently hospitalized chronic schizophrenic patients on chlorpromazine "proved to be more capable of handling primary process elements" than placebo-treated patients (p. 247). Moreover, improvement in this respect was positively correlated with clinical improvement, notwithstanding that "degree of thought pathology itself is not directly affected by drug treatment" (p. 247).

Unlike the study of Orzack, Kornetsky, and Freeman (1967), Saretsky's (1966) investigation attracted very little attention, is rather rarely cited, and has not been replicated. Three implications of this study are nonetheless of interest. The expression of thought pathology as measured in this study was episodic. That is, on drug treatment, patients verbalized less thought pathology than placebo controls, and less thought pathology than at admission and before drug treatment. Second, there was some residual thought pathology evident even after the achievement of symptomatic remission. Third, there is a hint in Saretsky's data that it was the expression of thought pathology rather than thought pathology itself that was reduced. This latter finding is consonant with an interpretation that self-editing processes were strengthened by neuroleptic treatment.

Two studies (Shimkunas, Gynther, and Smith 1966, 1967) reflect both conceptual and methodological problems in the determination of neuroleptic treatment effects on thought disorder. Acutely psychotic chronic schizophrenic patients were assessed on the Gorham Proverbs Test (Gorham 1956), the Wechsler Bellevue, and a global severity rating scale over a 26-week period of phenothiazine treatment. Placebo controls were not used in this study, whose central finding was that while pathology decreased, verbal IQ and proverbs scores did not increase.

Upon determining that the proverbs test scores were more highly correlated with verbal IQ than with psychopathology, Shimkunas, Gynther, and Smith (1967) devised an autistic response scoring system that was highly correlated with psychopathology. They found a significant decrease in autistic response frequency over the neuroleptic treatment period.

The results of these two studies raise a major conceptual issue in the measurement of thought disorder—namely, that of construct validity. The Gorham Proverbs Test, based on

Goldstein's (1939) theory of impaired abstracting ability, was found to lack construct validity, and the investigators were able to demonstrate construct validity for their autistic response scoring system, which appeared to be more sensitive to neuroleptic treatment than the concrete/abstract scoring system used by Gorham (1956).

There is yet another perspective that can be brought to bear on these two studies. It may be that abstract reasoning impairment in chronic schizophrenics is less susceptible to neuroleptic normalization than autistic reasoning. This interpretation suggests that some forms of thought pathology persist after symptomatic remission has been attained and are unaffected by drug treatment. Indeed, Harrow et al. (1972) have adduced evidence to this effect.

An attempt to replicate the findings of Shimkunas, Gynther, and Smith (1967) using their autistic response scoring system in a placebo-controlled study (Spohn et al. 1977) is instructive with respect to methodological issues. Spohn et al. (1977) did indeed find that in chronic schizophrenics returned to chlorpromazine treatment after a 6-week washout period, frequency of autistic responses on the Gorham Proverbs Test was substantially reduced. It could not be concluded, however, that chlorpromazine specifically reduced thought disorder of the kind measured by the Shimkunas scoring system because placebo control patients who remained testable after extended drug withdrawal also showed reduced autistic responses. Two factors combined to produce this counterintuitive finding. A number of placebo patients relapsed and therefore proved untestable. Accordingly, their data could not be included in drug/placebo comparisons. Also, there were some indications in the data obtained from the placebo patients who remained in remission during the testing period that they benefited from practice on repeated testing with the Gorham Proverbs Test, even though three alternate forms of the test were used.

The experience of Spohn et al. (1977) is not an isolated instance of unrepresentative placebo controls due to untestability of early relapsing patients. Payne et al. (1963), in a placebo-controlled study of the effects of a phenothiazine (Proketazine) on overinclusive thinking in chronic schizophrenics which produced negative results, comment:

> It must be emphasized that this [testable placebo sample] is not completely representative of chronic schizophrenic patients because of the need to eliminate 36% of the originally chosen group on account of disturbed behavior. [p. 530]

The Payne and Spohn studies pose very serious design and methodological problems confronting investigators concerned with the determination of neuroleptic effects on cognitive dysfunction in general and thought disorder in particular. The problem is especially acute in placebo-controlled studies using washout and on-placebo periods that are sufficiently long to permit reduction in neuroleptic control of psychotic symptoms. The duration of such periods is not coterminous with the 10- to 14-day washout required for the near total dissipation of drug substance and metabolites from the body system (Lacoursiere and Spohn 1976). It should be added that such problems can be mitigated neither by replacing relapsed placebo controls nor by estimating scores for untestable patients.

Perhaps the most successful demonstration of neuroleptic treatment effects on thought disorder is a study by Hurt, Holzman, and Davis (1983), which used the Thought Disorder Index (TDI) (Johnston and Holzman 1979) and involved a sample of schizophrenic inpatients with a relatively short hospitalization experience (80 days). These investigators were able to show that patients placed on haloperidol after a flexible 1- to 3-week washout period, and tested on completion of washout at 1, 3, 5, 12, and 19 days after washout and at discharge, steadily reduced the level of TDI scores (pathological verbalizations on the Rorschach) *and* that this pattern of TDI reduction was significantly congruent with clinical improvement as rated on the Brief Psychiatric Rating Scale (BPRS) (Overall and Gorham 1962). What mars this otherwise rather convincing demonstration of TDI sensitivity to neuroleptic treatment is that a no treatment-placebo control group was tested at only two points in the study period—namely, at 5 and 12 days after washout—and was not rated on the BPRS on these two occasions. Therefore, it was not possible to perform the same BPRS-TDI trend analyses over the total study period which had shown such convincing congruence in the drug-treated group. If such an analysis had revealed that an *increase* or *no change* in TDI scores over the same assessment period for which treated patient data were available was paralleled by a similar pattern of BPRS changes in the placebo group, it would be possible to rule out practice effects on the TDI and the effects of placebo/expectations and nonsomatic treatments on the BPRS ratings as factors leading to the kind of congruence of improvement on the TDI and BPRS in the haloperidol-treated group.

In addition, since the patient group in the study of Hurt, Holzman, and Davis (1983) is only rather sparsely described, it is difficult to determine comparability of this study to most of the studies heretofore discussed, all of which involved clearly chronic schizophrenics. On the other hand, unlike most other studies, the Hurt study did include normal controls and was therefore in a position to demonstrate that even at maximum benefit from neuroleptic treatment, the treated group manifested considerable residual thought pathology.

Andreasen's (1979*a*, 1979*b*) seminal studies of thought, language, and communication disorder in mania, depression, and schizophrenia have an important bearing on studies of neuroleptic treatment effects on thought disorder in schizophrenia. Of greatest relevance in the present context are her findings that with respect to "Bleulerian" thought disorder, manic and schizophrenic patients cannot be distinguished and, therefore, that Bleulerian symptoms are not pathognonomic of schizophrenia. This finding raises the question of specificity of thought disorder to schizophrenia. With the exception of the study of Hurt, Holzman, and Davis (1983), which used the well-standardized TDI, none of the other above described investigations were concerned with this issue. Moreover, there is undoubtedly considerable variation within this group of studies in diagnostic criteria and reliability in their application.

Andreasen and Olsen (1982) related Hughlings-Jackson's distinction between positive and negative symptoms to thought disorder in psychosis by identifying symptoms such as conceptual disorganization, delusions, and hallucinations as positive symptoms and symptoms such as anhedonia, alogia, and amotivation as negative symptoms. Their recommendation that these symptom types or categories be used as diagnostic/classification dimensions has found rather widespread acceptance. The relevance of these typologies to the study of neuroleptic treatment effects on thought disorder is not quite so salient as that of the specificity issue. Nonetheless, since there are some data suggesting that patients characterized by negative symptoms are less susceptible to neuroleptic treatment than patients with positive symptoms (Buchanan et al. 1975), this typology may have uses in research on neuroleptic treatment effects.

This critical literature review provides guidance both for the selection of thought disorder measurement instruments and for design and methodology in the development of a competent study of neuroleptic treatment effects on thought disorder in chronic schizophrenics. More specifically, it is our view that such instruments should be fairly broad-ranging in form and severity of thought pathology assessed, that some degree of construct validity should be demonstrable, and that the diagnostic boundaries of its applicability be specified. In design and methodology, care should be taken to obviate the loss of relapsed, untestable placebo patients, or stated more positively, to ensure that the placebo sample be as representative of the population as the on-drug sample. Moreover, since repeated measurement is inevitably a factor in the design of such studies, practice effects should be minimized and the schedule of repeated assessments in the on-drug group should parallel that in the placebo group. It will be seen below how well these specifications were met in our study.

Methods

Subjects. The core subject sample in this study comprised 100 chronic schizophrenics who agreed to participate after full disclosure of the nature of their participation and the risks entailed by drug withdrawal. All patients were rediagnosed by a project psychiatrist-social worker team who used the Schedule for Affective Disorders and Schizophrenia-Lifetime Version interview and the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1977). For all subjects, it was determined that they had manifested a therapeutic response to prior neuroleptic treatment. Patients with a history of organicity or substance abuse were excluded. The average age of the sample was 32.59 years, and average duration of illness was 9.7 years. Average cumulative hospitalization was 6.5 years, and average years of education totaled 12.58. Twenty-seven percent were female patients.

Also recruited to the project were 26 RDC-diagnosed schizoaffective disorder patients and 25 RDC-diagnosed bipolar (bipolar I) patients, as well as 39 normal controls. These diagnostic comparison groups were involved only in data analyses designed to determine TDI specificity.

Design. The 100 chronic schizophrenics were randomized to two conditions. A group of 36 patients remained on the active neuroleptic they had received before research entry throughout a 10-week study period. A second group of 64 patients were abruptly withdrawn from neuroleptics and placed on placebo matching in kind and dose the prewithdrawal neuroleptics they had received. No washout was used.

All patients were tested with an extensive performance test battery including the Rorschach and the Wechsler Adult Intelligence Scale (WAIS) in a baseline session before experimental assignment and while still fully medicated, and four times after experimental assignment at 2-week intervals. Also, on each of the five occasions of testing, patients were rated by a psychiatrist on the BPRS and by two research assistants on the Psychologic Rating Scale (King 1954). Testers, raters, project research nurses, patients, and hospital ward staff were unaware of patient drug status throughout.

Forty-four patients in the drug withdrawal condition and five patients in the active medication condition "relapsed" in the course of the 10-week study period. A patient was considered "relapsed" when both ward staff and project research nurses, who daily monitored the research patient's clinical condition, agreed that the first signs of symptom exacerbation were evident. No patient was permitted to undergo full-scale symptom exacerbation. A substantial proportion of relapsed patients were remedicated and completed the full five-test-session schedule.

For all relapsed patients, data obtained on the test session closest in time to their relapse were considered to be largely free of neuroleptic medication effects. Changes in test performance (other than those that could be imputed to practice) in the "relapse" session from test performance in the preexperimental assignment baseline session were regarded as reflecting medication effects. Another way of stating this assumption is to say that deterioration in test performance in relapsed patients was regarded as representing dysfunction normalized or otherwise modified by neuroleptic treatment.

While reduction in loss of data from untestable placebo patients was an important aim in this design, its effect was to reverse the roles of on-drug and placebo patients in the conventional, independent group drug evaluation design. Patients "on drug" in the present design served as controls for practice and attention effects; the drug-withdrawn, on-placebo patients served as experimental subjects. Drug withdrawal was the independent variable and test performance, the dependent variable. Drug effect hypotheses (i.e., the effect of neuroleptic treatment on thought disorder) were tested by contrasting baseline session to postwithdrawal session test changes in control patients (on drug) with those of experimental subjects (on placebo/relapsed).

A critical methodological problem associated with this unconventional design needs to be addressed. Randomization could not be extended to the "assignment" of drug-withdrawn patients to relapse and nonrelapse conditions. Factors determining when and whether a given patient relapsed could thus not be brought under experimental control by randomization. A consequence of this in the present study was one of significant dependent variable initial level differences among control, relapsed, and nonrelapsed groups.

Of the 44 patients who relapsed during the 10-week study period, 21 did so approximately 2 weeks after drug withdrawal, 11 about 4 weeks after withdrawal, and 12 during the remainder of the study period. Accordingly, 21 patients were considered relapsed in the second testing session (session 2), 11 in the third testing session (session 3), 5 in session 4, and 2 in session 5. Comparisons among these differing duration-to-relapse groups by means of multivariate analysis of variance (MANOVA) on BPRS symptom ratings scales, demographic and course of illness variables indicated that these several groups did not differ significantly. In the control group, relapse in five patients occurred in session 3 (for two) and in session 5 (for three). Drug-withdrawn patients who were not considered relapsed in sessions 2, 3, 4, and 5 did differ significantly from controls and relapsers in these several sessions principally in initial level (session 1) test scores, but on other variables as well.

That the relapsed patients differed in duration to relapse made possible several independent replicative tests of drug effect hypotheses. We present two such tests in the present report. The first involved data from 21 relapsers, 43 drug-withdrawn nonrelapsers, and 36 controls in session 2. The second test, involving session 3 data, compares relapsers (13: 11 withdrawn and 2 relapsed controls), nonrelapsers (29), controls (33), and 19 patients off drugs in session 2 and remedicated in session 3. Including the latter group in session 3 data analyses assured comparability of the results of analysis of session 2 and session 3 data sets because essentially the same groups of patients were involved in both analyses.

Tests, Rating Scales, and Scores. The Thought Disorder Index (TDI) is essentially a system for scoring pathological verbalizations on the Rorschach test and on the WAIS. Pathological verbalizations are weighted according to four levels of severity. "Minimal" severity of thought pathology (to which a .25 weight is assigned) comprises such response characteristics as concreteness, vagueness, word-finding difficulties, and perseveration. "Mild" severity (.50) reflects, among others, confused, loose, and fabulized responses. "Moderate" severity (.75) is represented by autistic logic, fluidity, absurd responses, and confabulations. "Maximally severe" responses (1.0) are contaminations, incoherence, and neologisms.

The Rorschach and the WAIS were administered in each of the five sessions by a qualified examiner who also administered a series of other tests that will be the subject of subsequent reports. All 10 cards of the Rorschach and four verbal subtests of the WAIS were administered in the baseline session (1) and in session 3. To mitigate practice effects, an achromatic Rorschach card (I), a red and black card (II), and a chromatic card (VIII) only were administered in session 2. Also, on these occasions, two verbal subtests of the WAIS (Vocabulary and Similarities) were administered. Interrater reliability in scoring using the TDI scoring manual, among three experienced clinical psychologists, was found to be $r = .84$ at the outset. A single senior clinical psychologist member of that group scored all Rorschach and WAIS protocols subsequently.

TDI scores employed in data analysis were (1) the sum of severity-weighted Rorschach verbalizations corrected by the number of Rorschach responses (TDR) and (2) the sum of weighted responses on the WAIS (TDW). Derived scores used in data analysis were presence (1) or absence (0) of one or more pathological responses on the Rorschach at each of the severity levels.

Brief Psychiatric Rating Scale. The 16 symptom subscales of the BPRS were administered by, for the most part, the same psychiatrist within a time band of ±24 hours of testing. Interrater reliability was established at the outset at $r = .87$. Several scores were derived from various combinations of the BPRS subscales for a variety of purposes in data analysis including a total pathology score, a schizophrenic symptom score (the mean of ratings on all subscales reflecting schizophrenic psychopathology), a positive/negative symptom index based on BPRS subscales: ([Blunted Affect + Emotional Withdrawal] − [Conceptual Disorganization + Unusual Thought Content])/([Blunted Affect and Emotional Withdrawal] + [Conceptual Disorganization and Unusual Thought Content]).

Psychologic Rating Scale. The Psychologic Rating Scale (King 1954) comprises subscales on which orientation to testing, cooperativeness, performance motivation, and attention to instructions in the test situation were rated by two examiners, one of whom observed patient behavior without awareness of performance efficiency. Interrater reliability was $r = .92$. The ratings of the latter examiner were considered measures of "General Deficit Functioning" and were used as such in data analyses.

Results

Brief Psychiatric Rating Scale. In an effort to determine whether patients considered relapsed in session 2 and session 3 did indeed manifest symptomatic exacerbation expected by design, changes in several BPRS scores from session 1 to 2 and from 1 to 3 for relapsed patients were compared with changes in control, nonrelapsed, and remedicated patients (in session 3 only) by analysis of covariance (ANCOVA). Session 1–2 and session 1–3 mean scores for "Total Pathology" and "Schizophrenic Symptoms" only are shown in table 1. ("Schizophrenic" symptoms represent mean ratings on all those subscales of the BPRS which clearly reflect psychopathology characteristic of schizophrenic patients.) Because of significant initial level differences among the several groups, initial level served as covariate in all analyses.

In session 1–2 contrasts, relapsers increased on all BPRS scores, while both controls and nonrelapsed patients decreased. In multivariate analysis of covariance (MANCOVA),

Table 1. Brief Psychiatric Rating Scale Total Pathology and schizophrenic symptom group means

	Total Pathology					Schizophrenic symptoms			
Group	S1	S2	S1	S3	Group	S1	S2	S1	S3
Control	23.11	21.69	22.82	22.53	Control	1.81	1.72	1.82	1.82
Nonrelapsed	16.47	15.21	15.62	13.54	Nonrelapsed	1.21	1.12	1.10	.953
Relapsed	18.50	22.18	19.64	23.51	Relapsed	1.39	1.69	1.59	1.89
Remedicated	—	—	17.53	19.13	Remedicated	—	—	1.24	1.37

Note.—S1, S2, S3 = session 1, session 2, session 3.

this pattern of differences in change was significant for all scores ($p \leq .01$).

In session 1-3 contrasts, relapsed patients *as well as* remedicated patients showed an increase in the several BPRS variables, while control patients remained at the same level in both sessions and nonrelapsed patients decreased somewhat. Again, in MANCOVA, these patterns of change differences were found to be significant ($p \leq .01$).

Several inferences may be drawn from these findings. Clearly, patients considered relapsed in sessions 2 and 3 manifested symptomatic exacerbation in these sessions. That remedicated patients also showed significant BPRS increases in session 3 implies that for most of these patients remedication had not yet regained symptomatic control and that therefore they could be construed to be in a state of functional relapse.

At the same time, mean increases in the BPRS variables in relapsed and remedicated patients, while significant, were not substantial, and in the distributions of BPRS scores in the several comparison groups, there was some degree of overlap. Accordingly, it must be granted that relapsed and remedicated patients were not in a state of acute, florid exacerbation of symptoms as a consequence of drug withdrawal. This inference is supported by the finding that on the Psychologic Rating Scale—which was construed to reflect general deficit functioning—relapsed and remedicated subjects in sessions 2 and 3 manifested marginally significant (S1–S2, $p < .08$) or nonsignificant increases (S1–S3, $p < .15$) over control and nonrelapsed patients.

These patterns of findings, in essence, imply that the "independent variable manipulation" aimed at in the design of the study was attained. Drug withdrawal in a substantial number of subjects in sessions 2 and 3 had resulted in symptom exacerbation reflecting some reduction in neuroleptic symptom containment. At the same time, this degree of symptom control reduction was not so great as to leave largely impaired the ability of relapsed and remedicated patients to render a scorable performance on the tests administered in the "relapse" sessions.

Several additional analyses were performed with three of the BPRS subscales: "Conceptual Disorganization," "Hallucinations," and "Unusual Thought Content." These analyses were done because the three subscales represent measures of thought disorder independent of the TDI and because they are readily identifiable as "positive symptoms" and were used as such in a Positive/Negative Symptom Index described above. The group means (see table 2) in session 1 for relapsed and remedicated patients were increased in sessions 2 and 3, while controls and nonrelapsed patients showed decreases. ANCOVA indicated that this pattern of change differences for all three subscales was significant ($p \leq .01$). One implication of this pattern of findings is that neuroleptic medication tends to normalize thought pathology reflected in these BPRS symptom subscales.

Specificity. In order to determine whether in the context of the present study, level of thought pathology as measured by the TDI was specifically characteristic of chronic schizophrenics, TDI scores for the latter were contrasted with TDI scores for 26 (RDC) schizoaffective patients and 25 (RDC) bipolar patients as well as for 39 normal controls. Table 3 shows mean TDI scores on the Rorschach (TDR) and the WAIS (TDW). Because of some departure from normality in most of these distributions of the raw scores, they were transformed by Freeman-Tukey transformations and are shown as such in table 3. Because of mean differences among these four groups in age, sex, and education, these variables served as covariates in one-way ANCOVA. The results of ANCOVAs performed both on TDR and TDW scores were virtually identical. Hence, only the TDR analysis is presented. In ANCOVA contrast of the four groups, an

Table 2. Group means on subscales of the Brief Psychiatric Rating Scale

	Conceptual Disorganization				Unusual Thought Content				Hallucinations			
Group	S1	S2	S1	S3	S1	S2	S1	S3	S1	S2	S1	S3
Control	1.83	1.73	1.82	1.83	3.00	2.76	3.00	2.83	1.89	1.79	1.79	1.78
Nonrelapsed	.884	.774	.724	.724	2.00	1.52	1.79	1.23	.907	.745	.621	.499
Relapsed	1.70	2.38	1.36	1.95	2.35	2.90	1.59	1.93	1.75	2.18	1.90	1.93
Remedicated	—	—	1.52	2.08	—	—	1.24	1.42	—	—	1.42	1.45

Note.—S1, S2, S3 = session 1, session 2, session 3.

Table 3. Thought Disorder Index group means: Schizophrenics, schizoaffectives, bipolars, and normal controls

	TDR		TDW	
Group	Mean	SD	Mean	SD
Schizophrenics	5.92	3.92	2.41	1.55
Schizoaffectives	5.10	3.03	2.29	1.44
Bipolars	3.08	2.59	1.68	1.02
Normals	3.47	2.46	1.47	.64

Note.—TDR = sum of severity-weighted Rorschach verbalizations, corrected by number of Rorschach responses. TDW = sum of weighted responses on the Wechsler Adult Intelligence Scale.

$F = 20.73$, $df = 3/179$, $p < .001$ was obtained. Orthogonal comparisons of schizophrenic patients with schizoaffective patients, and bipolar patients with normal controls, were not significant, while schizophrenic plus schizoaffective patients did differ significantly from bipolar patients and normal controls. These results are interpreted as implying that schizophrenic and schizoaffective patients manifested significantly greater thought pathology on the TDR than bipolar patients and normal controls and that within neither of these two pairs of groups were there significant differences in level of thought pathology. More generally speaking, these results suggest that a much higher level of thought pathology is characteristic not only of schizophrenic patients devoid of affective symptoms but also schizophrenics with marked affective symptomatology than is the case for affective disorder patients and normal controls.

Two further implications are worth noting. It comes as something of a surprise, in light of the research literature in this area, that in thought pathology schizoaffectives are more like schizophrenics than affective disorder patients. Moreover, it is equally surprising in light of Andreasen's (1979*a*) findings of similarities in "Bleulerian" thought pathology between schizophrenics and manic patients that in the present data bipolar patients (most of whom, to be sure, were not acutely psychotic) manifest significantly lower thought pathology on the TDI than schizophrenics. In order to clarify this latter finding, additional ANCOVAs were performed including only the three diagnostic groups and covarying out such nonspecific factors as general deficit functioning, duration of illness, and premorbid adjustment which were correlated positively with TDR in the schizophrenics and were greater in these patients than in the bipolars. ANCOVA applied to these data indicated that the differences continued to be significant ($F = 3.822$, $df = 2/141$, $p < .024$), although the removal of the three covariates "pulled" the adjusted means somewhat closer together. This finding provides the first hint in the present data that the TDI measures a distinctive form of thought pathology.

Correlates of TDI. In a further effort to clarify the nature of thought pathology measured by the TDI in chronic schizophrenics, it was correlated with demographic, symptom, and course of illness variables. In these correlations as well as in all subsequent data analyses, only the TDR was used, largely because it appears to be more sensitive to the elicitation of thought pathology than the TDW. Moreover, in the present as well as all subsequent data analyses, the TDR distributions were found to depart from normality and were accordingly transformed by means of a Freeman-Tukey transformation. More specifically, the variables correlated with TDR were sex, age, education, Total Pathology on the BPRS, the Positive/Negative Symptom Index (also BPRS based; see above), premorbid adjustment, duration of illness, and cumulative hospitalization time. Because of outlier duration-of-illness scores, a log transformation was applied. These correlations are shown in table 4. It will be evident from this table that severity of illness and chronicity indices dominate the correlational picture. The more severely ill and the longer the duration of illness and hospitalization, the greater the TDR thought pathology. The negative correlation of TDR with the Positive/Negative Symptom Index, which is, of course, strongly correlated with Total Pathology, indicates that positive symptoms vary directly with TDR severity.

Stepwise multiple regression analysis (also shown in table 4) generally confirms these inferences in demonstrating that length of hospitalization and severity of illness make independent contributions in accounting for TDR variance, and that sex (female patients manifested higher TDRs) and the Positive/Negative Symptom Index account for about 30 percent of the TDR variance. It is of some interest that the Positive/Negative Symptom Index accounts independently of BPRS Total Pathology for a minimal

Table 4. Correlates of TDR and stepwise multiple regression analysis

Predictors	*r*	Step	R^2	Incr. R^2	*F* to enter
Sex	.268[1]	1. Time hospitalized	.177	.177	19.58
Age	.323[2]	2. Total pathology	.254	.077	9.28
Education	.027	3. Sex	.292	.038	4.74
Positive/negative index	−.310[3]	4. Positive/Negative index	.305	.012	1.71
Total pathology	.396[2]				
Premorbid	.042				
Time hospitalized	.420[2]				
Duration of illness	.310[3]				

Note.—TDR = sum of severity-weighted Rorschach verbalizations corrected by number of Rorschach responses.

[1] $p < .05$, 2-tailed.

[2] $p < .001$, 2-tailed.

[3] $p < .01$, 2-tailed.

proportion of TDR variance. The general interpretation of these correlations is that the TDR is particularly sensitive to the kind of thought pathology characteristic of chronic schizophrenic patients and is to a quite limited extent a measure of "Bleulerian" positive symptom thought pathology. This does not imply, however, that the TDR is primarily a measure of "negative" symptoms.

TDR and Medication Effects. In table 5 the core findings of this study are shown: mean TDR in session 1 and session 2 for 21 relapsed patients, 39 nonrelapsed patients, and 31 control patients on active medication; and mean TDR in session 1 and session 3 for 12 patients considered relapsed in the latter session, for 19 patients relapsed in session 2 and remedicated in session 3, 25 nonrelapsed patients, and 28 controls. (All means reflect Freeman-Tukey transformations.) It will be obvious that there were significant initial level differences among the groups in both sessions. Accordingly, after homogeneity of regression of initial level on session 2 or 3 data in all groups was determined, MANCOVA was applied separately to session 1–2 and session 1–3 data. (Means in table 5 are covariance-adjusted.) The results for session 1–2 group comparisons indicated that although TDR in the relapsed group increased from session 1 to 2, while controls and nonrelapsers decreased, this pattern of change differences was not significant ($p < .229$). Similarly, in session 1 and 3 group comparisons, where both relapsed and remedicated patients increased in session 3, and controls and nonrelapsers decreased, this pattern of change differences was not significant ($p < .153$).

From a design/data-analytic perspective it is, of course, unfortunate that among the patients randomly assigned to the drug withdrawal condition, relapsers and nonrelapsers "self-selected" themselves in such a fashion as to give rise to sharp initial level TDR differences between these two groups. This finding implies differences between relapsers and nonrelapsers on other variables that determine duration to relapse after drug withdrawal. In order to mitigate the effect of these initial level differences on results, additional ANCOVAs were performed from which the apparently aberrant nonrelapsers were removed. These comparisons among controls and relapsers in session 1–2, and controls, relapsers, and remedicated patients in session 1–3, seemed justified conceptually. The results are, however, also negative, although the α levels were somewhat reduced (session 1–2, $p < .13$; session 1–3, $p < .12$).

Table 5. Group means for TDR in sessions 1 and 2, and sessions 1 and 3

Group	TDR		TDR	
	Session 1	Session 2	Session 1	Session 3
Controls	7.62	6.88	7.11	6.82
Nonrelapsed	4.27	3.76	3.46	3.19
Relapsed	6.58	6.87	6.17	6.23
Remedicated	—	—	6.49	7.41

Note.—MANCOVA contrasts of 3 groups in session 1–2: $p < .23$; MANCOVA contrasts of control/relapsed in session 1–2: $p < .13$; MANCOVA contrasts of 4 groups in session 1–3: $p < .15$; MANCOVA contrasts of controls/relapsed and remedicated, session 1–3: $p < .12$. TDR = sum of severity-weighted Rorschach verbalizations corrected by number of Rorschach responses.

In all these analyses, power sufficient to detect anything other than large mean differences was inadequate. It seems probable that with greater power the observed mean differences might have been significant. Even in this eventuality, however, it could not be concluded that the formal thought pathology measured by the TDR is normalized to more than a minimal extent by neuroleptic treatment in chronic schizophrenics.

These conclusions led us to a further consideration of the findings obtained in similar analyses of such positive symptom BPRS subscales as Conceptual Disorganization and Unusual Thought Content. For both of these scales (see table 2), with the same statistical power, the effect was large enough to yield significant mean differences with respect to patterns of change among all the comparison groups in sessions 1–2 and sessions 1–3. The directions of change differences suggest that these forms of thought pathology are substantially normalized by neuroleptic medication.

These forms of thought pathology are moderately correlated with TDR (see table 4), which is, it will be recalled, a *composite score* comprising *four levels* of thought pathology, the more severe of which are defined in terms similar to those of Conceptual Disorganization and Unusual Thought Content. On the basis of these considerations, we undertook a further set of TDR analyses in which this summary score was decomposed into its severity-weighted constituents. The aim of these analyses was to test the provisional hypothesis that significant change due to relapse was demonstrable only for the two most severe subscores (see **Methods** section) and not for the two less severe subscores. A further implication of this hypothesis is that neuroleptic treatment selectively normalized more severe thought pathology while leaving unmodified milder, less severe thought pathology as characterized by the TDR.

The scores for these analyses were in binary form: 0 = no pathological responses and 1 = ≥ 1 pathological response assigned to each of the four severity levels, "minimal" (.25), "mild" (.50), "moderate" (.75), and "severe" (1.0), for all patients. The proportions of pathological responses at each level in the prewithdrawal session (1) for all patients are shown in figure 1. The proportions are based on responses to all 10 Rorschach cards.

It is evident from figure 1 that minimal and mild thought pathology (reflecting concreteness, vagueness, and perseveration, as well as confusion, looseness, and fabulizations) is represented in substantially greater response proportion than moderate and severe thought pathology (reflecting autistic logic, absurd responses, incoherence, and neologisms). This may well explain why the Positive/Negative Symptom Index (comprising, among other subscales, Conceptual Disorganization and Unusual Thought Content) accounted for only a rather small proportion of the total TDR variance.

Table 6 presents proportions of present (1) and absent (0) thought pathology for control patients and relapsed patients at minimal, mild, moderate, and severe levels in sessions 1 and 2. For reasons already adduced above, nonrelapsers in session 2 were excluded in the analysis applied to these data. Table 7 shows the same data for controls and relapsed and remedicated patients combined in sessions 1 and 3. Combining the latter two groups seemed justified by the finding that both groups manifested symptomatic exacerbation in session 3.

Log linear analysis applied to these data yielded the following results. In session 1–2 (table 6) the χ^2 for the interaction of group by proportions of present/absent responses for minimal pathology in session 1 was significant ($p < .03$), indicating that the relapsed group showed a smaller proportion of pathological responses than the control group. No such significant χ^2 was obtained in session 2. The joint χ^2 for group comparison of the patterns of proportions in both sessions 1 and 2, however, was not significant. For mild thought pathology, neither the within-session interactions with group nor the between-session interactions with group were significant. It is concluded from this pattern of findings that neither control nor relapsed patients manifested an increase in minimal and mild thought pathology in session 2. Indeed, rather the opposite was true. Both groups showed reductions in session 2.

At the moderate and severe levels of TDR thought pathology, a

Figure 1. Percent thought pathology (TDR) at 4 levels of severity in session 1

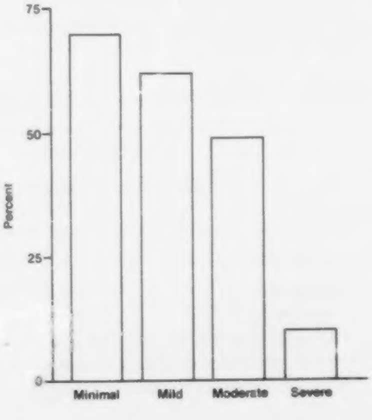

Table 6. Proportions of present/absent thought pathology by levels of TDR severity in sessions 1-2

Group	Minimal (.25)				Mild (.50)				Moderate (.75)				Severe (1.0)			
	Session 1		Session 2		Session 1		Session 2		Session 1		Session 2		Session 1		Session 2	
	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present
Control	.54	.46	.63	.37	.61	.39	.74	.26	.70	.30	.74	.26	.94	.06	.94	.06
Relapsed	.67	.34	.71	.29	.52	.46	.71	.29	.80	.20	.57	.43	.95	.05	.85	.15

Note.—TDR = sum of severity-weighted Rorschach verbalizations corrected by number of Rorschach responses.

somewhat different pattern emerges. In session 1, at both moderate and severe levels, the relapsed patients manifest a significantly smaller proportion of thought pathology than the controls ($p \leqslant .01$). In session 2, however, this pattern is reversed. Relapsed patients show a significant increase in proportion of both moderate and severe thought pathology, while the controls reduce thought pathology ($p \leqslant .01$). The joint χ^2 for session by group interactions at the moderate level (contrasting patterns of proportions between groups in session 1 and session 2) is significant ($p < .05$), while at the severe level, a marginally significant χ^2 ($p < .06$) is obtained. It thus appears that while at the minimal and mild levels significant differences in proportions of manifest thought pathology between controls and relapsed subjects within or between sessions could not be demonstrated, at the moderate and severe levels, relapsed patients relative to control patients and to themselves in session 1 showed significant increases in proportions of thought pathology in the session in which their relapse was manifest.

A similar pattern of results is seen in session 1–session 3 comparisons of controls, and relapsers and remedicated patients combined (see table 7). The χ^2 for the interaction of group by proportions of presence/absence of minimal thought pathology in session 1 and session 3 is significant for each session (session 1: $p < .02$; session 3: $p < .001$). The joint χ^2 contrasting the patterns of proportions between groups in both session 1 and session 3 is also significant ($p < .02$). Inspection of proportions shown in table 7 for minimal pathology indicates that both in session 1 and session 3 the relapsed/remedicated patients manifested less thought pathology than the controls, although both groups showed a reduction in pathology in session 3.

At the mild level in session 1, the group by present/absent proportions χ^2 is not significant. In session 3, however, this interaction is significant ($p < .002$). Moreover, the joint χ^2 contrasting the proportion patterns among groups for sessions 1 and 3 is marginally significant ($p < .06$). It appears, therefore, that while both controls and relapsed/remedicated patients showed reductions in mild thought pathology, the latter group did so to a significantly smaller extent than the former.

At both the moderate and severe levels of thought pathology, a different pattern of results obtains. Within-session interactions in sessions 1 and 3 are significant (session 1: $p \leqslant .01$; session 3: $p \leqslant .01$), and between-session interactions are similarly significant ($p \leqslant .03$). Inspection of table 7 indicates that at both moderate and severe

Table 7. Proportions of present/absent thought pathology by levels of TDR severity in sessions 1-3

Group	Minimal (.25)				Mild (.50)				Moderate (.75)				Severe (1.0)			
	Session 1		Session 3		Session 1		Session 3		Session 1		Session 3		Session 1		Session 3	
	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present
Control	.18	.82	.35	.65	.28	.72	.56	.44	.47	.53	.50	.50	.84	.16	.84	.16
Relapsed & remedicated	.32	.68	.41	.59	.29	.71	.43	.57	.61	.39	.35	.65	.87	.13	.80	.20

Note.— TDR = sum of severity-weighted Rorschach verbalizations corrected by number of Rorschach responses.

levels, relapsed/remedicated patients show a smaller proportion of pathological responses, while in session 3 this pattern is *reversed*; controls reduce proportions of pathological responses, while these are seen to be increased over session 1 in the relapsed/remedicated patients. From these results, both in session 1-2 and session 1-3 tests, the inference is drawn that neuroleptics tend to normalize moderate and severe thought pathology, while minimal and mild thought pathology is largely unaffected by these drugs.

Discussion

The central finding in this study is that more severe thought pathology in chronic schizophrenics, here defined both by subscales of the BPRS and scoring categories on the TDI, are to some extent normalized by neuroleptic medication, while less severe levels of thought pathology, specifically defined by types of scoring categories on the TDI, are unmodified by neuroleptic medication. Indeed, it may be said that the neuroleptics are not so much selective in normalizing *degree* of severity of thought pathology as they are selective in the *kind* of thought pathology that is reduced. Thus, it may be inferred that vivid, dramatic, gross distortions of reality, delusions, and hallucinations typically associated with an acute episode are reduced and even removed, while a kind of disorientation vis-à-vis reality, as well as concreteness and looseness, persists as residual thought pathology after a clinical remission has been attained by neuroleptic treatment. It remains now to examine the implications of these findings for diagnosis of medicated patients, for treatment and rehabilitation, for experimental study of thought disorder, for mechanisms of drug action, and for thought disorder as a trait in chronic schizophrenia.

From the perspective of descriptive psychiatry and diagnostic processes, the present findings suggest that the presenting clinical picture in medicated chronic schizophrenic patients will be to some extent distorted. The probable absence of Andreasen's "positive symptoms" exemplifies this point. Clinical psychologists will identify few severely pathological responses on psychological testing; neuropsychologists may interpret residual thought pathology as reflecting mild brain-based cognitive deficit.

In many mental hospitals, psychiatrists, already aware that medication may reduce presenting thought disorder in medicated new admissions, routinely withdraw such patients from neuroleptics in order to obtain a "pure" symptomatic and phenomenological picture. Investigators concerned with the experimental study of thought disorder would be well advised to do the same. Indeed, the present findings lend particular force to Chapman and Chapman's (1973) recommendation that experimental studies of thought disorder should be performed with drug-free schizophrenic patients only. At the same time, studies of language in medicated chronic schizophrenics are unlikely to reflect the full range of communication difficulties.

Our findings also have some implications for efforts at therapy and rehabilitation with chronic schizophrenic patients. The psychotherapist will find his medicated chronic schizophrenic patient less distracted by hallucinations and less preoccupied by and fixed on delusions. Thus, the process of therapy will proceed with a somewhat greater degree of attention to the immediate realities of the therapeutic situation.

Behavior therapists and social skills trainers (Wallace and Boone 1984) seeking to rehabilitate or even "habilitate" chronic schizophrenic patients have long recognized that such efforts are feasible only with medicated chronic schizophrenics. At the same time, it is generally agreed that social skills training is difficult to generalize and tends not to endure long beyond even persistent training efforts. Perhaps the present findings of residual concreteness, perseveration, vagueness, and looseness in thought processes may reflect the difficulties that social skills trainers encounter.

These findings suggest that remitted chronic patients do not experience events as connected. They have little confidence in the continuity of events and in their recurrence under specifiable circumstances. Their ability to derive meaning by induction is impaired. Subjectively, they experience reality as fragmentary, disjointed, and unpredictable. Perhaps most important of all, they sense a terrifying disparity between their ability to cope and the multiple challenges to cope with which family and community confront them.

The findings of this study leave unanswered questions regarding specific mechanisms whereby neuroleptic medication reduces the manifestation of the more severe aspects of thought pathology. On the basis of the research literature in this regard, two hypotheses are suggested. The drugs may specifically act on thought pathology such that the frequency with which grossly disordered thinking is in consciousness reduced or even eliminated. On the other hand, neuroleptics may not directly affect thought pathology but may strengthen the schizophrenic's awareness of the social inappropriateness of bizarre ideas and thus permit the "editing out" of these ideas in communication with such others in the present study as the Rorschach examiner and the psychiatrist interviewer. In view of the fact, however, that residual thought pathology as defined in the present study entails considerable lack of cognitive clarity, it seems unlikely that chronic patients' judgment of the significance of interpersonal situations would lead them to edit out all socially inappropriate ideation.

Finally, we address issues related to the question of whether residual thought pathology can be considered a trait of chronic schizophrenia as well as further implications of neuroleptic selectivity in normalizing thought pathology.

Our data satisfy some "trait" criteria but not others. We have shown that residual thought pathology is present both in clinical remission and in acute episodes. This fact satisfies an important criterion. Symptoms or dysfunctions that are modified or normalized by neuroleptic treatment clearly do not merit trait status. We have not been able to demonstrate, however, that what has been designated by us as residual thought pathology predates the initial onset of the illness and whether it has persisted throughout the entire course of illness. Our findings, however, should encourage research addressed to these issues because it may well lead to the identification of thought pathology as a marker of vulnerability in schizophrenia.

There is yet a further speculation that our findings of neuroleptic selectivity encourage. It may well be that those symptoms and dysfunctions that neuroleptics normalize are typically nonspecific to the illness. It is well known that neuroleptics do normalize "positive" symptoms in acute manics. On the other hand, those dysfunctions that neuroleptics do not normalize, e.g., residual thought pathology, eye tracking deviance (Holzman and Levy 1977), and skin conductance orienting response dysfunction (Bernstein et al. 1982), may well be illness-specific.

References

Andreasen, N.C. Thought, language, and communication disorders: I. Clinical assessment, definition of terms, and evaluation of their reliability. *Archives of General Psychiatry*, 36:1315–1321, 1979*a*.

Andreasen, N.C. Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1330, 1979*b*.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia. *Archives of General Psychiatry*, 39:789–794, 1982.

Bernstein, A.S.; Frith, C.D.; Gruzelier, J.H.; Patterson, T.; Straube, E.; Venables, P.H.; and Zahn, T.P. An analysis of the skin conductance orienting response in samples of American, British, and German schizophrenics. *Biological Psychology*, 14:155–211, 1982.

Buchanan, F.H.; Parton, R.V.; Warren, J.W.; and Baker, E.P. Double-blind trial of L-dopa in chronic schizophrenia. *Australian and New Zealand Journal of Psychiatry*, 9:269–271, 1975.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia.* New York: Appleton-Century-Crofts, 1973.

Goldstein, K. The significance of special mental tests for diagnosis and prognosis in schizophrenia. *American Journal of Psychiatry*, 96:575–587, 1939.

Gorham, D.R. Use of the proverbs test for differentiating schizophrenia from normals. *Journal of Consulting Psychology*, 20:435–440, 1956.

Harrow, M.; Tucker, G.; Himmelhoch, J.; and Putnam, N., Jr. Schizophrenic "thought disorders" after the acute phase. *American Journal of Psychiatry*, 128:824–829, 1972.

Holzman, P.S., and Levy, D.L. Smooth pursuit eye movements and functional psychoses: A review. *Schizophrenia Bulletin*, 3:15–27, 1977.

Hurt, S.W.; Holzman, P.S.; and Davis, J.M. Thought disorder—The measurement of its changes. *Archives of General Psychiatry*, 40:1281–1285, 1983.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking.* Washington: Jossey-Bass, 1979.

King, H.E. *Psychomotor Aspects of Mental Disease.* Cambridge, MA: Harvard University Press, 1954.

Lacoursiere, R.B., and Spohn, H.E. How long does chlorpromazine last? *Journal of Nervous and Mental Disease*, 163:267–275, 1976.

Maher, B.A. A tentative theory of schizophrenic utterance. In: Maher, B.A., ed. *Progress in Experimental Personality Research.* Vol. 12. New York: Academic Press, 1983. pp. 1–52.

Nuechterlein, K.H., and Dawson, M.E. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10:160–203, 1984.

Orzack, M.H.; Kornetsky, C.; and

Freeman, H. The effects of daily administration of carphenazine on attention in the schizophrenic patient. *Psychopharmacologia*, 11:31–38, 1967.

Overall, J.E., and Gorham, D.R. The Brief Psychiatric Rating Scale. *Psychological Reports*, 10:799–812, 1962.

Payne, R.W.; Friedlander, D.; Laverty, S.G.; and Haden, P. Overinclusive thought disorder in chronic schizophrenics and its response to "Proketazine." *British Journal of Psychiatry*, 109:523–530, 1963.

Pope, H.G., Jr., and Lipinski, J.F. Diagnosis in schizophrenia and manic illness. *Archives of General Psychiatry*, 35:811–828, 1978.

Rochester, S., and Martin, J.R. *Crazy Talk: A Study of the Discourse of Schizophrenic Speakers.* New York: Plenum Press, 1979.

Saretsky, T. Effects of chlorpromazine on primary-process thought manifestations. *Journal of Abnormal Psychology*, 71:247–252, 1966.

Shimkunas, A.M.; Gynther, M.D.; and Smith, K. Abstracting ability of schizophrenics before and during phenothiazine therapy. *Archives of General Psychiatry*, 14:79–83, 1966.

Shimkunas, A.M.; Gynther, M.D.; and Smith, K. Schizophrenic responses to the proverbs test: Abstract, concrete, or autistic. *Journal of Abnormal Psychology*, 72:128–133, 1967.

Spitzer, R.L.; Endicott, J.; and Robins, E. Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, 35:773–782, 1978.

Spohn, H.E.; Lacoursiere, R.B.; Thompson, K.; and Coyne, L. Phenothiazine effects on psychological and psychophysiological dysfunction in chronic schizophrenics. *Archives of General Psychiatry*, 34:633–644, 1977.

Wallace, J.C., and Boone, S.E. Cognitive factors in the social skills of schizophrenic patients: Implications for treatment. In: Spaulding, W.D., and Cole, J.K., eds. *Theories of Schizophrenia: Symposium on Motivation, 1983.* Lincoln, NB: University of Nebraska Press, 1984.

Wechsler, D. *The Measurement of Adult Intelligence.* Baltimore, MD: Williams and Wilkins, 1944.

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Attentional Vulnerability Indicators, Thought Disorder, and Negative Symptoms

by Keith H. Nuechterlein, William S. Edell, Margaret Norris, and Michael E. Dawson

Abstract

Deficits on two continuous performance test versions and the forced-choice span of apprehension task, which are potential vulnerability factors for schizophrenic disorders, were examined in relationship to particular symptoms of schizophrenic disorders, with emphasis on hypothesized relationships to formal thought disorder and negative symptoms. These interrelationships were determined concurrently within a group of 40 schizophrenic patients at an inpatient point. In addition, 32 of these patients were retested at a stabilized outpatient point to address the extent to which continued attentional deficits were associated with specific symptomatology during the hospitalized period. Signal-discrimination deficits on the three tasks were consistently associated with inpatient negative symptoms of schizophrenia as measured by the Anergia factor of the Brief Psychiatric Rating Scale (BPRS), across both the inpatient and outpatient assessments. The outpatient signal-discrimination deficits also showed significant, but less consistent, correlations with inpatient schizophrenic modes of thinking measured by the Rorschach Thought Disorder Index and with formal thought disorder measured by the BPRS Conceptual Disorganization rating. In contrast, no relationship with inpatient hallucinations or delusions was found. Combined with previous findings from high-risk samples, these results are consistent with the view that signal-discrimination deficits in situations demanding high levels of effortful processing are enduring vulnerability factors for schizophrenic negative symptoms and possibly for certain schizophrenic forms of thought disorder.

A series of recent studies has examined the possibility that certain deficits in attentional functioning and information processing might serve as indicators of personal vulnerability to schizophrenic disorders among some individuals within groups at increased statistical risk for these disorders (see reviews by Erlenmeyer-Kimling et al. 1982; Asarnow 1983; Nuechterlein and Dawson 1984). These studies have found that impairments that are present among many adult schizophrenic patients on specific laboratory performance tasks, such as the continuous performance test (CPT) and the forced-choice span of apprehension, are also found among a subgroup of the offspring of a schizophrenic parent and among nonpsychotic individuals at hypothesized risk due to schizotypal personality characteristics.

One important issue that has been raised recently is the extent to which such laboratory measures of cognitive processing are related to specific overt symptoms of schizophrenic disorders, as contrasted with the global diagnosis of schizophrenic disorder (George and Neufeld 1985; Neale, Oltmanns, and Harvey 1985). Associations with specific types of symptoms would help to explain the frequent observation that only certain schizophrenic patients show impairment on individual attentional or information-processing tasks (e.g., Kornetsky and Orzack 1978; Asarnow and MacCrimmon 1981) and would aid development of more specific models of the role of basic information-processing deficits in schizophrenic disorder. In the present study, we examine the extent to

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which signal-discrimination deficits in the CPT and the forced-choice span of apprehension, assessed during inpatient and stabilized outpatient periods, are related to specific symptom clusters among schizophrenic patients, particularly formal thought disorder and negative symptoms.

CPT and Forced-Choice Span of Apprehension Impairments

The CPT was originally developed by Rosvold et al. (1956) to detect deficits in sustained alertness in subjects with brain damage. The phrase "continuous performance test" suggests a single standardized testing procedure, but actually has referred to a group of visual vigilance tasks in which subjects monitor a continuous series of single letters or numbers that are presented very briefly (40–200 ms) one at a time with a relatively short, fixed interstimulus interval (1–1.5 seconds). The subject's task is to respond with a button press each time that a predesignated target stimulus appears in a random stimulus series. Variations of the task have included target stimuli that are single letters or numbers (Rosvold et al. 1956), sequences of two consecutive letters or numbers (Rosvold et al. 1956), or playing cards (Erlenmeyer-Kimling and Cornblatt 1978), perceptually degraded numbers (Nuechterlein 1983; Nuechterlein, Parasuraman, and Jiang 1983), or picture stimuli (Anderson et al. 1969).

Versions of the CPT that produce a relatively high momentary processing load through use of degraded stimuli or sequential targets have identified deficits in signal/noise discrimination (perceptual sensitivity) during vigilance in samples identified as at risk for schizophrenia on the basis of parental schizophrenia (Rutschmann, Cornblatt, and Erlenmeyer-Kimling 1977; Nuechterlein 1983; Cornblatt and Erlenmeyer-Kimling 1985) or personality test criteria for schizotypal features (Nuechterlein, Edell, and West, submitted for publication). Actively symptomatic schizophrenic patients (Asarnow and MacCrimmon 1978; Walker 1981) and relatively remitted schizophrenic patients (Wohlberg and Kornetsky 1973; Asarnow and MacCrimmon 1978) show similar target detection deficits on difficult versions of the CPT. Versions of the CPT that involve detection of a single, familiar target letter or number involve much more limited moment-to-moment processing load and have been successful in detecting deficits among chronic schizophrenic patients (Orzack and Kornetsky 1966) but not among children at risk for schizophrenia (Asarnow et al. 1977). Thus, signal/noise discrimination impairment during CPT versions with high processing loads, but not in versions with low processing loads, seems to reflect a vulnerability factor in schizophrenic disorder rather than any transient deficit that is secondary to psychotic symptomatology. In addition, inpatients with affective disorder and children of patients with affective disorder or with nonpsychotic disorders outside the schizophrenia spectrum do not appear to have this CPT performance deficit (Walker 1981; Nuechterlein 1983; Erlenmeyer-Kimling et al. 1984). Some hyperactive children (O'Dougherty, Nuechterlein, and Drew 1984) but not others (Nuechterlein 1983) do show this signal/noise discrimination deficit, so the deficit is not wholly specific to risk for schizophrenia, but hyperactive children show accompanying lower response criterion scores in signal detection theory analyses that suggest an additional impulsive responding component.

The forced-choice span of apprehension task has shown similar promise as a vulnerability indicator for schizophrenia. This procedure was developed to examine the efficiency of visual search in initial, sensory memory without burdening active short-term memory (Estes and Taylor 1964). Subjects are required to identify which of two target letters (usually T or F) appears in tachistoscopic presentations of letter arrays of varying sizes. Efficient target recognition in this task involves very rapid rejection of irrelevant, nontarget letters, without full processing to awareness, and effective discrimination of the critical features of the target letter.

The forced-choice span of apprehension task has detected a deficit in signal-discrimination accuracy among children at genetic risk due to parental schizophrenia (Asarnow et al. 1977) and among adults with schizotypal characteristics on personality inventory scales (Asarnow, Nuechterlein, and Marder 1983). Symptomatic schizophrenic inpatients (Neale et al. 1969; Neale 1971; Asarnow and MacCrimmon 1978) and relatively remitted schizophrenic outpatients (Asarnow and MacCrimmon 1978, 1981) show the same deficit. Schizophrenic inpatients score more deviantly than nonpsychotic inpatients (Neale 1971), and schizophrenic patients show greater deficits than manic-depressive patients among relatively recovered outpatients (Asarnow and MacCrimmon 1981).

The forced-choice span of apprehension shares with most CPT versions an emphasis on initial perceptual discrimination of briefly presented alphanumeric characters. Demands for acquisition of new information are minimal. Responses are relatively immediate, so long-term memory demands are limited to access to the stored representations of alphanumeric characters. One major difference concerns whether targets and nontargets are presented simultaneously in brief information bursts or sequentially in continuous trials. For this reason, the CPT has usually been referred to as a measure of sustained attention. However, the signal/noise discrimination deficit in the CPT among children at risk for schizophrenia appears to be present across all time periods of the task and the drop in signal/noise discrimination over time within task has not been significantly greater than normal (Nuechterlein 1983, 1985), so the schizophrenia-relevant deficit appears to be related to ongoing selection of target stimuli rather than to deficient target selection specifically at the end of a vigilance period. Thus, we will emphasize common features of the CPT and forced-choice span of apprehension by referring to the deficits on these two types of task as signal/noise discrimination impairments, or for convenience, as signal-discrimination impairments, while recognizing that the temporal nature of the discrimination demands are very distinctive. The lack of significant association between the forced-choice span of apprehension and a CPT version among schizophrenic outpatients in one report (Asarnow and MacCrimmon 1978) indicates that their differences are sufficient to merit separate examination of the clinical, symptomatic correlates of each of these potential indices of a vulnerability factor for schizophrenia.

Thought Disorder Index (TDI) and Schizophrenia

Formal thought disorder has long been considered a central clinical feature of the schizophrenic disorders (Bleuler 1911/1950; Kraepelin 1913/1919) and has been the most frequent focus of models that relate basic attentional and information-processing deficits to particular schizophrenic symptoms (Maher 1972; Neale and Oltmanns 1980; Schwartz 1982). In early research, the measurement of disordered thinking was based largely on global judgments of clinicians interacting in a relatively unstructured interview format with disturbed individuals. Concerns regarding reliability and validity of measurement were less salient. The seminal work of Rapaport, Gill, and Schafer (1946) involving extensive psychodiagnostic evaluations of individuals with various psychiatric disorders produced several categories of deviant verbalizations obtained from psychological test performance. Borrowing from this earlier work, Watkins and Stauffacher (1952) devised an index of pathological thinking called the "Delta Index," assigning weights from .25 to 1.0 to each type of verbalization to reflect degree of deviancy. This quantification of disordered thinking proved quite successful in discriminating schizophrenic from nonpsychotic populations using the Rorschach inkblot test (Powers and Hamlin 1955; Kataguchi 1959; Quirk et al. 1962).

To allow for the assessment of a wider range of types of thought slippage as expressed through verbal samples, Johnston and Holzman (1979) revised the Delta Index, renaming the refined measure the Thought Disorder Index (TDI). Categories which were felt to provide inadequate evidence of thought disorder, or which occurred too infrequently or were difficult to identify, were dropped. Categories which could be applied to the verbal subscales of the Wechsler Adult Intelligence Scale (WAIS), in addition to the Rorschach inkblot test, were added. Twenty scoring categories at four levels of severity were included, ranging from fairly subtle instances of cognitive slippage (e.g., vagueness, peculiar verbalizations, word-finding difficulties) to more grossly disturbed and psychotic levels of disordered thinking (e.g., contamination, incoherence, neologisms). The development of a fairly comprehensive, clearly written scoring manual containing multiple examples of each category of deviant verbalization resulted in quite respectable interrater reliability coefficients generally in the .80 to .90 range when applied to various diagnostic groups (Johnston and Holzman 1979).

Johnston and Holzman (1979) successfully discriminated chronic schizophrenics from groups of bipolar psychotic, neurotic, personality disordered, and normal control subjects based on overall TDI scores. A group of recently hospitalized schizophrenics with less than 1 year of lifetime hospitalization also scored more deviantly than the normal comparison subjects, but not more so than the other patient groups tested. The nonpsychotic patients were indistinguishable from the normal control subjects. The elevated scores of schizophrenic patients on the TDI were reflected in more grossly disturbed responses from the higher-level categories of deviant verbalizations. Interestingly, the TDI also discriminated the parents, but not the siblings, of recently hospitalized schizophrenic patients from those of the normal comparison subjects. Almost 90 percent of the schizophrenic patients with deviant TDI scores had at least one parent with high TDI scores as well, suggesting familial transmission of thought disorder. TDI scores were not significantly related to sex, ethnicity, socioeconomic status, IQ, Phillips ratings of premorbid adjustment, medication status at time of testing, or ratings of paranoia.

Further evidence for the usefulness of the TDI as a sensitive index of thought disorder has been reported by Holzman and colleagues. Hurt, Holzman, and Davis (1983) examined changes in thought disorder and symptom behavior, as measured by the TDI and the Brief Psychiatric Rating Scale (BPRS), respectively, following the initiation of neuroleptic (haloperidol) or placebo treatment in schizophrenic inpatients. Significant reductions in scores on the TDI and on the BPRS were evident by the third day following the initiation of drug treatment. Changes on two BPRS factors, Thought Disorder and Hostility-Suspicion, closely followed the rate of change on the TDI. Other BPRS subscale factors, such as Anergia, Activity, and Anxiety-Depression, followed different time courses. Residual thought disorder in the drug-treated patients was evident in the fact that, despite almost a 50 percent drop in overall mean TDI scores in the schizophrenic patients after 3 weeks, these patients remained significantly more thought disordered than normal control subjects. These findings support the efficacy of the TDI as a reliable index of cognitive slippage as a function of time, treatment, and clinical manifestations of psychotic behavior.

Finally, using a slightly revised version of the TDI with 22 scoring categories (Solovay et al. 1985), Holzman, Solovay, and Shenton (1985) examined possible qualitative distinctions in the thought disorder of carefully diagnosed manic and schizophrenic patients. Overall TDI scores were not significantly different for these psychotic groups, although each exceeded the normal control group. A principal components analysis with varimax rotation with all psychotic subjects resulted in six interpretable factors: Idiosyncratic Verbalizations, Expansive Thinking, Autistic Thinking, Fluid Thinking, Absurdity, and Confusion. Expansive Thinking was clearly most common in the manic group, while the schizophrenic subjects produced most of the thought-disordered responses from the other categories. As an alternative method of producing scores on qualitatively different forms of thought disorder, a post hoc rational clustering of TDI categories that individually discriminated manic and schizophrenic patients was organized into five factors. Combinatory Thinking and Irrelevant Intrusions best characterized the manic patients, whereas Confused Thinking, Fluid Thinking, and Peculiar Verbalizations were more common in schizophrenic patients. These findings are consistent with the notion that overall levels of thought disturbance do not distinguish psychotic patients. However, more fine-grained qualitative investigations do suggest important differences in the ways that schizophrenic and manic individuals organize their thoughts.

Combining these findings, one can reasonably conclude that the TDI is a highly reliable and most promising measure of formal thought disorder among psychotic patients, including both overall thought disturbance level and fine-grained distinctions among categories of disturbance. This instrument should allow a more thorough examination of the relationship of deficits in laboratory cognitive measures with clinical forms of thought disorder than has been possible with previous broader measures. Within this study, we investigate the extent to which the CPT and span of apprehension signal-discrimination deficits that are hypothesized to be vulnerability factors for schizophrenic disorders are related to those TDI features that are more characteristically schizophrenic than manic disturbances.

Schizophrenic Negative Symptoms and Attentional Impairment

In addition to the possible contributions of fundamental attentional and information-processing deficits to formal thought disorder, recent emphasis on the distinction between positive and negative symptoms of schizophrenia (Strauss, Carpenter, and Bartko 1974; Crow 1980; Andreasen and Olsen 1982) raises the possibility that certain laboratory measures of attentional functioning may be more relevant to negative symptoms of schizophrenia than to the more florid positive symptoms. Andreasen (1982) included clinically rated attentional impairment with affective flattening, alogia, avolition-apathy, and anhedonia-asociality in her Schedule for the Assessment of Negative Symptoms (SANS). This symptom complex is represented by individual ratings for work inattentiveness, inattentiveness during the mental status testing, and subjective complaints of inattentiveness as well as a global rating across these areas. In the initial sample of 26 patients, these ratings correlated .49, .35, .19, and .51, respectively, with the composite score from all five major areas of proposed negative symptoms, suggesting that, except for the subjective complaints rating, moderate correlations with a negative symptom summary score were present (Andreasen 1982).

Further evidence of the possible association of clinically rated attentional impairment with other negative symptoms of schizophrenia comes from a sample of 52 *DSM-III* schizophrenic inpatients (Andreasen and Olsen 1982). The SANS rating of attentional impairment correlated .56, .56, and .53 with avolition-apathy, alogia, and anhedonia-asociality, respectively, but not at all (−.01) with a clinical rating of positive formal thought disorder. Two other positive symptom complexes, delusions and hallucinations, actually correlated negatively (−.56 and −.36, respectively) with clinically rated attentional impairment. Thus, the relationship of laboratory measures of attentional functioning with negative symptoms of schizophrenia clearly needs evaluation to determine whether these more objective and fine-grained measures of attentional deficit show a similar predominant relationship to presence of negative symptoms of schizophrenia.

A possible theoretical link between schizophrenic attentional impairment and negative symptoms derives from the recent suggestion that the pattern of deficits in attentional functioning and information processing in schizophrenic patients and groups at risk for schizophrenia is consistent with a reduction in available processing capacity (Nuechterlein and Dawson 1984). This hypothesis regarding the nature of an attentional impairment in schizophrenic disorder follows from observations that cognitive tasks that demand relatively high levels of effortful, capacity-loading processing (Kahneman 1973; Posner 1978; Beatty 1982) appear most likely to detect a deficit across schizophrenic patients and risk populations. A reduction in the pool of nonspecific processing resources that is available for task-relevant cognitive processing might be expected to contribute to clinical symptoms of apathy, alogia, affective flattening, and emotional withdrawal through restrictions on the depth and breadth of cognitive processing.

Hypotheses

Drawing on this background information, we used the degraded-stimulus CPT (Nuechterlein, Parasuraman, and Jiang 1983), a CPT with an alphanumeric sequence as the target (Rosvold et al. 1956), and a standard version of the forced-choice span of apprehension (Asarnow and MacCrimmon 1978) as laboratory measures of attentional and information-processing impairments in schizophrenia that also have promise as vulnerability indicators among risk populations. To summarize performance on each of the CPTs, we focused particularly on a signal-detection-theory index of overall signal/noise discrimination level, d′ (Green and Swets 1966; McNicol 1972), that has been productive in specifying the nature of CPT deficits in recent studies of children at risk for schizophrenia (Rutschmann, Cornblatt, and Erlenmeyer-Kimling 1977; Nuechterlein 1983). The span of apprehension performance was indexed by the target discrimination accuracy for the largest (10-letter) array, which has been found to be the most sensitive measure of schizophrenic deficits within this paradigm (Asarnow and MacCrimmon 1978, 1981).

We hypothesized that deficits in signal-noise discrimination on these tasks are contributory factors (1) to the types of formal thought disorder that are more frequent in schizophrenia than in mania and (2) to schizophrenic negative symptoms. We examined these hypotheses within a schizophrenic sample to determine whether the broad range of deficits on these laboratory measures within the schizophrenic population could be linked to the presence of formal thought disorder and negative symptoms in subgroups of schizophrenic patients. Specifically, for thought disorder assessed through the Rorschach, we hypothesized that signal-discrimination deficits on the CPT and span of apprehension would be related to the TDI factors that Holzman, Solovay, and Shenton (1985) found to be more characteristic of schizophrenic patients than of manic patients. We also hypothesized that these signal-discrimination impairments would be associated with thought disorder assessed through clinical ratings, specifically the Thought Disturbance factor and particularly the Conceptual Disorganization item of the Brief Psychiatric Rating Scale (BPRS) (Overall and Gorham 1962; Guy 1976).

Schizophrenic negative symptomatology was assessed by the Anergia factor of the BPRS (Overall and Gorham 1962). We hypothesized that within a schizophrenic sample, the signal-discrimination deficits on the CPT and the forced-choice span of apprehension would be associated with high ratings on the Anergia factor, which reflects affective flattening, motor retardation, emotional withdrawal, and disorientation.

The signal-discrimination deficits are hypothesized to serve as ongoing vulnerability factors rather than only temporary concomitants of acute schizophrenic symptoms. Therefore, to examine the association between the signal-discrimination deficits and schizophrenic symptomatology in a way that might shed light on whether the signal-discrimination deficits are more than secondary components of acute symptomatology, we evaluated the CPT and span of apprehension performance of a group of schizophrenic patients both during an inpatient period and a subsequent stabilized, outpatient period. Associations between signal-discrimination deficits during the stabilized outpatient period and clinical symptomatology during the inpatient period are particularly relevant to the hypothesis that the signal-discrimination deficits serve as enduring vulnerability factors.

Methods

Subjects. The 40 schizophrenic subjects are participants in a larger longitudinal follow-through study of the early phase of schizophrenic disorders entitled "Developmental Processes in Schizophrenic Disorders" (Principal Investigator: Keith H. Nuechterlein, Ph.D.; Co-Principal Investigator: Michael E. Dawson, Ph.D.). These patients were selected from the western and San Fernando Valley regions of the Los Angeles metropolitan region during an index hospitalization at one of the following public hospitals: UCLA Neuropsychiatric Hospital, Camarillo State Hospital, Harbor/UCLA Medical Center, and Olive View Medical Center. These subjects were required to have the following characteristics: (1) a diagnosis of schizophrenia or schizoaffective disorder, mainly schizophrenic, by Research Diagnostic Criteria (Spitzer, Endicott, and Robins 1978), based on an expanded version of the Present State Examination administered to the patient and consideration of any additional data gathered from relatives; (2) a recent onset of major psychosis lasting at least 2 weeks (first episode not longer than 2 years before project contact); (3) age between 16 and 45 years; and (4) Caucasian or acculturated Asian or Hispanic in ethnic background. Subjects with any of the following were excluded: (1) evidence of an organic central nervous system disorder (e.g., epilepsy, traumatic brain injury, infectious or toxic cerebrovascular disease); (2) significant and habitual drug or alcohol abuse in the 6 months preceding the current episode, or past drug or alcohol abuse that clouded the diagnostic picture; or (3) mental retardation (premorbid IQ < 70).

This schizophrenic sample was young, with an average age of 22.3 years (SD = 3.5; range = 18-32 years). The mean educational level was 12.3 years (SD = 2.1, range = 5-16 years). Ninety-five percent of the patients were Caucasian and 5 percent, acculturated Hispanic. Eighty-five percent of these patients were male.

Test Instruments. Two versions of the CPT, a forced-choice span of apprehension task, and the Rorschach were administered to each patient. Ratings of symptomatology were obtained on the BPRS.

The adult version of degraded-stimulus CPT (Nuechterlein, Parasuraman, and Jiang 1983) and a numeral version of the conventional A-X CPT (Rosvold et al. 1956) were used. The processing load of the degraded-stimulus CPT involves discrimination of the highly blurred numeric stimuli, whereas that of the conventional sequential-target CPT derives from the short-term-memory burden associated with detection of the stimulus sequences that comprise the target. Both were administered using an automated system in which a Kodak Ektagraphic E-2 Carousel Slide Projector and Ilex No. 4 Synchro-Electronic Shutter, under the control of a microcomputer, presented numeric stimuli on a 12 × 12 inch (30.5 × 30.5 cm) Carroll rear projection screen. Single digits "0" through "9" were presented one at a time with a duration of 40 ms and an interstimulus interval of 1 second. The projected digits were 6.3 cm high and 4.8 cm wide when clearly focused. The subject, with eyes 1 meter from the screen, was told to monitor the rapid series of numerals and to respond as quickly as possible by pressing a hand-held button to each target stimulus. Any subject whose eyes were directed away from the screen long enough to miss a stimulus was immediately reminded to focus on the numeric stimuli until the task was completed.

In the degraded-stimulus CPT, the numerals were blurred to an extent that required a 2.8 diopter lens correction to refocus the image at the same screen distance.[1] The degradation level also requires the use of the fully open setting (40 mm) of the shutter. (The authors thank Enoch Callaway, M.D., for pointing out the relevance of aperture size.) A visual mask, which consisted of typed + characters on a transparency, was mounted on the back of the screen to decrease figure-ground contrast and visual persistence. The subject was told to respond to each "0," which occurred in a quasi-random 20 of the 81 positions in the slide carousel. Identical digits never followed one another, and nontarget digits preceding target numerals were balanced to eliminate recognition of stimulus sequences. Subjects were instructed that it was equally important to press to targets and to avoid pressing to nontargets. After two carousel revolutions for practice, six continuous carousel revolutions were administered, including 120 targets and 366 nontargets, over a 8.1-minute period.

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[1] The diopter correction value was derived through direct measurement, using an optical lens placed immediately in front of the lens barrel. Values derived from direct measurement were found to be somewhat higher than the values from the equation reported by Nuechterlein, Parasuraman, and Jiang (1983), which uses screen-to-lens and slide-to-lens distances. Direct measurement of the degree of blurring with optical lenses is recommended to avoid the need to estimate placement of the projector lens within the lens barrel.

In the numeric adaptation of the A-X CPT, the subject was required to respond to each successive sequence of a "3" followed by a "7" in a quasi-random series of clearly focused single digits. This "3-7" sequence appeared 10 times (20 slide positions) within an 81-slide carousel. Additional processing load was presented in the form of auditory distraction, using an audiotaped male voice reading rapidly the same quasi-random series of digits, but lagged substantially in starting point, on a counterbalanced half of the carousel revolutions.[2] To increase difficulty level and alleviate the ceiling effects that typify the conventional CPT, 10 additional "3" stimuli and 10 additional "7" stimuli were included among the nontargets in the carousel in quasi-random order. To have sufficient target stimuli for signal-detection-theory analyses of changes over time within task in later analyses, the task was made somewhat longer than the degraded-stimulus CPT. After two practice carousel revolutions, eight continuous revolutions were administered, including 80 target sequences and 568 nontargets, over a 10.8-minute period. This 3-7 version of the CPT was not available for the first six inpatient assessments because it was developed after an easier conventional CPT with a single, clearly focused target digit was found to yield extreme ceiling effects.

The forced-choice span of apprehension was administered with the same apparatus, with the projector-to-screen distance adjusted to make the individual letters within arrays 1-inch (2.5-cm) high. Stimulus conditions replicated those of Asarnow and MacCrimmon (1978, 1981), except that exposure duration was 70 ms rather than 50 ms and that a small arrow appeared in the center of the screen from 1000 ms to 500 ms before each array to serve as a fixation point.[3] Each stimulus array contained the letter "T" or "F" in one of 16 locations produced by dividing the stimulus area into a 4 × 4 matrix. Either 0, 2, 4, or 9 nontarget letters were placed randomly in other positions of the matrix, with the constraint that no two letters in a given array be identical. Twenty stimuli were presented for each array size. Blocks of 10 for each array size were arranged in two counterbalanced orders. Order was alternated across subjects and was also alternated within each subject across two testing sessions. Equal numbers of the two targets appeared in each array size. The subject, seated with eyes 1 meter from the screen, was told that either a "T" or an "F" would be flashed on the screen, usually with other letters. The subject's task was to report verbally after each trial which of the two target letters was present, using the following rating scale to allow a later finer-grained analysis of performance: "certainly T," "probably T," "probably F," and "certainly F." After 20 practice trials, the 160 experimental trials were administered with approximately 3 seconds between trials. The experimenter did not initiate a trial unless the subject's eyes were directed to the screen. Testing time was approximately 20 minutes.

The Rorschach was administered by the procedure indicated in Johnston and Holzman (1979). The sessions were recorded on audiotape. The experimenter also recorded the responses in writing as the session proceeded. All audiotapes were carefully transcribed verbatim for scoring, using the handwritten record as a cross-check on accuracy.

An expanded version of the BPRS was used. Developed by David Lukoff, Ph.D., Keith Nuechterlein, Ph.D., and Joseph Ventura, M.A., of the UCLA Mental Health Clinical Research Center for the Study of Schizophrenia, this expanded BPRS includes structured questions for each of 25 items and behavioral anchors for the scale points "1" through "7" to improve applicability and reliability in outpatient assessments. Only the commonly employed 18 items (Guy 1976) were used for the present study. Interrater reliability coefficients of four raters with a criterion rater for 15 co-rated interviews had a mean of .85 across these scales, with a range from .60 to 1.00.

Test Sessions. The initial testing occasion with each patient occurred during the inpatient period as soon as diagnosis was assessed through the administration of the expanded Present State Examination. This occasion focused on the assessment of the patient during a psychotic episode and was therefore completed as soon as the patient was judged able to understand task instructions. The first inpatient occasion included the two versions of the CPT, the forced-choice span of apprehension the Rorschach, and the BPRS, as well as other psychophysiological and psychological assessments that are components of the larger longitudinal project. The first session included the two CPTs, the span of apprehension, and the BPRS, with the order of the two CPT versions and the span of apprehension counterbalanced across subjects and within subjects across sessions. The Rorschach was administered in a second session as soon as possible after this session, usually within 3 days. Thirty-six of 40 patients were on antipsychotic medication at this inpatient assessment, with mean chlorpromazine equivalent dosage of 645 mg (SD = 512; range = 143–1875 mg). Antiparkinsonian medications were withheld for 48 hours before testing whenever possible clinically (29 of 40 cases) to avoid possible associated anticholinergic effects on information processing.

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[2] A distractibility index was also derived from this 3-7 CPT for use in the larger longitudinal study, but was not included in data analyses for this report.

[3] The authors are very grateful to Dr. Robert F. Asarnow for his generous help in providing copies of slides and administration instructions for this forced-choice span of apprehension task.

The outpatient attentional assessment occurred after subjects had been clinically stabilized and after a standardized, injectable, maintenance dosage of fluphenazine (Prolixin) decanoate, 12.5 mg every 2 weeks, had been established for 1 month. This standardized medication is a component of the protocol of the outpatient longitudinal study. Three patients who showed intolerable side effects on 12.5 mg fluphenazine decanoate were placed on a lower fixed dosage (7.5 or 10 mg). Of the 40 schizophrenic patients who were assessed at the inpatient point, 32 were enrolled in the outpatient, standardized-medication, longitudinal study and were, therefore, assessed at the outpatient occasion. One of these subjects did not complete the 3–7 CPT or span of apprehension. Antiparkinsonian medications were again withheld 48 hours before testing whenever possible clinically (26 of 32 cases). This outpatient assessment occurred at a mean of 86 days subsequent to hospital discharge (SD = 54), as soon as clinical stabilization and standardized medication could be established. The two CPTs and the forced-choice span of apprehension administered at this assessment point were used in this report.

Results

Data Reduction. The signal detection indices, d′ and natural log of β (ln β), were derived from the hit rate and false alarm rate for each of the time periods of the two CPTs (Green and Swets 1966; Nuechterlein 1983). The degraded-stimulus CPT was divided into three time periods for purposes of analysis, each consisting of 40 targets and 122 nontargets presented over a 162-second period. To maintain this same number of targets in each time period, the 3–7 CPT was divided into two time periods, each consisting of 40 targets and 244 nontargets presented over a 324-second period. To allow the signal detection indices to be calculated for the occasional instances of perfect hit or false alarm rates during a time period, the hit rates were estimated as $2^{-1/t}$ and false alarms rates as $1 - 2^{-1/t}$, where t = the number of trials on which the relevant stimulus type (target or nontarget) occurred, as suggested by Davies and Parasuraman (1982). An overall hit rate, false alarm rate, d′ level, and ln β level were calculated by taking the mean of the time periods within each of these CPT conditions. For the degraded-stimulus CPT, which has been shown to elicit unusually rapid d′ decrements over time (Nuechterlein, Parasuraman, and Jiang 1983), the first time period was subtracted from the last to yield an index of d′ change over time. The means, standard deviations, and ranges for the CPT variables are listed in table 1 for the inpatient testing and table 2 for the outpatient testing.

The proportion of correct detections, P(C), for the forced-choice span of apprehension task was calculated for each array size. This variable is a criterion-free index of sensitivity analogous to the d′ index (Green and Swets 1966; McNicol 1972). The proportion of correct detections for the 10-letter arrays was used as the performance index for this report. The mean, standard deviation, and range for this variable are shown in tables 1 and 2 for the inpatient and outpatient testings, respectively.

The five BPRS factor scores (Guy 1976) were calculated from the 18 scales using the conventional unit-weighting procedures to yield Anxiety-Depression, Anergia, Thought Disturbance, Activation, and Hostile-Suspiciousness scores. Each sum of item scores was divided by the number of items composing the factor to make each factor score have a range from 1 to 7, identical to that of the individual items. A total BPRS score was created by summing the 18 individual item scores. Means, standard deviations, and ranges for these scores are presented in table 1.

The Rorschach was scored by the third author using the Thought Disorder Index (TDI) procedures of Johnston and Holzman (1979) as recently modified by Solovay et al. (submitted for publication). Interrater reliability between the third author and another trained scorer on the first 18 protocols was .95 for the

Table 1. Means, standard deviations, and ranges for key variables at the inpatient testing[1]

Variable	Mean	SD	Range
Degraded-stimulus continuous performance test (CPT)			
d′ level	2.11	.94	.40–4.03
ln β level	.82	.81	−.99–2.54
d′ change over time	−.61	.64	−3.05–.49
Hit rate	.67	.22	.18–.98
False alarm rate	.09	.10	.005–.54
3–7 CPT			
d′ level	3.58	.80	2.02–4.87
ln β level	2.52	.81	−.14–3.67
Hit rate	.80	.17	.41–1.00
False alarm rate	.007	.012	.00–.065
Forced-choice span of apprehension			
10-letter array P(C)	.82	.09	.63–.98
Brief Psychiatric Rating Scale (BPRS)			
Anxiety-Depression	2.47	1.03	1.00–6.00
Anergia	1.67	.76	1.00–3.50
Thought Disturbance	2.33	1.24	1.00–5.00
Activation	1.54	.73	1.00–4.33
Hostile-Suspiciousness	1.66	.80	1.00–3.67
BPRS Total (18 scales)	35.28	10.12	22–60
Rorschach			
Total Thought Disorder Index	23.24	25.35	.00–133.40
Total no. of responses	22.85	9.53	6–44

[1] $n = 40$, except for 3–7 CPT variables, for which $n = 34$.

total TDI, which indicates that a high level of agreement was achieved.

In addition to the total TDI score, scores on two sets of TDI factors were derived. For the primary analyses, five post hoc rational factors constructed by Holzman, Solovay, and Shenton (1985) to maximize the discrimination of schizophrenic and manic patients, as well as the conceptual relatedness of items within factors, were scored by a procedure comparable to that of Holzman, Solovay, and Shenton (1985). Square root transformation of the scores on the 22 categories of thought disorder was used to reduce the prominent skew of the distributions. Scores were then standardized to equate means and standard deviations. These square-root-transformed, standardized item scores were summed to achieve factor scores for the manic factors of Combinatory Thinking and Irrelevant Intrusions and for the schizophrenic factors of Fluid Thinking, Confused Thinking, and Peculiar Verbalizations.

For secondary analyses, empirical factors were also derived. To determine whether the current data set could be summarized well by the varimax factors that Holzman, Solovay, and Shenton (1985) derived through principal components analysis of 97 psychotic patients, we subjected the 22 square-root-transformed, standardized category scores for our 41 schizophrenic patients[4] to the same form of analysis using the Statistical Analysis System (SAS) FACTOR program (SAS Institute, Inc., 1982). This analysis would be expected to yield factors that are less generalizable than those derived by Holzman, Solovay, and Shenton (1985) due to the very lean subject-to-variable ratio, and would be unlikely to yield the manic factor, Expansive Thinking, because no manic patients were included. Nevertheless, it provides a useful summary of the most prominent dimensions in this TDI data set.

Three factors were found to account for 49 percent of the variance and were identified as an appropriate number for rotation by a scree plot. The varimax factors accounted for 27, 11, and 10 percent of the variance, respectively, and were named Associative Disorganization, Perceptual/Cognitive Fluidity, and Combinatory Cognition. Items loading above .50 on these factors are shown in table 3. Because each factor was well-defined, having at least five items with loadings above .50, and was reasonably coherent conceptually, these factors were used to summarize this data set in an efficient fashion for secondary analyses. Factor scores were compiled with standardized scoring coefficients to keep the three factor scores uncorrelated. On the basis of patterns found by Holzman, Solovay, and Shenton (1985), the first two of these three factors would

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[4] One additional schizophrenic subject who had Rorschach data but not inpatient CPT and span of apprehension data was included in this principal components analysis.

Table 2. Means, standard deviations, and ranges for key variables at the outpatient testing[1]

Variable	Mean	SD	Range
Degraded-stimulus continuous performance test (CPT)			
d′ level	2.24	1.08	.30–4.65
ln β level	.86	.83	−.55–2.39
d′ change over time	−.34	.49	−1.30–.74
Hit rate	.68	.25	.11–1.00
False alarm rate	.08	.08	.00–.38
3-7 CPT			
d′ level	3.80	.74	2.28–4.87
ln β level	2.36	.84	.04–3.77
Hit rate	.85	.16	.33–1.00
False alarm rate	.007	.010	.00–.048
Forced-choice span of apprehension			
10-letter array P(C)	.84	.08	.70–.95

[1] n = 32 for degraded-stimulus CPT; n = 31 for 3–7 CPT and span of apprehension.

Table 3. TDI item loadings on factors from varimax rotation of PCA[1]

Factor 1: Associative Disorganization	
Incoherent	.92
Relationship verbalization	.79
Perseveration	.63
Vagueness	.60
Queer	.60
Confusion	.56
Looseness	.55
Absurd	.55
Factor 2: Perceptual/ Cognitive Fluidity	
Fluidity	.73
Autistic logic	.71
Peculiar	.63
Word-finding difficulty	.60
Confusion	.58
Factor 3: Combinatory Cognition	
Inappropriate distance (excluding flippant)	.74
Confabulation (excluding playful confabulation)	.70
Incongruous combination	.65
Idiosyncratic symbolism	.62
Contamination	.58

[1] TDI = Thought Disorder Index; PCA = principal components analysis; all loadings > .50 are listed.

be expected to reflect schizophrenic thought patterns and were hypothesized to be related to CPT and span of apprehension performance.

The fact that these three varimax factors differ rather substantially in item composition from those of Holzman, Solovay, and Shenton (1985) may reflect the smaller subject-to-variable ratio or the more homogeneous subject diagnostic composition (no affective psychoses) of the present analysis. Another probable influential contributor to such analyses is the presence of a few severely thought-disordered subjects whose particular combination of high TDI categories is unduly weighted in factor analyses, despite the attempt to reduce such influence by square root or other similar transformations. Because the empirical factors have these limitations, the combined use of Holzman, Solovay, and Shenton's (1985) post hoc rational factors and the present empirical factors was judged the best way to represent the major TDI dimensions for this study.

Relationship of CPT and Span of Apprehension Performance to TDI Scores. To examine the predicted relationship of the CPT d′ levels and the span of apprehension 10-letter array P(C) to the schizophrenic TDI factors of Fluid Thinking, Confused Thinking, and Peculiar Verbalizations, Pearson product-moment correlations were calculated. During the inpatient period, none of these nine correlations were statistically significant, suggesting an absence of concurrent relationship between these hypothesized vulnerability indicators for schizophrenic disorder and the severity of formal thought disorder during the inpatient psychotic period. A parallel set of correlations used the outpatient CPT and span of apprehension performance, which should represent a more stable, residual level of the CPT and span of apprehension vulnerability factors. Low outpatient degraded-stimulus CPT d′ level was related, as hypothesized, to high inpatient Fluid Thinking ($r = -.34$, $p < .05$, one-tailed) and showed a nonsignificant tendency to be related to high Confused Thinking ($r = -.28$, $p < .10$, one-tailed). This predicted relationship of the outpatient degraded-stimulus CPT performance to inpatient formal thought disorder was also evident in the correlation of d′ and the total TDI ($r = -.34$, $p < .05$, one-tailed). As predicted, none of the correlations between the CPT and span of apprehension indices and manic TDI factor scores were significant.

Table 4. Correlations of signal detection indices from inpatient CPTs and span of apprehension task with BPRS factor scores during inpatient period [1]

	BPRS factor				
Task	Anxiety-Depression	Anergia	Thought Disturbance	Activation	Hostile-Suspiciousness
Degraded-stimulus CPT					
d ′ level	– .24	– 36[3]	– .08	– .05	– .36[3]
3-7 CPT					
d ′ level	– .30[2]	– .38[3]	– .03	– .01	– .11
Forced-choice span of apprehension					
10-letter array					
P(C)	– .20	– .36[3]	.02	.04	– .06

[1] CPT = continuous performance test; BPRS = Brief Psychiatric Rating Scale; correlations are based on $n = 40$, except for those involving 3-7 CPT, for which $n = 34$.

[2] $p < .10$, 2-tailed.

[3] $p < .05$, 2-tailed.

Secondary analyses that used the TDI factor scores from the principal components analysis yielded a similar picture. None of the predicted concurrent relationships with the Associative Disorganization or Perceptual/Cognitive Fluidity factor were significant, although low inpatient degraded-stimulus CPT d ′ level showed a tendency to correlate with high inpatient Associative Disorganization ($r = -.25$, $p < .10$, one-tailed). This same relationship with high Associative Disorganization became slightly stronger and significant when the outpatient degraded-stimulus CPT d ′ level was examined ($r = -.30$, $p < .05$, one-tailed). Correlations of the 3-7 CPT d ′ and span of apprehension P(C) with these TDI factors were not significant.

In light of the fact that the significant correlations with inpatient schizophrenic TDI factors were restricted to the degraded-stimulus CPT performance during the outpatient period, the results provide only limited support for the hypothesis that the CPT and span of apprehension vulnerability indicators are directly related to schizophrenic formal thought disorder during psychotic periods. To the extent that relationships were found, the degraded-stimulus CPT tended to be associated with schizophrenic rather than manic TDI factors.

Relationship of Inpatient CPT and Span of Apprehension Performance to Inpatient BPRS Thought Disturbance and Anergia Factors. Pearson product-moment correlations were used to examine the concurrent BPRS ratings to test the hypotheses that low CPT d ′ level and span of apprehension P(C) level would be related to higher clinical ratings of positive schizophrenic symptoms (BPRS Thought Disturbance) and negative schizophrenic symptoms (BPRS Anergia). The d ′ change over time with degraded-stimulus CPT and the CPT response criterion levels (ln β) were expected to be uncorrelated with these BPRS factors, because they have not been found to be deviant in schizophrenic patients or persons at risk for schizophrenia. Correlations with other BPRS factor scores will be presented as evidence for discriminative validity. Because these other scales have less relevance to core positive and negative symptoms of schizophrenia, they were expected to be unrelated with the CPT and span of apprehension performance. In light of the number of BPRS factor scores and individual scales that needed to be examined, correlations were evaluated with two-tailed tests of significance despite this pattern of hypothesized relationships.

As shown in table 4, the hypothesis that concurrent low CPT d ′ and span of apprehension P(C) would be related to high BPRS Anergia ratings was supported for each of the three laboratory signal-discrimination measures. The degraded-stimulus CPT d ′ level, the 3-7 CPT d ′ level, and the span of apprehension P(C) were correlated $-.36$, $-.38$, and $-.36$, respectively, with concurrent BPRS Anergia ratings. Correlations of these three laboratory signal-discrimination indices with the BPRS Thought Disturbance scores were, contrary to our hypothesis, near zero. The expected lack of significant relationship of the CPT and span of apprehension performance with the other BPRS factors was observed, with minor exceptions.

Secondary analyses to determine the separate contributions of CPT hits and false alarms to the significant d ′ level correlations indicated that high Anergia ratings were associated with low hit rate for the degraded-stimulus CPT and 3-7 CPT and also high false alarm rate for the latter (r's $= -.39$, $-.34$, and $.34$, all $p < .05$). Correlations of the

Table 5. Correlations of signal detection indices from inpatient CPTs and span of apprehension task with individual scales of the BPRS Anergia factor during inpatient period[1]

	BPRS subscale			
Task	Emotional Withdrawal	Motor Retardation	Blunted Affect	Disorientation
Degraded-stimulus CPT				
d′ level	−.15	−.38[3]	−.33[3]	−.19
3-7 CPT				
d′ level	−.30[2]	−.33[2]	−.32[2]	−.26
Forced-choice span of apprehension				
10-letter array P(C)	−.08	−.40[3]	−.38[3]	−.19

[1] CPT = continuous performance test; BPRS = Brief Psychiatric Rating Scale; correlations are based on n = 40, except for those involving 3-7 CPT, for which n = 34.

[2] $p < .10$, 2-tailed.

[3] $p < .05$, 2-tailed.

BPRS factor scores with response criterion levels were, as expected, not significant. A significant relationship was found between the Anergia factor and d′ change over time in the degraded-stimulus CPT (r = .35, $p < .05$). This correlation suggests that high Anergia scores were related to relatively uniform d′ across time periods, as opposed to the substantial d′ decrements over time that are indicated by large negative numbers for this d′ change index. Although initially this association seems counterintuitive, it becomes sensible when the relationship between d′ level and d′ change over time in the degraded-stimulus CPT in this schizophrenic sample is considered ($r = -.44$, $p < .01$). Schizophrenic patients with high overall d′ levels had large d′ decrements over time, whereas patients with low overall d′ levels showed uniformly low d′ levels across time periods.

To examine further the specific nature of the symptoms that contribute to the relationship between CPT and span of apprehension performance and the BPRS Anergia factor, correlations with the individual scales of this factor were calculated. As shown in table 5, low d′ on the degraded-stimulus CPT and low P(C) on the span of apprehension were related to significantly higher BPRS Blunted Affect and Motor Retardation ratings. Low 3-7 CPT d′ showed nonsignificant tendencies to be related to higher BPRS Emotional Withdrawal, Blunted Affect, and Motor Retardation ratings that were similar in magnitude but based on somewhat fewer subjects than the correlations with the degraded-stimulus CPT and span of apprehension.

To ensure that the lack of relationship of the information-processing indices with the BPRS Thought Disturbance factor did not obscure an association with one of the BPRS component scales, particularly Conceptual Disorganization, correlations with the four individual scales (Conceptual Disorganization, Hallucinations, Unusual Thought Content, and Grandiosity) were examined. None of these BPRS individual scale correlations with the two CPT d′ levels or span of apprehension P(C) were significant. The correlations with Conceptual Disorganization were consistently in the predicted direction (−.25, −.29, and −.21 for the degraded-stimulus CPT d′, 3-7 CPT d′, and span of apprehension P(C), respectively), but not statistically significant for this sample size.

In summary, these analyses of BPRS data indicate that the signal-discrimination indices from the CPT and span of apprehension were significantly related to clinically assessed, concurrent negative symptoms of schizophrenia, but were not significantly associated with positive psychotic symptoms. The lack of a significant concurrent relationship with positive symptoms is consistent with the general absence of a concurrent inpatient association of the CPT and span of apprehension performance with the key TDI variables, as presented in the last section.

Relationship of Outpatient CPT and Span of Apprehension Performance to Inpatient BPRS Thought Disturbance and Anergia Factors. To determine whether the CPT and span of apprehension performance in the stabilized outpatient period showed a similar pattern in relation to the clinically rated negative and positive schizophrenic symptomatology during the inpatient period, a parallel set of analyses was completed. Again, the relationships with the Anxiety-Depression, Activation, and Hostile-Suspiciousness factors were predicted to be nonsignificant due to the lesser relevance of these factors to core positive and negative schizophrenic symptoms.

Table 6 presents the correlations

with the BPRS factor scores. Significant relationships between low outpatient signal discrimination and high inpatient BPRS Anergia factor scores were evident for two of the three hypothesized vulnerability indices from these tasks, in this case for the two CPT d′ level indices but not for the span of apprehension P(C). Correlations of the CPT d′ levels and span of apprehension P(C) with the BPRS Thought Disturbance factor scores were again not significant. As expected, correlations with other BPRS factors were not significant, although it might be noteworthy that the tendency for low outpatient degraded-stimulus CPT d′ levels to be associated with higher inpatient Hostile-Suspiciousness scores is consistent with the parallel significant association of this BPRS factor with the inpatient degraded-stimulus CPT d′ level.

Correlations of the hypothesized vulnerability indicators with component scales of the Anergia factor are shown in table 7. The outpatient CPT d′ levels and span of apprehension P(C) show generalized nonsignificant tendencies to be negatively related to the Anergia component scales. Significant associations are in this case present between low degraded-stimulus CPT d′ level and high inpatient Emotional Withdrawal and Motor Retardation ratings and between low 3–7 CPT d′ level and high inpatient Disorientation ratings.

Examination of the correlations of the three CPT and span of apprehension indices with the component scales of the BPRS Thought Disturbance factor was again used to ensure that specific relationships, particularly with Conceptual Disorganization, were not obscured by the overall factor score results. Correlations of inpatient Conceptual Disorganization ratings with the three outpatient information-processing performance indices were all in the expected direction and were significant ($p < .05$) for the 3–7 CPT d′ level ($r = -.44$) and span of apprehension P(C) ($r = -.38$). On the

Table 6. Correlations of signal detection indices from outpatient CPTs and span of apprehension task with inpatient BPRS factor scores [1]

Task	BPRS factor				
	Anxiety-Depression	Anergia	Thought Disturbance	Activation	Hostile-Suspiciousness
Degraded-stimulus CPT					
d′ level	−.10	−.44[3]	−.05	−.02	−.32[2]
3-7 CPT					
d′ level	−.08	−.36[3]	−.05	.05	−.31[2]
Forced-choice span of apprehension					
10-letter array P(C)	−.13	−.19	−.16	.24	−.24

[1] CPT = continuous performance test; BPRS = Brief Psychiatric Rating Scale; based on $n = 32$ for correlations involving the degraded-stimulus CPT and $n = 31$ for those involving 3-7 CPT or span of apprehension.
[2] $p < .10$, 2-tailed.
[3] $p < .05$, 2-tailed.

Table 7. Correlations of signal detection indices from outpatient CPTs and span of apprehension task with individual scales of the BPRS Anergia factor rated during inpatient period [1]

Task	BPRS subscale			
	Emotional Withdrawal	Motor Retardation	Blunted Affect	Disorientation
Degraded-stimulus CPT				
d′ level	−.50[4]	−.45[4]	−.21	−.20
3-7 CPT				
d′ level	−30[2]	−.27	−.13	−.41[3]
Forced choice span of apprehension				
10-letter array P(C)	.06	−.21	−.18	−.33[2]

[1] CPT = continuous performance test; BPRS = Brief Psychiatric Rating Scale; based on $n = 32$ for correlations involving degraded-stimulus CPT and $n = 31$ for those involving the 3-7 CPT or span of apprehension.
[2] $p < .10$, 2-tailed.
[3] $p < .05$, 2-tailed.
[4] $p < .01$, 2-tailed.

other hand, correlations with Unusual Thought Content, Hallucinations, and Grandiosity hovered around zero.

In summary, these results relating the outpatient CPT and span of apprehension signal-discrimination indices to the inpatient BPRS ratings are quite consistent with those that involved the inpatient signal-discrimination levels. The outpatient signal-discrimination vulnerability indicators were more strongly related to the inpatient negative symptoms of schizophrenia than to the positive symptoms. The only significant correlations of the outpatient signal-discrimination indices with inpatient positive symptoms were with Conceptual Disorganization, the Thought Disturbance component scale with which the closest association was expected.

Discussion

Our results indicate that signal-discrimination deficits on the degraded-stimulus CPT, the 3-7 CPT, and the forced-choice span of apprehension are quite consistently related to the presence of negative symptoms (BPRS Anergia factor scores) among young patients with a recent onset of schizophrenic disorder, as evidenced by significant correlations in five of six evaluated relationships. The relationship of these attentional deficits to formal thought disorder (BPRS Conceptual Disorganization ratings and Rorschach Thought Disorder Index factors) among these recent-onset patients appears to be less consistent, although the hypothesized correlations do achieve statistical significance somewhat more frequently than expected by chance. When significant relationships were observed between the signal-discrimination deficits and the Rorschach TDI factors, they involved, as hypothesized, modes of formal thought disorder that are more typically schizophrenic than manic in nature. Relationships between signal/noise discrimination impairment on the attentional tasks and the presence of delusions and hallucinations were never significant and hovered around zero.

Impaired signal discrimination on the CPT and span of apprehension tasks is associated with schizophrenic negative symptoms in a fashion that is consistent with the hypothesized role of these attentional deficits as vulnerability factors for these clinical symptoms. The relationship of the signal-discrimination deficits on the three attentional tasks to the inpatient negative symptoms (BPRS Anergia factor scores) was present whether the signal discrimination was measured during the inpatient period or during a stabilized outpatient period. Furthermore, the magnitude of the relationship did not change across these two attentional assessment points for two of three measures. Thus, the relationship is not limited to a cross-sectional association within episodes, which would be expected if the signal-discrimination impairment was secondary to clinical symptomatology.

The correlations of the outpatient signal-discrimination deficits with inpatient negative symptoms are, furthermore, not simply due to the continuation of a cross-sectional association between clinical symptomatology and these laboratory-measured deficits. As might be expected, the range of the BPRS Anergia factor score was reduced at the stabilized outpatient point (from 1.00–3.50 at the inpatient point to 1.00–2.50 at the outpatient point, SDs = .76 and .46, respectively). The cross-sectional correlations with the three signal-discrimination indices at the outpatient assessment were not significant (r's = − .12, .18, and − .15 for the degraded-stimulus CPT, 3-7 CPT, and span of apprehension, respectively). Yet the signal-discrimination deficits were moderately stable over time in these recent-onset schizophrenic patients, despite the large changes in both clinical state and medication across these two assessment points (r's = .49, .64, and .43 for the degraded-stimulus CPT, 3–7 CPT, and span of apprehension, respectively). Furthermore, as noted earlier, deficits on these attentional tasks also characterize children at risk for schizophrenia who have not experienced a schizophrenic episode (Asarnow et al. 1977; Rutschmann, Cornblatt, and Erlenmeyer-Kimling 1977; Nuechterlein et al. 1982; Nuechterlein 1983) as well as schizophrenic patients in psychotic remission after an episode (Wohlberg and Kornetsky 1973; Asarnow and MacCrimmon 1978). Taken together, this pattern of relationships is consistent with the view that ongoing levels of signal-discrimination impairments on these tasks are associated with schizophrenic negative symptoms during episodes.

An important additional relationship that is being investigated in our current longitudinal study of recent-onset schizophrenic patients is whether ongoing signal-discrimination deficits are predictive of the return of positive symptoms or of the development of later more persistent negative symptoms after the resolution of the index psychotic episode. Schizophrenic negative symptoms have been seen as more strongly associated with poor prognosis than are positive symptoms (Strauss, Carpenter, and

Bartko 1974; Crow 1980), a view supported by early data regarding the prognostic value of restricted affect (Carpenter et al. 1978). Furthermore, negative symptoms appear to be less responsive to neuroleptic treatment than do positive symptoms (Johnstone et al. 1978; Angrist, Rotrosen, and Gershon 1980). The association in the current study between the signal-discrimination deficits and negative symptoms, therefore, serves to bolster the possible utility of these attentional measures as prognostic indicators, a role that has been suggested in studies of simple reaction time (Cancro et al. 1971; Zahn and Carpenter 1978) and the forced-choice span of apprehension (Marder, Asarnow, and Van Putten 1984) among schizophrenic patients and in a study of a composite attentional index among offspring of a schizophrenic parent (Cornblatt and Erlenmeyer-Kimling 1985). Given the current results, it might be fruitful in further evaluations of these prognostic relationships to differentiate the prognostic value of such attentional measures for negative symptoms as opposed to florid positive symptoms of schizophrenia.

The closer association of the CPT and forced-choice span of apprehension deficits with negative, as opposed to positive, symptoms of schizophrenia within this schizophrenic sample might also be meaningfully connected to the recent finding that concordance rates for schizophrenia for monozygotic twins tend to be higher when probands have a larger number of negative symptoms (Dworkin and Lenzenweger 1984). Within schizophrenic samples, poor CPT performance has been found to be associated with a family history of schizophrenia (Walker and Shaye 1982) or severe mental illness (Orzack and Kornetsky 1971). This combination of findings suggests the possibility that poor CPT performance is likely to characterize a subtype of schizophrenia with prominent negative symptoms that has a particularly strong genetic component.

The relationship of the three signal-discrimination measures to negative symptoms seems to cut across the individual BPRS scales of the Anergia factor, with fluctuations in the level of the correlations across scales and occasions possibly being due to chance rather than systematic differences. Blunted Affect and Motor Retardation showed statistically significant relationships with signal-discrimination deficits in concurrent inpatient assessments, whereas inpatient Emotional Withdrawal, Motor Retardation, and Disorientation showed significant associations with an outpatient index of signal-discrimination deficit. The Anergia factor score appears to show more consistent relationships to the signal-discrimination indices than the individual component scales by providing a summary of the individual scale tendencies. Additional examination of the relationship of specific schizophrenic negative symptoms to these signal-discrimination deficits will be undertaken with data from subscales of the Schedule for Assessment of Negative Symptoms (Andreasen 1982) that are currently being collected, because this instrument, unlike the BPRS, was designed specifically for assessment of the full range of such symptoms.

The fact that the relationships with negative symptoms generalized across the signal-discrimination measures from the two CPTs and the forced-choice span of apprehension is consistent with the possibility discussed elsewhere (Nuechterlein and Dawson 1984) that a substantial portion of the schizophrenia-relevant variance in these tasks is associated with a shared high momentary demand on processing capacity. Although the tasks differ as to whether visual target and nontarget stimuli are presented simultaneously or sequentially, accurate signal discrimination in the two versions of the CPT employed here, as well as in the 10-letter array of the forced-choice span of apprehension, demands substantial effortful, capacity-demanding processing (Kahneman 1973; Hasher and Zacks 1979). The stimulus-input loads are high and the conditions of these tasks do not facilitate the development of automatic processing (Schneider and Shiffrin 1977; Treisman and Gelade 1980). Another compatible and related interpretation of these deficits would emphasize that slowed cognitive processing associated with negative symptoms contributes to deficits in these CPT and span of apprehension conditions (Green and Walker 1984).

A logical connection is apparent between deficits in active, effortful processing and the symptoms of affective flattening, emotional withdrawal, and motor retardation. The possible interrelationship of attentional and affective disturbances in schizophrenia was noted early by Bleuler (1911/1950):

> As a partial phenomenon of affectivity, attention is affected with it by deterioration. . . where affect is lacking, there will also be lacking the drive to pursue the external and internal processes, to direct the path of the senses and the thoughts; i.e., active attention will be lacking. [p. 68]

The current evidence supports the interrelationship of these domains. However, the outpatient data suggest that the deficit in active, effortful processing may remain as a vulnerability factor after any anergic symptoms become less apparent. Furthermore, given the data on the existence of such deficits in active, effortful processing in high-risk populations, it appears that these attentional deficits might be present before the clinical symptoms of affective flattening and emotional withdrawal become evident. Thus, the linkage of active, effortful processing to weakened affect, noted clinically by Bleuler, is clearly supported, but the deficit in effortful processing might serve as an enduring vulnerability factor, rather than a secondary reflection of anergic symptoms.

The relationship of these attentional vulnerability indicators to formal thought disorder was less consistent in the current study. To the extent that significant associations were found, the clinically stabilized, outpatient level of signal discrimination, specifically on the degraded-stimulus CPT, was related to TDI factor scores for Fluid Thinking (Holzman, Solovay, and Shenton 1985) and Associative Disorganization during the inpatient period. No significant relationships between concurrent inpatient assessment of these attentional deficits and formal thought disorder were found. This pattern is consistent with the view that the outpatient signal-discrimination deficit is a vulnerability factor for these modes of schizophrenic thinking, rather than a simultaneous acute effect of psychotic disorganization. The relationship of the signal-discrimination deficits to TDI factors that are typically schizophrenic, as opposed to manic, in nature should also be evaluated within a manic sample, to determine the generalizability of this finding. The significant correlations of the outpatient 3-7 CPT and span of apprehension indices with inpatient Conceptual Disorganization ratings are also congruent with the interpretation that outpatient signal-discrimination deficit is a vulnerability factor for certain aspects of formal thought disorder. Thus, the CPT and span of apprehension signal-discrimination deficits do show some potential as vulnerability factors for thought disorder in schizophrenia, although this evidence is less consistent than that for an association with negative symptoms.

Additional investigation will be needed to determine whether the greater consistency of the association of these CPT and forced-choice span of apprehension measures with negative symptoms, as compared to formal thought disorder, generalizes to other forms of information-processing disturbance in schizophrenia. Certain other measures might be more strongly related to formal thought disorder or other positive symptoms than are the ones used in this study, but less clearly related to negative symptoms. Oltmanns, Ohayon, and Neale (1978), for example, found that a measure of auditory distractibility that is a promising vulnerability indicator among children of schizophrenic parents (Harvey et al. 1981) was strongly associated with formal thought disorder in schizophrenic inpatients. Green and Walker (in press) and Cornblatt et al. (1985) have recently found evidence that is also consistent with the connection of distractibility to positive symptoms, while a measure of performance under processing overload conditions in the latter study was associated with negative symptoms. These promising cross-sectional observations will be bolstered if longitudinal predictive relationships between a relatively enduring information-processing deficit and symptom development can be further established. If consistent divergence in the symptomatic correlates of these fundamental attentional and information-processing deficits can be isolated, the role of such potential vulnerability factors in the development of different schizophrenic symptom clusters may be greatly clarified.

References

Anderson, V.E.; Siegel, F.S.; Fisch, R.O.; and Wirt, R.D. Responses of phenylketonuric children on a continuous performance test. *Journal of Abnormal Psychology*, 74:358–362, 1969.

Andreasen, N.C. Negative symptoms in schizophrenia: Definition and reliability. *Archives of General Psychiatry*, 39:784–788, 1982.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia: Definition and validation. *Archives of General Psychiatry*, 39:789–794, 1982.

Angrist, B.; Rotrosen, J.; and Gershon, S. Positive and negative symptoms in schizophrenia—Differential response to amphetamine and neuroleptics. *Psychopharmacology*, 72:17–19, 1980.

Asarnow, R.F. Schizophrenia. In: Tarter, R.E., ed. *The Child at Psychiatric Risk*. New York: Oxford University Press, 1983. pp. 150–194.

Asarnow, R.F., and MacCrimmon, D.J. Residual performance deficit in clinically remitted schizophrenics: A marker of schizophrenia? *Journal of Abnormal Psychology*, 87:597–608, 1978.

Asarnow, R.F., and MacCrimmon, D.J. Span of apprehension deficits during postpsychotic stages of schizophrenia. *Archives of General Psychiatry,* 38:1006–1011, 1981.

Asarnow, R.F.; Nuechterlein, K.H.; and Marder, S.R. Span of apprehension performance, neuropsychological functioning, and indices of psychosis-proneness. *Journal of Nervous and Mental Disease,* 171:662–669, 1983.

Asarnow, R.F.; Steffy, R.A.; MacCrimmon, D.J.; and Cleghorn, J.M. An attentional assessment of foster children at risk for schizophrenia. *Journal of Abnormal Psychology,* 86:267–275, 1977.

Beatty, J. Task-evoked pupillary responses, processing load, and the structure of processing resources. *Psychological Bulletin,* 91:276–292, 1982.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias.* (1911) Translated by J. Zinkin. New York: International University Press, Inc., 1950.

Cancro, R.; Sutton, S.; Kerr, J.B.; and Sugarman, A.A. Reaction time and prognosis in acute schizophrenia. *Journal of Nervous and Mental Disease,* 153:351–359, 1971.

Carpenter, W.T., Jr.; Bartko, J.J.; Strauss, J.S.; and Hawk, A.B. Signs and symptoms as predictors of outcome: A report from the International Pilot Study of Schizophrenia. *American Journal of Psychiatry,* 135:940–944, 1978.

Cornblatt, B.A., and Erlenmeyer-Kimling, L. Global attentional deviance as a marker of risk for schizophrenia: Specificity and predictive validity. *Journal of Abnormal Psychology,* 94:470–486, 1985.

Cornblatt, B.A.; Lenzenweger, M.F.; Dworkin, R.H.; and Erlenmeyer-Kimling, L. Positive and negative schizophrenic symptoms, attention, and information processing. *Schizophrenia Bulletin,* 11:397–408, 1985.

Crow, T.J. Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal,* 280:66–68, 1980

Davies, D.R., and Parasuraman, R. *The Psychology of Vigilance.* London: Academic Press, Inc., 1982.

Dworkin, R.H., and Lenzenweger, M.F. Symptoms and the genetics of schizophrenia: Implications for diagnosis. *American Journal of Psychiatry,* 141:1541–1546, 1984.

Erlenmeyer-Kimling, L., and Cornblatt, B. Attentional measures in a study of children at high-risk for schizophrenia. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia:New Approaches to Research and Treatment.* New York: John Wiley & Sons, 1978. pp. 359–365.

Erlenmeyer-Kimling, L.; Cornblatt, B.; Friedman, D.; Marcuse, Y.; Rutschmann, J.; Simmens, S.; and Devi, S. Neurological, electrophysiological, and attentional deviations in children at risk for schizophrenia. In: Henn, F.A., and Nasrallah, H.A., eds. *Schizophrenia as a Brain Disease.* New York: Oxford University Press, 1982. pp. 61–98.

Erlenmeyer-Kimling, L.; Marcuse, Y.; Cornblatt, B.; Friedman, D.; Rainer, J.D.; and Rutschmann, J. The New York high-risk project. In: Watt, N.F.; Anthony, E.J.; Wynne, L.C.; and Rolf, J.E., eds. *Children at Risk for Schizophrenia: A Longitudinal Perspective.* New York: Cambridge University Press, 1984. pp. 169–189.

Estes, W.K., and Taylor, H.A. A detection method and probabilistic models for assessing information processing from brief visual displays. *Proceedings of the National Academy of Sciences of the United States of America,* 52:446–454, 1964.

George, L., and Neufeld, R.W.J. Cognition and symptomatology in schizophrenia. *Schizophrenia Bulletin,* 11:264–285, 1985.

Green, D.M., and Swets, J.A. *Signal Detection Theory and Psychophysics.* New York: John Wiley & Sons, Inc., 1966.

Green, M., and Walker, E. Susceptibility to backward masking in schizophrenic patients with positive or negative symptoms. *American Journal of Psychiatry,* 141:1273–1275, 1984.

Green, M., and Walker, E. Attentional performance in positive and negative symptom schizophrenia. *Journal of Nervous and Mental Disease,* in press.

Guy, W. *ECDEU Assessment Manual for Psychopharmacology.* DHEW Pub. No. (ADM) 76-338. Rockville, MD.: National Institute of Mental Health, 1976.

Harvey, P.; Winters, K.; Weintraub, S.; and Neale, J.M. Distractibility in children vulnerable to psychopathology. *Journal of Abnormal Psychology,* 90:298–304, 1981.

Hasher, L., and Zacks, R.T. Automatic and effortful processes in memory. *Journal of Experimental Psychology: General,* 108:356–388, 1979.

Holzman, P.S.; Solovay, M.R.; and Shenton, M.E. Thought disorder specificity in functional psychoses. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies.* New York: Guilford Press, 1985. pp. 228–245.

Hurt, S.W.; Holzman, P.S.; and Davis, J.M. Thought disorder: The measurement of its changes. *Archives of General Psychiatry,* 40:1281–1285, 1983.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking: A Clinical and Research Instrument for*

Measuring Thought Disorder. San Francisco: Jossey-Bass, 1979.

Johnstone, E.C.; Crow, T.J.; Frith, C.D.; Carney, M.W.P.; and Price, J.S. Mechanism of the antipsychotic effect in the treatment of acute schizophrenia. *Lancet*, I:848–851, 1978.

Kahneman, D. *Attention and Effort*. Englewood Cliffs, NJ: Prentice-Hall, Inc., 1973.

Kataguchi, Y. Rorschach schizophrenic score (RSS). *Journal of Projective Techniques*, 23:214–222, 1959.

Kraepelin, E. *Dementia Praecox and Paraphrenia*. (1913) Translated by R.M. Barclay. Edinburgh: E. & S. Livingstone, 1919.

Kornetsky, C., and Orzack, M.H. Physiological and behavioral correlates of attention dysfunction in schizophrenic patients. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment*. New York: John Wiley & Sons, 1978. pp. 196–204.

Maher, B.A. The language of schizophrenia: A review and interpretation. *British Journal of Psychiatry*, 120:3–17, 1972.

Marder, S.R.; Asarnow, R.F.; and Van Putten, T. Information processing and neuroleptic response in acute and stabilized schizophrenic patients. *Psychiatry Research*, 13:41–49, 1984.

McNicol, D. *A Primer of Signal Detection Theory*. London: George Allen & Unwin, 1972.

Neale, J.M. Perceptual span in schizophrenia. *Journal of Abnormal Psychology*, 77:196–204, 1971.

Neale, J.M.; McIntyre, C.W.; Fox, R.; and Cromwell, R.L. Span of apprehension in acute schizophrenics. *Journal of Abnormal Psychology*, 74:593–596, 1969.

Neale, J.M., and Oltmanns, T.F. *Schizophrenia*. New York: John Wiley & Sons, 1980.

Neale, J.M.; Oltmanns, T.F.; and Harvey, P.D. The need to relate cognitive deficits to specific behavioral referents of schizophrenia. *Schizophrenia Bulletin*, 11:286–291, 1985.

Nuechterlein, K.H. Signal detection in vigilance tasks and behavioral attributes among offspring of schizophrenic mothers and among hyperactive children. *Journal of Abnormal Psychology*, 92:4–28, 1983.

Nuechterlein, K.H. Converging evidence for vigilance deficit as a vulnerability indicator for schizophrenic disorders. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies*. New York: Guilford Press, 1985.

Nuechterlein, K.H., and Dawson, M.E. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10:160–203, 1984.

Nuechterlein, K.H.; Edell, W.S.; and West, A.P. "Signal Detection During Vigilance and Span of Apprehension in Nonpsychotic Schizotypal Individuals." Submitted for publication, 1985.

Nuechterlein, K.H.; Parasuraman, R.; and Jiang, Q. Visual sustained attention: Image degradation produces rapid sensitivity decrement over time. *Science*, 220:327–329, 1983.

Nuechterlein, K.H.; Phipps-Yonas, S.; Driscoll, R.M.; and Garmezy, N. The role of different components of attention in children vulnerable to schizophrenia. In: Goldstein, M.J., ed. *Preventive Intervention in Schizophrenia: Are We Ready?* DHHS Pub. No. (ADM) 82-111. Washington, DC: Superintendent of Documents, U.S. Government Printing Office, 1982. pp. 54–77.

O'Dougherty, M.; Nuechterlein, K.H.; and Drew, B. Hyperactive and hypoxic children: Signal detection, sustained attention, and behavior. *Journal of Abnormal Psychology*, 93:178–191, 1984.

Oltmanns, T.F.; Ohayon, J.; and Neale, J.M. The effect of antipsychotic medication and diagnostic criteria on distractibility in schizophrenia. *Journal of Psychiatric Research*, 14:81–91, 1978.

Orzack, M.H., and Kornetsky, C. Attention dysfunction in chronic schizophrenia. *Archives of General Psychiatry*, 14:323–326, 1966.

Orzack, M.H., and Kornetsky, C. Environmental and familial predictors of attention behavior in chronic schizophrenics. *Journal of Psychiatric Research*, 9:21–29, 1971.

Overall, J.E., and Gorham, D.R. The Brief Psychiatric Rating Scale. *Psychological Reports*, 10:799–812, 1962.

Posner, M.I. *Chronometric Explorations of Mind*. Hillsdale, NJ: Lawrence Erlbaum, 1978.

Powers, W.T., and Hamlin, R.M. Relationship between diagnostic category and deviant verbalizations on the Rorschach. *Journal of Consulting Psychology*, 19:120–124, 1955.

Quirk, D.A.; Quarrington, M.; Neiger, S.; and Slemon, A.G. The performance of acute psychotic patients on the index of pathological thinking and on selected signs of idiosyncracy on the Rorschach. *Journal of Projective Techniques*, 26:431–441, 1962.

Rapaport, D.; Gill, M.; and Schafer, R. *Diagnostic Psychological*

Testing. Vol. 2. Chicago: Yearbook Publishers, 1946.

Rosvold, H.E.; Mirsky, A.; Sarason, I.; Bransome, E.D., Jr.; and Beck, L.H. A continuous performance test of brain damage. *Journal of Consulting Psychology*, 20:343–350, 1956.

Rutschmann, J.; Cornblatt, B.; and Erlenmeyer-Kimling, L. Sustained attention in children at risk for schizophrenia: Report on a continuous performance test. *Archives of General Psychiatry*, 34:571–575, 1977.

SAS Institute, Inc. *SAS User's Guide: Statistics, 1982 Edition*. Cary, NC: SAS Institute, Inc., 1982.

Schneider, W., and Shiffrin, R.M. Controlled and automatic human information processing. I: Detection, search and attention. *Psychological Review*, 84:1–66, 1977.

Schwartz, S. Is there a schizophrenic language? *The Behavioral and Brain Sciences*, 5:579–626, 1982.

Solovay, M.R.; Shenton, M.E.; Holzman, P.S.; and Johnston, M.H. "Scoring Manual for the Thought Disorder Index: Revised Edition." Unpublished manuscript, 1985.

Solovay, M.R.; Shenton, M.E.; Johnston, M.H.; Gasperetti, C.; and Holzman, P.S. "The Thought Disorder Index revised." Submitted for publication.

Spitzer R.L.; Endicott, J.; and Robins, E. Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, 35:773–782, 1978.

Strauss, J.S.; Carpenter, W.T., Jr.; and Bartko, J.J. An approach to the diagnosis and understanding of schizophrenia: Part III. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin*, 1(Experimental Issue No. 11):61–69, 1974.

Treisman, A.M., and Gelade, G. A feature-integration theory of attention. *Cognitive Psychology*, 12:97–136, 1980.

Walker, E. Attentional and neuromotor functions of schizophrenics, schizoaffectives, and patients with other affective disorders. *Archives of General Psychiatry*, 38:1355–1358, 1981.

Walker, E., and Shaye, J. Familial schizophrenia: A predictor of neuromotor and attentional abnormalities in schizophrenia. *Archives of General Psychiatry*, 39:1153–1156, 1982.

Watkins, J.G., and Stauffacher, J.C. An index of pathological thinking in the Rorschach. *Journal of Projective Techniques*, 16:276–286, 1952.

Wohlberg, G.W., and Kornetsky, C. Sustained attention in remitted schizophrenics. *Archives of General Psychiatry*, 28:533–537, 1973.

Zahn, T.P., and Carpenter, W.T. Jr. Effects of short-term outcome and clinical improvement on reaction time in acute schizophrenia. *Journal of Psychiatric Research*, 14:59–68, 1978.

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Concurrent and Predictive Validity of Components of Disordered Thinking in Schizophrenia

by Raymond A. Knight, David S. Elliott, James D. Roff, and Charles G. Watson

Abstract

Although the various manifestations of disordered thought have played a central role in diagnostic and theoretical considerations of schizophrenia, such symptoms have not been found specific to schizophrenia, and at best have shown weak relations to outcome. As part of a 7-year followup study of a sample of young, acute, psychotic inpatients, we explored the hypothesis that despite these poor results there might be particular components of disordered thought that might have prognostic utility and be able to discriminate narrowly defined schizophrenics. Using extant symptom scales as our models, we constructed five internally consistent scales of disordered thought from behavioral ratings made using the Psychotic Inpatient Profile—Poverty of Speech, Attentional Impairment, Incoherence, Delusions, and Hallucinations. We tested the ability of these scales to predict various aspects of outcome adaptation including outcome diagnosis. In general, the Attentional Impairment scale demonstrated the best prognostic utility, predicting poorer outcome in several domains. Paradoxically, patients diagnosed by Research Diagnostic Criteria (RDC) as definite schizophrenic at outcome had been rated as showing less attentional impairment than probable and nonschizophrenics. An examination of the pattern of correlations between Attentional Impairment and performance on cognitive tasks suggested that attentional difficulties may be related to different underlying cognitive processes in those diagnosed schizophrenic and nonschizophrenic at outcome by RDC.

Numerous studies have found that symptoms of disordered thinking are neither diagnostically specific to schizophrenia nor useful in predicting outcome (e.g., Astrup and Noreik 1966; Jansson 1968; Strauss and Carpenter 1972, 1974; Hawk, Carpenter, and Strauss 1975; Pope and Lipinski 1978; Stephens 1978; Knight et al. 1979; Bland and Orn 1980; Knight and Roff 1985). Nevertheless, the most frequently used criteria for differentiating schizophrenia from other psychopathologies—for example, *DSM-III* (American Psychiatric Association 1980), the Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1978) and the St. Louis Criteria (Feighner et al. 1972)—either give substantial weight to various manifestations of disordered thinking or have focused exclusively on such symptoms. The poor diagnostic specificity and prognostic utility of these symptoms do not necessarily mean that disordered thinking is an unimportant construct in schizophrenia. Rather, recent evidence suggests that different kinds of disordered thought may vary in their diagnostic and predictive utility, and thus supports efforts aimed at subdividing this broad class into more homogeneous and potentially valid components. For instance, many of the studies that have failed to find thought symptoms to be effective discriminators or predictors have focused more on disorders of content (delusions and hallucinations) than on well-defined, specific disorders of form (e.g., Strauss and Carpenter 1974; Hawk, Carpenter, and Strauss 1975; Pope and Lipinski 1978; Bland and Orn 1980). Further study of the more "formal" aspects of disordered thought is required, despite the

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disagreements about how these should be defined (for examples of diverse criteria, see Spitzer, Endicott, and Robins 1978; Andreasen 1979*a*; Johnston and Holzman 1979). Indeed, there is evidence that some of these formal components have more diagnostic specificity than others (e.g., Andreasen 1979*b*; Holzman, Solovay, and Shenton 1985).

One aim of the present study is to examine the differential prognostic utility of several components of disordered thinking in a sample of young, acute psychotic psychiatric inpatients, who were part of a 7-year, prospective followup study. A second goal of this study is the exploration of the relation of these components of disordered thought to performance on two cognitive/perceptual tasks. There is suggestive evidence that some manifest symptoms of disordered thought may be the result of different underlying processes in patients with different disorders. One way to investigate the issue of underlying processes is to relate manifest symptoms to more basic cognitive and perceptual processes. Clearly, if different underlying processes can be identified, they might allow for better diagnostic discrimination and prediction than the manifest symptoms they produce.

Components of Disordered Thought

Recent attempts to partition the range of schizophrenic symptoms into positive and negative symptom clusters or syndromes have focused on the issues of diagnostic specificity, longitudinal stability, and process differentiation. They offer a potential solution to the disordered thought categorization problem. Theoretically, this dichotomy aims at distinguishing losses or diminutions of normal functioning (negative symptoms) from abnormalities or excesses of functioning (positive symptoms). Negative symptoms purportedly are more stable and have more severe prognostic implications than positive symptoms, are supposedly diagnostically more specific to schizophrenia, and hypothetically reflect the operation of distinct pathophysiological processes (Andreasen 1985; Crow 1985). Although some evidence has accrued supporting the viability of these hypotheses about this dichotomy (e.g., Crow 1985), the problems of both symptom operationalization and categorization mentioned above plague this research also. Investigators have not reached agreement on the operational definition of individual symptoms (Knight and Roff 1985; Lewine and Sommers 1985; Sommers 1985), and no consensus has emerged either theoretically (e.g., Andreasen 1985; Crow 1985; Lewine and Sommers 1985; Sommers 1985) or empirically (e.g., Lewine, Fogg, and Meltzer 1983; Bilder et al. 1985; Gibbons et al. 1985) on what symptoms should be considered positive or negative.

The positive-negative symptom classification problem is especially serious in the thought domain. Despite its problems, the dichotomy, because of both the rationale for its attempted discrimination and its preliminary support, provides a reasonable hypothetical structure for our inquiry about the components of disordered thinking. We were limited in our assessment of thought symptoms to the data already gathered at the outset of our study. Nevertheless, we were able to generate from behavioral ratings made during the initial assessment period using the Psychotic Inpatient Profile (PIP) three scales covering domains of disordered thought whose positive-negative statuses have achieved some consensus, and two scales covering domains whose statuses are controversial. In the former category are delusions, hallucinations, and poverty of speech, and in the latter category are incoherence and attentional impairment. These five scales provided an adequate sampling of components of disordered thought for studying the issues of prognosis and cognitive processes.

Most researchers agree that disorders in the content of thought (e.g., delusions and hallucinations) are positive symptoms. The PIP has a sufficient sampling of instances of both delusions and hallucinations to permit the construction of adequate scales assessing each. Poverty of speech is widely accepted as a negative symptom (e.g., Wing 1978; Pogue-Geile and Harrow 1984; Crow 1985), but there is some evidence that it is also prevalent among depressed patients (Andreasen 1979*b*; Oltmanns et al. 1985). We were able to construct a poverty of speech scale from the PIP whose items closely parallel those of Pogue-Geile and Harrow's (1984) scale.

Several items on the PIP allowed us to assess two symptom constructs whose positive-negative statuses are controversial: incoherence/tangentiality and attentional impairment. Lewine, Fogg, and Meltzer (1983), using a latent-trait model to cluster the symptoms of carefully diagnosed schizophrenics, found that incoherence loaded on their empirically derived negative symptom scale. Gibbons et al. (1985) found further that incoherent speech had a substantial loading on what they interpreted to be a Bleulerian "loss of goal" negative-symptom factor. Other investigators (e.g., Andreasen 1979*b*; Oltmanns et al. 1985) have found, however, that incoherence did not discriminate schizophrenics from manics, thus raising questions about its negative status.

Attentional impairment is included among negative symptoms only by those (e.g., Andreasen 1982) who equate the negative symptom syndrome with the Bleulerian fundamental symptoms. The preponderance of the data from studies testing schizophrenics' performance on attention-demanding tasks indicates that some kind of an attentional input dysfunction predates the onset of schizophrenia and is a consistent trait of a subsample of schizophrenics in both the acute and remitted stages of the disorder (Knight 1984; Nuechterlein and Dawson 1984). Such evidence of stability would seem to support its classification as a negative symptom. Two studies that have explored the relation of cognitive performance to symptomatology suggest, however, that a globally defined attentional impairment construct may be tapping aspects of both positive and negative syndromes. Bilder et al. (1985) found that Andreasen's Attentional Impairment scale had high loadings on two symptom factors. Its highest loading was on a factor defined by the "positive" symptoms of thought disorder and bizarre behavior. Of the three factors generated, this factor was the only one to have significant correlations with performance on tests in a neuropsychological battery. Attentional impairment loaded only secondarily on their negative symptom factor. Cornblatt et al. (1985) found that different kinds of problems in attentional performance were associated with different kinds of symptomatology. Measures of distractibility correlated with positive symptoms, but an assessment of resource overload correlated with negative symptoms. These two studies suggest that "attentional dysfunction" may be produced by multiple factors. Our Attentional Impairment scale, which closely approximates Andreasen's (1982) scale, allowed us to explore this further.

Underlying Processes

An important part of the search for distinctly different types of thought disorder is the evaluation of evidence suggesting that the same manifest behavior used to infer disordered thought may be produced by different underlying processes (Strauss, Carpenter, and Bartko 1974). When the same symptom is manifested in two disorders, it is often concluded that this is due to a shared common process. Thus, when manics and schizophrenics show the same thought symptom, this is frequently attributed to the general psychotic state that they hypothetically share. An alternative explanation, however, must be seriously considered—that cognitive processes unique to each disorder can result in similar symptoms. Such an alternative hypothesis would explain why many thought symptoms lack diagnostic specificity (e.g., Pope and Lipinski 1978; Andreasen 1979*b*; Oltmanns et al. 1985), whereas cognitive deficit studies are beginning to isolate cognitive/perceptual processes that appear singularly schizophrenic (cf. Knight 1984; Nuechterlein and Dawson 1984).

In our 30-year followup study of the child guidance records of preschizophrenics (cf. Knight and Roff 1983), we found evidence to support this alternative hypothesis. The same Thought Disorder Symptom scale was related to different prior and concurrent symptoms and measures at different times in the course of the disorder. A possible explanation for this cross-temporal change in the pattern of correlations focuses on the constitution of the sample, which was composed of broadly defined *DSM-II* (American Psychiatric Association 1968) "schizophrenics," only a portion of whom were "true" process schizophrenics from a longitudinal perspective. The less severely disordered, good prognosis paranoid and affective psychotics in the sample, whose young adult thought disorder symptoms were indistinguishable from their more seriously disordered counterparts, were asymptomatic at middle adulthood. Thus, their thought disorder symptoms influenced the correlations in young adulthood, but only the persisting symptoms of the process cases determined the pattern of the middle adult correlations. It seems reasonable to conclude from the differences both in childhood antecedents and in the course of the disorders to middle adulthood that the good prognosis, paranoid/affective psychotics, whose thought disorder symptoms abated, differed in underlying pathology from the process cases. Such a conclusion also suggests that the thought disorder symptoms in each group, although indistinguishable in young adulthood, might be the result of different underlying processes.

It could be argued that our Thought Disorder Symptom scale, which comprised hallucinations, delusions, bizarre or psychotic thinking, and confusion, was too general and that the support for multiple underlying processes rests on weak inferences. Moreover, although these results are not consistent with the shared psychotic state hypothesis, they do not disconfirm it. In the present followup study, we were able to test more directly the hypothesis that the changes in the pattern of correlations from young to middle adulthood were due to the abatement of good prognosis cases' symptoms and the relative persistence of process cases' symptoms through the followup period. To accomplish this goal in the present study, we used diagnosis at outcome to partition the sample into subgroups that differ in outcome symptomatology. For the entire sample and for these various diagnostic subgroups, we calculated correlations between the behaviorally assessed components of disordered thought that we derived from the PIP and performance on our two perceptual/cognitive tasks—choice reaction time and Shipley-Hartford Abstraction. We hypothesized that to the degree that the different performance measures tapped differences in underlying processes among particular patient groups, there should be changes in the patterns of correlations across outcome diagnostic specifications. These pattern changes should be most pronounced for Attentional Impairment, because there is independent evidence (Bilder et al. 1985; Cornblatt et al. 1985) that multiple causative factors may contribute to the behaviors used to infer attentional deficiencies. It would be difficult to attribute any correlational pattern differences among diagnostic groups to changes in psychotic state, because all groups shared this state when they were assessed on the components of disordered thought and the cognitive/perceptual tasks.

The questions addressed in the present study can be summarized as follows: (1) Are there discriminable components of disordered thought that vary in their prognostic utility? How do they relate to each other and to other selected concurrent measures? (2) Does the pattern of prognosis and relation to outcome diagnosis coincide with the current hypotheses about positive and negative symptoms? (3) Is there evidence, especially from the Attentional Impairment scale, that different underlying cognitive processes may produce similar manifest difficulties in patients with different disorders?

Methods

Design. The 75 subjects in this study were sampled from a larger group of psychiatric inpatients who were part of a 7-year followup study (cf. Knight and Roff 1985). This sample consisted of all those patients who during the followup period were either diagnosed schizophrenic by ward psychiatrists or met the criteria for schizophrenia in any one of the five diagnostic systems we applied: RDC, St. Louis criteria, New Haven Schizophrenia Index (NHSI) (Astrachan et al. 1972), Taylor-Abrams criteria (Taylor, Abrams, and Gaztanaga 1975), or the World Health Organization Flexible 6 (WHO-6) (Carpenter, Strauss, and Bartko 1973). These five diagnoses were made by two independent raters from our extensive followup records (cf. Marvinney, Rosenberg, and Knight 1982).

A number of behavioral, performance-based, self-report, interview, and medical-file-derived measures (described below) were gathered during the initial hospitalization between January 1970, and June 1971. Followup data were collected during 1978–79 by a thorough search of VA medical and claim files. Additional record sources were investigated, when hospitalizations outside of the VA system were indicated.

As the outcome diagnostic criteria used to partition our sample for the subgroup correlational analyses designed to explore our underlying processes hypothesis, we chose RDC for pragmatic reasons. The Taylor-Abrams criteria were too unreliable; and the NHSI and WHO-6 were too inclusive and exclusive, respectively, and thus did not yield sufficiently large subgroups for the correlational analyses we planned. The St. Louis criteria were eliminated because only a small number of schizophrenics rated definite by these criteria had been rated on the PIP. For the entire sample and for each of the RDC subgroups, we examined the concurrent and predictive validity of scales of disordered thought, rationally derived from the PIP. We correlated these scales with other selected, prospective measures and with measures of several domains assessed at outcome. We will discuss the procedures for each of these prospective and outcome domains in turn.

Subjects. We focused on the diagnostic distinctions made by the RDC for making comparisons within our sample. Patients classed as nonschizophrenic by our use of RDC met either the RDC for major affective or schizoaffective disorder or the *DSM-III* criteria for paranoid disorder. Table 1 presents the descriptive characteristics of the entire sample and separately for the RDC groups of nonschizophrenic, probable schizophrenic, and definite schizophrenic patients. The three RDC groups did not differ in age, Shipley-Hartford Vocabulary and Abstract scores, average Phillips (1953) ratings, or total number of months hospitalized before the initial assessment (all F's < 1). The groups did differ, however, in their number of hospitalizations before the initial assessment ($F = 3.82$; $df = 2, 58$; $p < .05$). The RDC probable schizophrenics had had fewer hospitalizations than both of the other groups ($p < .05$ by Newman-Keuls).

Prospective Measures

Psychotic Inpatient Profile. The Psychotic Inpatient Profile (PIP) is an inventory designed to quantify observable verbal and nonverbal behavior of psychiatric inpatients. It was developed from factor-analytic studies of extensive ward observations (Lorr and Vestre 1968, 1969). Of the 96 items, 74 describe behaviors that are rated as occurring "not at all," "occasionally," "fairly often," or "nearly always." Specific verbal statements, such as "says people or machines make him do or say things" (item 75), are assessed by 17 dichotomous, true-false items. Finally, five true-false items assess orientation.

Procedure. The PIP was filled out by a group of experienced nurses or psychiatric aides, chosen by the head nurse as competent, conscientious individuals who had ample opportunity to observe and talk to the patient. Many had previous experience with the PIP because a study that used it (Watson and Klett 1972) had just been completed when the ratings for the present study were initiated. The raters were given a list of PIP items, and asked to observe and talk to their assigned patient for 3 days. The standard rating forms were given to the raters after the observation period was completed. We familiarized the raters with the rating procedures and attempted to impress on them the importance of making accurate and independent judgments. We asked them not to fill out the form unless they had observed and interacted adequately with the patient. Pairs of raters observed individual patients during the same 3-day period, and ratings for each patient were completed within 2 weeks of admission.

Scale construction. We employed a combined rational/empirical approach to scale construction using the 96 PIP items. Our goal was to maximize internal consistency and to sample as many thought disorder domains as the content of the PIP permitted. For each of the five thought disorder categories that we judged could be adequately assessed—attentional impairment,

Table 1. Descriptive variables for 75 patients

	Entire sample ($n = 75$)	RDC non-sz[1] ($n = 35$)	RDC probable sz[1] ($n = 11$)	RDC definite sz[1] ($n = 26$)
Age (years)				
Mean	27.04 (75)[2]	27.24 (35)	27.19 (11)	26.40 (26)
SD	4.86	4.96	5.24	4.75
Shipley Verbal				
Mean	26.63 (68)	27.34 (32)	24.70 (10)	25.83 (23)
SD	5.42	5.01	4.37	6.07
Shipley Abstract				
Mean	21.04 (68)	22.03 (32)	21.20 (10)	18.96 (23)
SD	9.91	10.56	9.05	9.92
Phillips average				
Mean	14.54 (72)	15.50 (33)	13.95 (10)	13.67 (26)
SD	5.43	5.24	6.32	5.59
Months hospitalized before 1971				
Mean	7.96 (62)	8.92 (27)	5.33 (10)	8.29 (22)
SD	8.72	10.30	7.41	7.39
Number of hospitalizations before 1971				
Mean	3.19 (64)	3.38 (29)	1.60 (10)	3.59 (22)[3]
SD	2.37	2.58	1.71	2.06

[1] sz = schizophrenics.

[2] *n* is shown in parentheses.

[3] RDC probable schizophrenics had fewer hospitalizations than the other two RDC groups.

poverty of speech, incoherence, delusions, and hallucinations—we created scales by averaging the independent ratings of each item and adding these averaged scores. We also calculated Cronbach's α to determine the internal consistency of these scales. Because interrater reliabilities of the revised scales were lower than desired, we calculated the individual item reliabilities for each scale to determine whether the deletion of specific unreliable items in each item group could enhance interrater reliability without loss of internal consistency. This was possible for two scales (Delusions and Hallucinations). Because the enhancement of reliability resulting from the deletion of several items in the delusion and hallucination item groups limited the sampling of these positive symptom domains, we created an additional composite item group, called Delusions/Hallucinations, which comprised all possible relevant behaviors addressed by PIP items. This item group had reasonable internal consistency, but somewhat lower interrater reliability than either of its subcomponent scales.

The final scales and their component items are presented in table 2, along with their internal consistencies and the estimated interrater reliabilities of the averaged scores of the two raters (cf. Roff 1981). The internal consistencies of these scales, though moderate, indicate sufficient cohesion to warrant further analyses. The interrater reliabilities were adequate for Poverty of Speech, borderline for Delusions, Hallucinations, and the combined scale Delusions/Hallucinations, and low for Attentional Impairment and Incoherence. The low reliabilities for the last two variables indicate that any failure to find significant concurrent and predictive correlations with these variables must be interpreted cautiously.

A perusal of the content of these scales indicates close similarity to scales developed by other inves-

Table 2. Final items and reliabilities for the 6 Psychotic Inpatient Profile (PIP) thought disorder scales

Item group	PIP #	Items	Cronbach α	Interrater reliability of averaged scores
Attentional Impairment	24.	Drifting off subject	.60	.44
	55.	Answer unrelated		
	64.	Unable to follow instructions		
	88.	Reports cannot concentrate		
Poverty of Speech	32.	Repeats same words over and over	.70	.74
	35.	Does not start conversations		
	46.	Speaks slowly		
	56.	No back and forth conversation		
	68.	No answer to questions		
Incoherence	6.	Difficult to understand	.60	.48
	55.	Answer unrelated		
	60.	Talk not sensible		
Delusions	76.	Divine mission	.75	.68
	81.	Special knowledge/power		
	83.	Believes famous person		
Hallucinations	77.	Voices say "bad" things	.73	.65
	87.	Hears sounds others don't		
	89.	Voices command him		
	90.	Voices praise him		
Delusions/ Hallucinations		Delusions + Hallucinations (above) +	.80	.60
	75.	People/machines make him do things		
	79.	Voices threaten		
	85.	Too important to work		

tigators. The Attentional Impairment scale approximates the "interview inattentiveness" and the "complaints of inattentiveness" items that define Andreasen's (1982) Attentional Impairment scale. The Poverty of Speech scale closely parallels Pogue-Geile and Harrow's (1984) version of this scale. The Incoherence scale was so labeled because it is most heavily weighted with "incomprehensibility" items (cf. Andreasen 1979*a*). Because our scale lacks specificity about where in discourse incomprehensibility is produced (i.e., within or across sentences), it does not clearly differentiate incoherence from derailment and tangentiality as Andreasen's (1979*a*) scale does. Since incoherence, derailment, and tangentiality have all been found to be highly related (Andreasen 1979*b*), it seems reasonable to assume that this PIP Incoherence scale captures the common core of these three scales. The Delusions scale focuses on delusions of grandiosity. The Hallucinations scale measures auditory hallucinations.

Reaction Time Stimuli and Procedure. The stimuli for the reaction time task consisted of 11 Munsell color chips, necessitating that one of the initial subject selection criteria be the absence of color blindness. The standard color was a middle yellow (5 Y). The comparative colors were five "redder" (2.5 Y, 10 YR, 7.5 YR, 5 YR, and 2.5 YR) and five "greener" (7.5 Y, 10 Y, 2.5 GY, 5 GY, and 7.5 GY) colors that varied from the standard in just noticeable difference units (1–5 in each direction). All stimuli were presented with an Iconix 6135 tachistoscope. For each trial, the experimenter first indicated verbally that the stimulus for that trial was ready. The subject then depressed a foot pedal. After a 500 ms delay, the standard was presented for 1 second, followed immediately by 1 of the 10 comparative stimuli. This stimulus remained on until the subject depressed one of two response keys to indicate whether the stimulus was "redder" or "greener" than the standard color, or until 3 seconds had elapsed. Reaction time was measured as the duration from the onset of the comparative stimulus until one key was depressed. The 10 comparative colors were randomized in blocks of 10 for a total of 60 blocks, and 10 trials were given within each block. On 3 days within a span no longer than 10 days, each subject was given 200 trials per day. Three reaction time performance variables were calculated for each subject: overall mean reaction time, the mean of the variance within each block of 10 trials, and the mean number of incorrect responses within each block.

Medical-File Ratings. Using medical file abstracts created during the 1970–71 hospitalization, which included social histories, mental status exams, psychological testing, and summaries of the patients' ward behavior taken from nurses' ward notes, two clinical psychologists (R.K. and J.R.) independently made global judgments of the severity of disordered thought on a 5-point scale: not present (1) through severe, obvious, and persistent (5). These clinicians also rated the severity of affective deficit on a 5-category scale: normal or depressed/anxious (1); hostile, irritable, or affectively unstable (manic-depressive) (3); flat or inappropriate, and withdrawn (5). The average of the two clinicians' thought disorder judgments and their consensus judgment of affective deficit were used in all analyses. The estimated interrater reliability (Roff 1981) of the average rating of disordered thought was high ($r = .86$, $df = 59$, $p < .001$), but the estimated reliability of the consensus affectivity judgment was low ($r = .46$, $df = 60$, $p < .001$), because of the difficulty differentiating flat affect from depression (cf. Knight and Roff 1985).

Intelligence and Social Competence. Because both intellectual impairment and poor social competence have often been found characteristic of schizophrenics with negative symptoms (e.g., Crow 1980; Andreasen and Olsen 1982; Farmer, McGuffin, and Spitznagel 1983; Knight and Roff 1983; Levin et al. 1985), we included assessments of both among our concurrent measures. Our intelligence measure, the Shipley-Hartford Abstraction Test, was administered according to standard procedure (Shipley 1940). In a previous study (Knight, Epstein, and Zielony 1980), poor performance on this test correlated with both our medical-file judgment of disordered thought and high scores on the Whitaker Index of Schizophrenic Thinking. Premorbid social competence was assessed by the Phillips (1953) scale. All patients were rated on Farina's (Farina et al. 1962; Farina, Garmezy, and Barry 1963) adaptation of this scale on the basis of the subjects' responses both to DeWolfe's (1968) General Information Questionnaire and to our social competence interview. The interrater reliability of the scale was high ($r = .97$, $df = 59$, $p < .001$).

Outcome Measures

Followup Procedures. After a period of 7 years, we were able to obtain followup data for all 75 of the patients in the present sample from VA medical and claim files, and State hospital and criminal records.

Six research assistants were trained to make ratings on the extensive clinical file data that were gathered. They had access to and rated only information about the patients between 1972 and 1978.

The assistants were divided into two groups. The two assistants who had had the most extensive experience with clinical files were trained to make diagnostic and symptoms judgments and to rate global outcome. For each patient, they made independent diagnostic ratings of schizophrenia, affective disorder, and nonpsychotic using the five diagnostic criteria listed in the **Design** section. These two assistants also independently rated all subjects on the Symptom-Sign Inventory (SSI) (Gordon and Gregson 1970), on the same thought disorder and affect deficit judgments used by the clinicians in the prospective ratings, and on a 7-point Global Outcome scale. This outcome scale was a slight modification of a 6-point scale whose development and use we have described elsewhere (Knight et al. 1979; Roff, Knight, and Wertheim 1976). It ranges from Outcome 1, recovered, to Outcome 7, definite process schizophrenic, deteriorated or unimproved.

After completing their independent ratings, the two assistants resolved discrepancies with a consensus procedure. The reliabilities of their independent ratings are presented in table 3. Their consensus ratings were used in all subsequent analyses.

The other four assistants were trained to make social competence and work history judgments and to compile data on hospitalizations. A consensus procedure, like that of the diagnostic raters, was used for the social competence scales. It involved a comparison of two independent ratings and discussion of discrepant ratings to reach a mutually agreeable judgment. Items about which no agreement could be attained were coded as missing. The hospitalization and work ratings were made by two assistants. One assistant compared the two ratings, checked for errors, and rechecked the clinical file when discrepancies were found. Table 3 presents the rating scales and the estimated interrater reliabilities of the consensus ratings where appropriate.

Outcome Thought Disorder Scale Construction. Although there were no outcome scales that directly assessed positive and negative

Table 3. Descriptions and reliabilities of outcome measures

Variable	Description	Consensus interrater reliability
Consensus outcome	1. Recovered	.88
	2. Neurotic, characterological symptoms	
	3. Nonschizophrenic psychosis	
	4. Paranoid/hostile, questionable schizophrenia	
	5. Schizoid (flat with minimal thought disorder)	
	6. Process schizophrenia (with periods of remission)	
	7. Process schizophrenia (deteriorated)	
Symptom-Sign Inventory (Gordon & Gregson 1970)		.82
Thought disorder judgment	1. Not present, to	.92
	5. Severe, obvious, and persistent	
Affect deficit judgment	1. Normal or depressed/anxious	.83
	3. Hostile, irritable, affectively unstable	
	5. Flat, inappropriate, and withdrawn	
Hospitalization	*Number* of admissions between 1972 & 1978	
	Total cumulative *months* between 1972 & 1978	
Social relationships/ contacts	1. Sociable and outgoing	.74
	2. Good social contact	
	3. Some social contact	
	4. Avoided social contact	
Social relationships/ depth	1. Married or cohabiting	.92
	2. Some close friends	
	3. Some casual friends	
	4. No friends	
Steadiness of employment	1. Steady & progressive	
	2. Steady	
	3. Erratic	
	4. Nonexisting	

thought disorder symptoms, the diagnostic checklists and symptom rating sheets of the two diagnostic raters contained adequate information to allow the post hoc creation of several outcome thought disorder scales. We sorted selected thought disorder symptoms into three rational item groups: formal thought disorder, delusions of any type, and hallucinations of any type. The Formal Thought Disorder scale was composed of instances of formal thought disorder and thought symptoms that some investigators have considered to be negative symptoms. The latter two categories were considered to represent positive symptoms of disordered thought *content*. For each individual rater and for the consensus ratings, scale scores were obtained for each patient by summing the values (symptom present = 1; absent = 0) of each symptom on a scale and dividing this value by the number of valid, nonmissing items. Cronbach's α calculated on the consensus ratings of the two assistants and the interrater reliability calculated on the independent scale scores for each rater appear in table 4, along with the final list of thought disorder symptoms on each scale.

Results

Correlations Among the PIP Scales. Table 5 presents the correlations among the six PIP scales for the entire sample. With the exception of

Table 4. Thought disorder outcome scales

Item group	Symptom	Source	Cronbach α	Interrater reliability
Formal thought disorder			.72	.86
	Loose associations/illogicality	NHSI		
	Blocking	NHSI		
	Incoherent speech	WHO-6		
	Problems with concentration	St. Louis		
	Formal thought disorder	RDC		
Hallucinations			.84	.84
	Hallucinations (auditory)	NHSI		
	Hallucinations (visual)	NHSI		
	Hallucinations (other)	NHSI		
	Hallucinations, any type	RDC		
	Nonaffective verbal hallucinations	RDC		
	Voices commenting/conversing	RDC		
	Hears voices	SSI		
Delusions			.84	.90
	Delusions	NHSI		
	Thoughts aloud	WHO-6		
	Widespread delusions	WHO-6		
	Bizarre delusions	WHO-6		
	Nihilistic delusions	WHO-6		
	Thought broadcasting, insertion, or withdrawal	RDC		
	Delusions of control, multiple or bizarre delusions	RDC		
	Nonparanoid delusions	RDC		
	Thoughts read, actions influenced	SSI		
	Fears of being harmed	SSI		
	Fears of being plotted against	SSI		

Note.—NHSI = New Haven Schizophrenia Index; WHO-6 = World Health Organization Flexible 6; St. Louis = Feighner Criteria; RDC = Research Diagnostic Criteria; SSI = Symptom-Sign Inventory.

the Poverty of Speech scale, all the correlations among these scales were highly significant. Poverty of Speech did not correlate with either Attentional Impairment or with Delusions, and its correlations with Incoherence, Hallucinations, and Delusions/Hallucinations were the lowest of the significant correlations. This general pattern of correlations remained constant even when subgroups selected by the more stringent RDC inclusionary criteria for probable and definite schizophrenia were analyzed. The only difference in these analyses was that the correlations between Poverty of Speech and other PIP scales were lower in the more restricted subgroups. The correlations of these scales among patients considered not schizophrenic by RDC also paralleled those in the entire sample. In sum, the pattern of all sets of correlations was similar to that shown in table 5.

Table 5. Correlations among the Psychotic Inpatient Profile thought disorder scales

Scale	1	2	3	4	5	6
1. Attentional Impairment	—	.15	.64[1]	.40[1]	.42[1]	.47[1]
2. Poverty of Speech		—	.36[1]	.06	.34	.29
3. Incoherence			—	.51[1]	.53[1]	.60[1]
4. Delusions				—	.57[1]	.81[1]
5. Hallucinations					—	.90[1]
6. Delusions/Hallucinations						—

[1] $p < .01$.

Delusions and Hallucinations ($r = .57$, $df = 52$[1], $p < .001$) and Attentional Impairment and Incoherence ($r = .64$, $df = 52$, $p < .001$) were the two most highly correlated symptom pairs. Incoherence was highly related to the Delusions and Hallucinations scale pair (mean $r = .52$), and Attentional Impairment was moderately correlated with this pair (mean $r = .41$). For the entire sample Poverty of Speech was not correlated with either of these symptom pairs, mean $r = .25$ and .20, respectively, for the Attention-Incoherence and Delusion-Hallucination pairs, and the correlations were even lower for probable and definite schizophrenics.[2]

Correlations With Concurrent Measures. The correlations of the PIP scales with a priori selected concurrent measures for the entire sample and for the diagnostic subgroups are presented in table 6. There are two noteworthy patterns of results relevant to the focus of this article.

The most important pattern of results is the changing relation between the Attentional Impairment scale and the cognitive/perceptual tasks across the RDC subgroups. For the entire sample and for the nonschizophrenics, neither the Shipley Abstraction score nor the three reaction time measures were related to any of the PIP scales. For the probable and definite schizophrenics, only the Attentional Impairment scale consistently correlated with these measures, $r = -.39$, $df = 23$, $p < .07$, $r = .50$ and .45, $df = 18$, $p < .05$, and $r = .63$, $df = 18$, $p < .01$, respectively, for the Shipley, mean reaction time, number incorrect, and variance reaction time for the combined probable and definite schizophrenics, and $r = -.74$, $df = 13$, $p < .01$, $r = .73$, $df = 12$, $p < .01$, and $r = .76$, $df = 12$, $p < .001$, for the Shipley and mean and variance reaction times for the definite schizophrenics only. Thus, as predicted, Attentional Impairment's relation to measures of cognitive performance varied with outcome diagnosis.

The second notable pattern in table 6 is the weak or nonexistent relation of each of the PIP scales in each of the RDC groups both to the Phillips premorbidity scale, a frequent correlate of the negative-positive symptom distinction (e.g., Farmer, McGuffin, and Spitznagel 1983), and to the affective deficit medical-file judgment, a commonly agreed upon negative symptom (Lewine and Sommers 1985) and a rather substantial predictor of poor

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[1] Differences in the degrees of freedom (n-2) reflect different amounts of missing data. In all the analyses described, we used all the data available on these subjects. We were not able to gather the entire battery of measures on all subjects. For example, 55 patients were rated on the PIP, 51 were given the reaction time task, and 62 received medical-file judgments of their affectivity and thought disorder. Of the 55 patients rated on the PIP, 42 had been given the reaction time task, and 48 had received affectivity and thought disorder judgments.

[2] A principal component analysis (PCA) of the five PIP scales on the entire sample yielded a single factor on which Poverty of Speech had a low factor loading and the remaining four PIP scales had high loadings. When other variables that did not measure thought disorder (e.g., Phillips premorbid social competence, affectivity) were introduced into the correlation matrix, and the PCA was repeated with varimax rotation, Poverty of Speech did not load on the factor on which the other four PIP scales continued to load, and it had a high factor loading on another factor that was orthogonal to the PIP Disordered Thought factor.

Table 6. Concurrent correlations of the Psychotic Inpatient Profile (PIP) scales with other prospective measures for the entire sample and for levels of Research Diagnostic Criteria (RDC) diagnostic specification

	Medical-file judgments				Reaction time		
PIP scale	Thought Disorder	Affect Deficit	Shipley Abstraction	Phillips Scale	Mean	Variance	Number Incorrect
Entire Sample (*n* = 55)							
Attentional Impairment	.27	.27	.08	.31[1,2]	.14	.20	.24
Poverty of Speech	.03	.14	.21	.35[2]	−.09	.09	−.15
Incoherence	.31[2]	.10	−.08	.12	.03	.07	.02
Delusions	.35[2]	.20	−.03	.09	.11	.01	.21
Hallucinations	.35[2]	.09	−.05	.09	.18	.17	.06
Delusions/Hallucinations	.35[2]	.18	−.02	.09	.13	.09	.09
RDC definite schizophrenics (*n* = 18)							
Attentional Impairment	.58[2]	.48	−.74[3]	.21	.73[3]	.76[3]	.34
Poverty of Speech	−.42	−.41	.00	.20	−.42	−.14	−.56[2]
Incoherence	.18	−.02	−.44	−.12	.22	.41	−.21
Delusions	.56[2]	.30	−.26	−.04	.35	.59[2]	−.39
Hallucinations	.01	−.35	−.28	−.27	.13	.19	−.41
Delusions/Hallucinations	.18	−.13	−.16	.16	.11	.27	.06
RDC probable or definite schizophrenics (*n* = 27)							
Attentional Impairment	.51[2]	.29	−.39	.09	.50[2]	.63[3]	.45[2]
Poverty of Speech	−.13	.02	−.14	.33	−.29	−.10	−.46[2]
Incoherence	.25	−.03	−.40	−.14	.15	.37	−.25
Delusions	.38	.19	−.17	−.17	.27	.41	−.31
Hallucinations	.02	−.24	−.39	−.31	.12	.14	−.11
Delusions/Hallucinations	.19	−.03	−.03	−.26	.16	.24	−.36
RDC nonschizophrenic (*n* = 26)							
Attentional Impairment	.28	.24	.34	.37	.12	−.01	.11
Poverty of Speech	.19	.28	−.26	.36	.30	.34	.12
Incoherence	.48[4]	.14	.04	.20	.25	.03	.13
Delusions	.42[2]	.22	.00	.25	.17	−.08	.34
Hallucinations	.58[3]	.31	−.04	.36	.33	.22	.31
Delusions/Hallucinations	.48[2]	.25	−.03	.28	.27	.07	.28

[1] Because of missing data, different absolute levels of correlations are significant at different levels.

[2] $p < .05$.

[3] $p < .01$.

outcome (cf. Knight and Roff 1985). The Attentional Impairment and Poverty of Speech scales correlated with poor premorbidity in the full sample, $r = .31$ and $.35$, $df = 48$, $p < .025$, respectively; and both showed a very weak relation to poor premorbidity among the nonschizophrenics, $r = .37$ and $.36$, $df = 22$, $p < .10$, respectively. Neither, however, was related to the Phillips scale in the probable or definite schizophrenic subdivisions. The only scale even remotely related to the affective deficit judgment was the Attentional Impairment scale, $r = .27$, $df = 46$, $p < .07$, and $r = .48$, $df = 12$, $p < .09$, for the entire sample and for the definite schizophrenics, respectively. There appears, therefore, only weak, inconsistent support for the notion that the purportedly negative symptoms of affective deficit, social incompetence, and poverty of speech covary sufficiently to form a cohesive cluster.

Predictive Validity of the PIP Scales. The correlations of the PIP scales with various outcome measures are presented in table 7. Three major results are evident in this table. First and most important, Attentional Impairment was clearly the most consistent predictor among the PIP scales of poor outcome. No matter how the sample was divided, higher Attentional Impairment scores significantly predicted a greater incidence and amount of hospitalization between 1972 and 1978, $r = .36$ and $.34$, $df = 53$, $p < .01$, respectively, for the entire sample; $r = .58$ and $.46$, $df = 25$, $p < .01$ and $.05$, respectively, for the combined probable and definite schizophrenics; $r = .60$ and $.57$, $df = 16$, $p < .01$, respectively, for the definite schizophrenics; and $r = .45$ and $.52$, $df = 24$, $p < .05$ and $.01$, respectively, for the nonschizophrenics. It was also weakly related to poorer Global Outcome for the entire sample, $r = .25$, $df = 51$, $p < .08$, and significantly predicted poorer Global Outcome for each of the subdivisions; $r = .39$, $df = 25$, $p < .05$ for the probable and definite schizophrenics; $r = .47$, $df = 16$, $p < .05$ for the definite schizophrenics alone; and $r = .52$, $df = 23$, $p < .01$ for the nonschizophrenics. Moreover, it tended to forecast problems in establishing deep social relationships during the followup period; $r = .37$, $df = 50$, $p < .01$ for the entire sample; $r = .36$, $df = 25$, $p < .07$ for the probable and definite schizophrenics; $r = .50$, $df = 16$, $p < .05$ for the definite schizophrenics alone; and $r = .35$, $df = 21$, $p < .10$ for the nonschizophrenics. Finally, among the schizophrenic subdivisions, it was prognostic of lack of employment stability; $r = .46$, $df = 23$, $p < .05$ for the probable and definite schizophrenics; and $r = .48$, $df = 15$, $p < .05$ for the definite schizophrenics.

The second finding is the relative lack of prognostic utility of Poverty of Speech and Incoherence, both of which have been classified by some researchers as negative symptoms (e.g., Lewine, Fogg, and Meltzer 1983; Pogue-Geile and Harrow 1984). Consistent with its concurrent associations, Poverty of Speech did predict one measure of social competence, social contacts, for the entire sample, $r = .30$, $df = 49$, $p < .05$, and it was related to the outcome affect deficit judgment for the entire sample, $r = .31$, $df = 51$, $p < .05$, but no clear pattern of relationships to outcome measures emerged. The Incoherence scale only predicted the outcome thought disorder medical-file judgment and Global Outcome for the nonschizophrenics, $r = .51$, $df = 24$, and $r = .48$, $df = 23$, $p < .01$.

The third major finding is that among those diagnosed probable or definite schizophrenic, the Outcome Hallucinations scale was the only outcome disordered thought scale that was related to any earlier disordered thought measures. It was predicted by the PIP Hallucinations and Attentional Impairment scales, for the combined RDC probable and definite schizophrenics, $r = .40$ and $.56$, $df = 24$, $p < .05$ and $.01$, respectively, and for the definite schizophrenics only, $r = .48$ and $.69$, $df = 15$, $p < .05$ and $.01$, respectively. The outcome Formal Thought Disorder and Delusion scales were related to several earlier PIP scales, but only for the nonschizophrenics. Within this group, the Attentional Impairment scale, $r = .40$, $df = 24$, $p < .05$, and the Delusion scale, $r = .41$, $df = 24$, $p < .05$, predicted more outcome Formal Thought Disorder, and less Poverty of Speech foreshadowed more outcome Delusions, $r = -.40$, $df = 24$, $p < .05$. Thus, the stability of our measures of disordered thought was not great. Of note is the finding that Attentional Impairment did predict outcome disordered thought in addition to its relation to the concurrent thought measures.

Relation to RDC Outcome Diagnosis. To determine the relation of scores on the PIP thought disorder scales (the Delusions/Hallucinations scale was excluded because of overlap with its two component scales) to subsequent status on the RDC, we calculated two separate stepwise discriminant function analyses using Wilk's λ (Cooley and Lohnes 1971). In the first, we attempted to discriminate with these five scales the patients included as definite schizophrenics by the RDC from those considered not schizophrenic by these criteria. In the second, we attempted to discriminate the combined RDC definite and probable schizophrenics from the nonschizophrenics. In both analyses, the results were the same—only the Attentional Impairment scale was selected from among the five scales. It alone significantly discriminated the definite schizophrenics from the nonschizophrenics ($\lambda = .89$, $F = 5.34$, $df = 1/42$, $p < .05$), and the combined

Table 7. Predictive correlations of the Psychotic Inpatient Profile (PIP) scales with outcome measures for the entire sample and for levels of Research Diagnostic Criteria (RDC) specification[1]

	Hospitalization		Social relationships			Medical-file judgments		Symptom scales			
PIP scale	Number	Months	Contact	Depth	Employment steadiness	Thought disorder	Affect deficit	Formal thought disorder	Delusions	Hallucinations	Global outcome
Entire sample (n = 55)											
Attentional Impairment	.36^{3}	.34^{3}	.22	.37^{3}	.10	.12	.21	.19	−.06	.18	.25
Poverty of Speech	−.05	−.06	.30^{2}	.20	−.13	−.07	.31^{2}	−.09	−.21	−.20	.24
Incoherence	.09	.16	.02	.17	−.08	.25	.05	.08	.04	.14	.22
Delusions	.07	.17	.03	.05	.02	.17	−.02	.25	.12	.33^{2}	.17
Hallucinations	.04	.09	.08	.10	−.09	.07	.03	.08	.02	.17	.23
Delusions/Hallucinations	.08	.14	.06	.11	−.06	.09	.01	.15	.01	.21	.20
RDC definite schizophrenics (n = 18)											
Attentional Impairment	.60^{3}	.57^{3}	.42	.50^{2}	.48^{2}	.26	.17	.09	.26	.69^{3}	.47^{2}
Poverty of Speech	.26	.08	.18	−.07	.03	.50^{2}	.15	.41	.09	.24	.25
Incoherence	.31	.36	.00	.22	.15	.39	.12	.07	.07	.41	.27
Delusions	.14	.09	.11	.30	.14	.06	.05	−.13	−.27	.32	.16
Hallucinations	.11	.03	.37	−.08	−.28	.13	−.27	−.17	−.20	.48^{2}	.02
Delusions/Hallucinations	.21	.12	.20	.11	−.16	.11	−.14	−.10	−.23	.36	.08
RDC probable and definite schizophrenics (n = 27)											
Attentional Impairment	.58^{3}	.46^{2}	.37	.36	.46^{2}	.24	.20	.19	.08	.56^{3}	.39^{2}
Poverty of Speech	.06	.06	.28	.28	−.06	.08	.27	.01	.07	−.22	.29
Incoherence	.25	.25	.02	.23	.03	.25	.14	−.04	.01	.29	.22
Delusions	.15	.09	.03	.12	.13	.07	−.10	−.11	−.05	.29	.04
Hallucinations	.10	.03	.20	−.13	−.28	.06	−.40^{2}	−.23	.12	.40^{2}	−.10
Delusions/Hallucinations	.24	.14	.09	.01	−.13	.13	−.28	−.06	.03	.38	−.00
RDC nonschizophrenic (n = 26)											
Attentional Impairment	.45^{2}	.52^{3}	.10	.35	−.04	.36	.45^{2}	.40^{2}	−.00	.33	.52^{3}
Poverty of Speech	−.25	−.27	.31	.14	−.15	−.16	.40^{2}	−.18	−.40^{2}	−.17	.27
Incoherence	.08	.23	−.00	.05	−.10	.51^{3}	.11	.27	.16	.36	.48^{3}
Delusions	.03	.35	.03	.00	−.01	.27	.04	.41^{2}	.20	−.50^{3}	.32
Hallucinations	.08	.24	.00	.20	.00	.17	.34	.27	−.00	.32	.57^{3}
Delusions/Hallucinations	.09	.29	.04	.11	−.03	.20	.21	.31	.06	.39^{2}	.48^{3}

[1] Because of missing data, different absolute levels of correlations are significant at different levels.

[2] $p < .05$.

[3] $p < .01$.

probable and definite schizophrenics from the nonschizophrenics ($\lambda = .86$, $F = 8.19$, $df = 1/50$, $p < .01$). Surprisingly, however, in both analyses the nonschizophrenics' scores on the Attentional Impairment scale were significantly *higher* than in the comparison groups.

This paradoxical finding seems even stranger, when compared with the results for these same RDC subgroups of the analysis of another measure of attention, reaction time performance. Table 8 presents the mean reaction time, the mean reaction time variance, and the mean number of incorrect responses per block of trials for the entire sample and for each subgroup separately. One-way analyses of variance comparing the three diagnostic subgroups revealed no group differences on any of these variables, although the mean reaction time differences did approach significance, $F = 2.97$, $df = 2/46$, $p = .06$. A subsequent two-group analysis of variance revealed that the definite schizophrenics had higher reaction times than the probable and nonschizophrenics, $F = 5.83$, $df = 1/47$, $p < .025$.

Discussion

In the analyses of our followup data, we addressed questions about the prognostic utility of components of disordered thought, about the viability of the positive-negative syndrome symptom classification systems, and about the cognitive processes underlying disordered thought symptoms.

Specificity and Prognosis of Thought Components. We found that various components of disordered thinking could be distinguished by the pattern of their interrelations, their correlations with concurrent variables, and their differential predictive power. The correlations among the five PIP scales (excluding the hybrid Delusions/Hallucinations scale, whose results differed little from its constituent parts) suggested that Poverty of Speech was distinct from the other four scales. It had low or nonsignificant correlations with the other scales, which all correlated moderately or highly with each other. Among the remaining four scales, Attentional Impairment and Incoherence, and Delusions and Hallucinations were the most highly correlated scale pairs, but did not form clear subclusters, because all scales were significantly correlated.

The five scales showed distinctly different patterns of correlations with other prospective measures and with the various outcome criteria. Consistent with Pogue-Geile and Harrow's (1984, 1985) findings, our Poverty of Speech scale correlated with premorbid social competence. The predictive power of this scale was limited to social and affective competence within the entire psychotic sample, except for two significant correlations with the outcome thought disorder medical-file record judgment and the Formal Thought Disorder outcome scale among the definite schizophrenics and nonschizophrenics, respectively. Thus, it seems likely that the behaviors included in this scale were

Table 8. Reaction time performance for 75 patients

	Entire sample ($n = 75$)	RDC non-sz ($n = 35$)	RDC probable sz ($n = 11$)	RDC definite sz ($n = 26$)
Mean reaction time (seconds)				
Mean	.794 (51)	.745 (23)	.703 (7)	.884 (19)[1]
SD	.217	.187	.156	.253
Mean reaction time Variance (seconds)				
Mean	.089 (51)	.078 (23)	.065 (7)	.104 (19)
SD	.076	.074	.027	.090
Mean number incorrect per block				
Mean	.074 (51)	.075 (23)	.097 (7)	.064 (19)
SD	.043	.042	.067	.035

Note.—RDC = Research Diagnostic Criteria; sz = schizophrenic; *n*'s are shown in parentheses.

[1] RDC definite schizophrenics had significantly longer reaction times than the combined probable schizophrenics and nonschizophrenics.

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tapping more social than cognitive competence, and its status as a disordered thought symptom might be questioned.

Although the Attentional Impairment and the Incoherence scales were highly correlated with each other, both the pattern of their concurrent correlations and their predictive power were radically different. Incoherence was only related to the thought disorder judgment at initial assessment for the entire sample and for the nonschizophrenics. It failed as a predictor, showing only minor sporadic relations to outcome for the nonschizophrenics and no ability to discriminate outcome diagnosis. Because of its poor interrater reliability, final evaluation of the status of this symptom must, however, await further evidence. In contrast, the Attentional Impairment scale was the only thought disorder scale to have a strong relation to poor outcome. It predicted hospitalization, social competence, work steadiness, and global outcome for both schizophrenics and nonschizophrenics, suggesting that it is assessing a persistent, nonspecific, core deficit. Contrary to the expectation that the behaviors assessed on the Attentional Impairment scale would be more characteristic of narrowly defined schizophrenics, they were found to a greater degree in RDC nonschizophrenics than in definite schizophrenics.

It is noteworthy that the Attentional Impairment scale showed its predictive power in spite of its low interrater reliability. It had the lowest interrater reliability of the six PIP scales assessed. The consistency and pervasiveness of its predictions argue against attributing these results to chance. It is possible that among the multiple raters there were several less sensitive raters whose ratings lowered the overall reliability, yet did not destroy the scale's ability to tap a predictive, reasonably cohesive domain. Despite the very low interrater reliability, the internal consistency of the scale was still moderate, suggesting that it is also unlikely that the scale served simply as a broadband catchment for psychopathology and was predictive simply because of its wider sampling of pathological behaviors (Ghiselli, Campbell, and Zedeck 1981). It seems more probable that the scale is globally tapping behavioral problems in attending and that a more precise defining of items and training of raters (cf. Knight and Blaney 1977) might yield better prediction. Both these results and our previously reported affective deficit results (Knight and Roff 1985) are cogent reminders that reliability and discriminatory power are not necessarily linked (cf. Nicewander and Price 1983) and that theoretically important measures should not be discarded simply because they are difficult to assess reliably.

The remaining three scales were composed exclusively of measures of thought content. The Delusions scale was consistently related to the concurrent thought disorder medical-file judgment across the various divisions of the sample, was correlated with larger variance of reaction times for the RDC definite schizophrenics, but was unrelated to any outcome measures except for the Outcome Hallucinations scale for the entire sample. The PIP Hallucinations scale was related to the concurrent thought disorder medical-file judgment for the entire sample and for the nonschizophrenics, but not for the schizophrenics. The only outcome measures it predicted were Hallucinations for the schizophrenics and better affectivity for the combined RDC probable and definite schizophrenics. The sixth scale, the combined Delusions/Hallucinations scale, which had a wider sampling of thought content, was not a better predictor than its constituents. Thus, all of the scales of thought content showed weak predictive validity.

In sum, only one of the PIP disordered thinking scales, Attentional Impairment, predicted general aspects of adaptation 7 years later. Paradoxically, however, it was found to be less prevalent in patients diagnosed as definite schizophrenics by RDC at outcome than in psychotic patients diagnosed nonschizophrenic by these same criteria. Two other scales showed evidence of only domain-specific prediction. Poverty of Speech predicted social and affective competence for the entire sample, and the Hallucinations scale was related to outcome hallucinations, but only among the schizophrenics. Finally, neither Delusions nor Incoherence predicted either outcome adaptation or diagnosis.

Positive and Negative Syndromes. The pattern of results provides only weak support for the hypothesis that the symptom clusters Delusions/Hallucinations and Poverty of Speech/Affective Deficit tap separate, cohesive syndromes with distinguishable predictive and discriminatory power. First, there was some evidence that the two hypothesized syndrome clusters were separable. Consistent with the majority of previous analyses of the relation between positive and negative symptoms, Poverty of Speech/Affective Deficit and Delusions/Hallucinations did not correlate with each other (Johnstone et al. 1981; Lewine, Fogg, and Meltzer 1983; Pogue-Geile and Harrow 1984, 1985; Rosen et al. 1984; but not Andreasen and Olsen

1982). Second, although the positive symptoms that we assessed were cohesive, the two negative symptoms were not. Delusions and Hallucinations were highly correlated, suggesting that these scales might be tapping the same positive syndrome. In contrast, Poverty of Speech was only correlated with the outcome Affective Deficit judgment and not with the concurrent judgment, indicating weak cohesion between these supposedly negative symptoms. Third, consistent with the expectations for positive symptoms, Delusions and Hallucinations showed little predictive validity and no ability to discriminate RDC definite schizophrenics. Poverty of Speech, however, unlike the Affective Deficit judgment (cf. Knight and Roff 1985), did not show the predictive power expected of a negative symptom. It was only weakly related to specific domains of outcome and did not discriminate narrowly defined schizophrenics. In sum, the positive symptoms results were in accord with the expectations of the syndrome model, but for the negative symptoms only Affective Deficit results and not Poverty of Speech results supported the model. Thus, Poverty of Speech, at least as measured in this study, does not appear to tap a negative syndrome.

Because of poor interrater reliability of our Incoherence scale and the lack of items on the PIP to allow us to differentiate it adequately from tangentiality and derailment, its failure to discriminate diagnostic groups and to predict outcome cannot be taken as conclusive evidence that it should be excluded from the negative symptom syndrome. These failures should not, however, be completely discounted. Attentional Impairment had similar problems with interrater reliability and definitional specificity, yet predicted better than any measure of disordered thought, including our highly reliable medical-file Thought Disorder judgment (cf. Knight and Roff 1985).

The predictive power of the Attentional Impairment scale supports the categorization of attention as a negative symptom. The fact that the attentional behaviors assessed by our scale were more characteristic of outcome RDC nonschizophrenics than of RDC definite schizophrenics, however, contradicts this conclusion. This paradoxical combination of pervasive predictive power for all subgroups assessed and diagnostic discrimination opposite to that which would be predicted suggests that the behavioral manifestations of attentional problems in these different diagnostic subgroups might result from different underlying cognitive processes, each of which has prognostic utility. Such a hypothesis is supported by other studies (e.g., Bilder et al. 1985; Cornblatt et al. 1985) that have yielded evidence suggesting the complexity of global behavioral attention measures. Our analysis of the concurrent correlations of our Attentional Impairment scale, described below, also corroborates this conclusion. If this speculation is correct, we may not be able to solve the problem of partitioning such overt thought symptoms until we have a better understanding of the cognitive processes that produce them. It may well be that when such processes are better understood, the positive-negative distinction will be shown to be an oversimplified dichotomy, as others have suggested (Bilder et al. 1985; Gibbons et al. 1985).

Underlying Cognitive Processes. As a way of exploring the possibility that different underlying cognitive processes might be responsible for similar disordered thinking behaviors in different pathologies, we examined the pattern of correlations between the five components of disordered thinking and the two cognitive measures across subgroups defined by outcome diagnosis. This analytic strategy, as we explained in the introduction, was generated to test our interpretation of the results of another followup study (Knight and Roff 1983). Because performance on choice reaction time, one of our cognitive measures, is vulnerable to attentional problems, and because other studies have found evidence suggesting that the behaviors used to infer attentional problems may be multiply determined, we hypothesized that we were most likely to find evidence for different underlying processes for the behaviors assessed by our Attentional Impairment scale. Our speculation was correct. The lack of relation between the reaction time measures and the Attentional Impairment scale for the entire sample and for the RDC nonschizophrenics, and the high correlation between these measures for the definite schizophrenics (cf. table 6) suggests that the manifest behaviors captured by the Attentional Impairment scale might be produced by different processes for the nonschizophrenics and probable schizophrenics than for the definite schizophrenics.

Paradoxically, behavioral attention problems were more prevalent among the RDC nonschizophrenics than among the probable or definite schizophrenics. Because there was no correlation between Attentional Impairment and the cognitive measures for these patients, and because their reaction times were faster than those of the definite schizophrenics, we have no basis in our data for speculation about the nature of the deficit producing their behavioral attention problems. Whatever the deficit, our results suggest that it is related to poorer outcome among these patients. As indicated in the **Methods** section, this group was composed almost completely of affective, schizoaffective, and paranoid cases. Because the Attentional Impairment scale predicted poorer long-term outcome among these patients, it is unlikely that their attentional problems were due to transient acute affective or paranoid states. It is important when thinking about the seeming paradox of finding the supposedly better prognosis nonschizophrenics to have a greater amount of a debilitating deficit to remember that RDC criteria for schizophrenia focus less on chronicity and poor outcome than do other diagnostic criteria (Marvinney, Rosenberg, and Knight 1982). Indeed, these criteria selected as definite schizophrenics patients who did not differ from the excluded patients in their Phillips premorbid adjustment scores.

The high correlation between Attentional Impairment and choice reaction time for definite schizophrenics and the predictive power of both of these measures (cf., also, Elliott and Knight 1982) suggests that for a subset of these definite schizophrenics, some deficiency involving attentional processes constitutes a core deficit. The nature of this deficit is far from certain. The specification of an underlying process requires more than performance on any single laboratory task (cf. Knight 1984). Moreover, choice reaction time performance involves too many processing levels to yield specific implications (e.g., Smith 1980). Thus, the covariation of reaction time performance and the Attentional Impairment scale among the definite schizophrenics does not lead us to believe that we have explained the deficit producing attentional difficulties in a subset of RDC definite schizophrenics. Indeed, these data raise more questions than they answer. For instance, it has been found in the cognitive research on schizophrenics that an early encoding deficit covaries with premorbidity (Saccuzzo and Braff 1981; Knight, Elliott, and Freedman 1985). In the present study, neither reaction time performance nor scores on the Attentional Impairment scale covaried with premorbidity. This pattern suggests the possibility that the reaction time deficiency might be the result of a different underlying cognitive deficit, and that it is this deficit and not the early encoding deficit that is responsible for the kinds of behavioral problems assessed by the Attentional Impairment scale. The independence of reaction time level and early encoding deficiencies is consistent with the results of recent cognitive studies in our laboratory (Knight et al. 1985). Clearly, more research is needed to resolve this and other process-oriented questions. Moreover, the results of the present study must be replicated, especially because of the paradoxical and unexpected nature of certain findings.

Finally, these data highlight the complexity of the task of specifying the cognitive processes responsible for the disordered thought behaviors encountered in schizophrenia. In attempts at integrating laboratory tasks that supposedly assess specific processes with scales of more manifest and naturalistic disordered thought behaviors, it is not sufficient to correlate the two (Neale, Oltmanns, and Harvey 1985). Rather, more complex approaches must be undertaken, such as the application to psychiatric populations of the strategies proposed for the cognitive assessment of individual differences (Sternberg 1981). In addition to the preliminary groundwork of examining the relation of various processing tasks to symptom manifestations within specific patient groups (the cognitive correlates approach), we must also ultimately create and test cognitive process models of symptom manifestation (cf. Knight 1984, for an example of a primitive model). This latter approach, which is similar to Sternberg's cognitive components strategy, enhances the opportunity for disconfirmation and thus maximizes the potential for advancing our knowledge (Popper 1972). The data from the present study suggest that in attempting these strategies, we need to be guided not only by specific models of cognition, but also by better typological models of psychoses and schizophrenia.

References

American Psychiatric Association. *DSM-II: Diagnostic and Statistical Manual of Mental Disorders*. 2nd ed. Washington, DC: The Association, 1968.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

Andreasen, N.C. Thought, language, and communication disorders: I. Clinical assessment, definition of terms, and evaluation of their reliability. *Archives of General Psychiatry*, 36:1315–1321, 1979*a*.

Andreasen, N.C. Thought, language, and communication disorders: II. Diagnostic significance. *Archives of General Psychiatry*, 36:1325–1330, 1979*b*.

Andreasen, N.C. Negative symptoms

in schizophrenia: Definition and reliability. *Archives of General Psychiatry*, 39:784–786, 1982.

Andreasen, N.C. Positive vs. negative schizophrenia: A critical evaluation. *Schizophrenia Bulletin*, 11:380–389, 1985.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia. *Archives of General Psychiatry*, 39:789–794, 1982.

Astrachan, B.M.; Harrow, M.; Adler, D.; Brauer, L.; Schwartz, A.; Schwartz, C.; and Tucker, G. A checklist for the diagnosis of schizophrenia. *British Journal of Psychiatry*, 121:529–539, 1972.

Astrup, C., and Noreik, K. *Functional Psychoses: Diagnostic and Prognostic Models*. Springfield, IL: Charles C Thomas, Publisher, 1966.

Bilder, R.M.; Mukherjee, S.; Rieder, R.O.; and Pandurangi, A.K. Symptomatic and neuropsychological components of defect states. *Schizophrenia Bulletin*, 11:409–419, 1985.

Bland, R.C., and Orn, H. Schizophrenia: Schneider's first-rank symptoms and outcome. *British Journal of Psychiatry*, 137:63–68, 1980.

Carpenter, W.T., Jr.; Strauss, J.S.; and Bartko, J.J. A flexible system for the diagnosis of schizophrenia: Report from the WHO International Pilot Study of Schizophrenia. *Science*, 182:1275–1278, 1973.

Cooley, W.W., and Lohnes, P.R. *Multivariate Data Analysis*. New York: John Wiley & Sons, 1971.

Cornblatt, B.A.; Lenzenweger, M.F.; Dworkin, R.H.; and Erlenmeyer-Kimling, L. Positive and negative schizophrenic symptoms, attention, and information processing. *Schizophrenia Bulletin*, 11:397–408, 1985.

Crow, T.J. Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal*, 280:1–9, 1980.

Crow, T.J. The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin*, 11:471–486, 1985.

DeWolfe, A.S. Self-reports and case histories of schizophrenic patients: Reliability and validity of Phillips scale ratings. *Journal of Clinical Psychology*, 24:415–418, 1968.

Elliott, D.S., and Knight, R.A. "The Predictive Validity of Components of Reaction Time Performance in Psychiatric Patients." Presented at the Annual Meeting of the Eastern Psychological Association, Baltimore, MD, April, 1982.

Farina, A.; Garmezy, N.; and Barry, H. III. Relationship of marital status to incidence and prognosis of schizophrenia. *Journal of Abnormal and Social Psychology*, 67:624–630, 1963.

Farina, A.; Garmezy, N.; Zalusky, M.; and Becker, J. Premorbid behavior and prognosis in female schizophrenic patients. *Journal of Consulting Psychology*, 26:56–60, 1962.

Farmer, A.E.; McGuffin, P.; and Spitznagel, E.L. Heterogeneity in schizophrenia: A cluster-analytic approach. *Psychiatry Research*, 8:1–9, 1983.

Feighner, J.; Robins, E.; Guze, S.; Woodruff, R., Jr.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 26:57–63, 1972.

Ghiselli, E.E.; Campbell, J.P.; and Zedeck, S. *Measurement Theory for the Behavioral Sciences*. San Francisco: Freeman, 1981.

Gibbons, R.D.; Lewine, R.R.J.; Davis, J.M.; Schooler, N.R.; and Cole, J.O. An empirical test of a Kraepelinian vs. a Bleulerian view of negative symptoms. *Schizophrenia Bulletin*, 11:390–396, 1985.

Gordon, A.V., and Gregson, R.A.M. The symptom-sign inventory as a diagnostic differentia for paranoid and non-paranoid schizophrenics. *British Journal of Social and Clinical Psychology*, 9:347–356, 1970.

Hawk, A.B.; Carpenter, W.T., Jr.; and Strauss, J.S. Diagnostic criteria and 5-year outcome in schizophrenia: Report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry*, 32:343–356, 1975.

Holzman, P.S.; Solovay, M.R.; and Shenton, M.E. Thought disorder specificity in functional psychoses. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies*. New York: Guilford Press, 1985. pp. 228–245.

Jansson, B. The prognostic significance of various types of hallucinations in young people. *Acta Psychiatrica Scandinavica*, 44:401–409, 1968.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking*. San Francisco: Jossey-Bass, 1979.

Johnstone, E.C.; Owens, D.G.C.; Gold, A.; Crow, T.J.; and MacMillan, J.F. Institutionalization and the defects of schizophrenia. *British Journal of Psychiatry*, 139:195–203, 1981.

Knight, R.A. Converging models of cognitive deficit in schizophrenia. In: Spaulding, W.D., and Cole, J.K., eds. *Nebraska Symposium on Motivation, 1983: Theories of Schizophrenia and Psychosis*. Lincoln, NE: University of Nebraska Press, 1984. pp. 93–156.

Knight, R.A., and Blaney, P.H. The interrater reliability of the Psychotic Inpatient Profile. *Journal of Clinical Psychology*, 33:647–653, 1977.

Knight, R.A.; Elliott, D.S.; and Freedman, E.G. Short-term visual memory in schizophrenics. *Journal of Abnormal Psychology*, 94:427–442, 1985.

Knight, R.A.; Elliott, D.S.; Hershenson, M.; and Frost, R. "Matching-Task Assessments of Perceptual Organization in Schizophrenics." Unpublished raw data, 1985.

Knight R.A.; Epstein, B.; and Zielony, R.D. The validity of the Whitaker Index of Schizophrenic Thinking. *Journal of Clinical Psychology*, 36:632–639, 1980.

Knight, R.A., and Roff, J.D. Childhood and young adult predictors of schizophrenic outcome. In: Ricks, D., and Dohrenwend, B., eds. *Origins of Psychopathology: Research and Public Policy*. Cambridge: Cambridge University Press, 1983. pp. 129–153.

Knight, R.A., and Roff, J.D. Affectivity in schizophrenia. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies*. New York: Guilford Press, 1985. pp. 280–316.

Knight, R.A.; Roff, J.; Barrnett, J.; and Moss, J. Concurrent and predictive validity of thought disorder and affectivity: A 22-year follow-up of acute schizophrenics. *Journal of Abnormal Psychology*, 88:1–12, 1979.

Levin, S.; Hall, J.A.; Knight, R.A.; and Alpert, M. Verbal and nonverbal expression of affect in speech of schizophrenic and affective disorder patients. *Journal of Abnormal Psychology*, 94:487–497, 1985.

Lewine, R.R.J.; Fogg, L.; and Meltzer, H.Y. Assessment of negative and positive symptoms in schizophrenia. *Schizophrenia Bulletin*, 9:368–376, 1983.

Lewine, R.R.J., and Sommers, A.A. Clinical definition of negative symptoms as a reflection of theory and methodology. In: Alpert, M., ed. *Controversies in Schizophrenia: Changes and Constancies*. New York: Guilford Press, 1985. pp. 267–279.

Lorr, M., and Vestre, N.D. *Psychotic Inpatient Profile Manual*. Los Angeles: Western Psychological Services, 1968.

Lorr, M., and Vestre, N.D. The Psychotic Inpatient Profile: A nurse's observation scale. *Journal of Clinical Psychology*, 25:137–140, 1969.

Marvinney, D.A.; Rosenberg, E.; and Knight, R.A. "The Identification of Schizophrenics: Differential Selection by Five Research Diagnostic Systems." Presented at the Annual Meeting of the Eastern Psychological Association, Baltimore, MD, April 1982.

Neale, J.M.; Oltmanns, T.F.; and Harvey, P.D. The need to relate cognitive deficits to specific behavioral referents of schizophrenia. *Schizophrenia Bulletin*, 11:286–291, 1985.

Nicewander, W.A., and Price, J.M. Reliability of measurement and the power of statistical tests: Some new results. *Psychological Bulletin*, 94:524–533, 1983.

Nuechterlein, K.H., and Dawson, M.E. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10:160–203, 1984.

Oltmanns, T.F.; Murphy, R.; Berenbaum, H.; and Dunlop, S.R. Rating verbal communication impairment in schizophrenic and affective disorders. *Schizophrenia Bulletin*, 11:292–299, 1985.

Phillips, L. Case history data and prognosis in schizophrenia. *Journal of Nervous and Mental Disease*, 117:515–525, 1953.

Pogue-Geile, M.F., and Harrow, M. Negative and positive symptoms in schizophrenia and depression: A followup. *Schizophrenia Bulletin*, 10:371–387, 1984.

Pogue-Geile, M.F., and Harrow, M. Negative symptoms in schizophrenia: Their longitudinal course and prognostic importance. *Schizophrenia Bulletin*, 11:427–439, 1985.

Popper, K.R. *Objective Knowledge: An Evolutionary Approach*. Oxford: Clarendon Press, 1972.

Pope, H.G., and Lipinski, J.F. Diagnosis in schizophrenia and manic-depressive illness. *Archives of General Psychiatry*, 35:811–828, 1978.

Roff, J.D. Reminder: Reliability of global judgments. *Perceptual and Motor Skills*, 52:315–318, 1981.

Roff, J.D.; Knight, R.; and Wertheim, E. A factor analytic study of childhood symptoms antecedent to schizophrenia. *Journal of Abnormal Psychology*, 85:543–549, 1976.

Rosen, W.G.; Mohs, R.C.; Johns, C.A.; Small, N.S.; Kendler, K.S.; Horvath, T.B.; and Davis, K.L. Positive and negative symptoms in schizophrenia. *Psychiatry Research*, 13:227–284, 1984.

Saccuzzo, D.P., and Braff, D.L. Early information processing deficit in schizophrenia: New findings using schizophrenic sub-groups and manic control subjects. *Archives of General Psychiatry*, 38:175–179, 1981.

Shipley, W.C. A self-administering scale for measuring intellectual impairment and deterioration. *Journal of Psychology*, 9:371–377, 1940.

Smith, G.A. Models of choice reaction time. In: Welford, A.T., ed.

Reaction Times. London: Academic Press, Inc., 1980. pp. 173-214.

Sommers, A.A. "Negative symptoms": Conceptual and methodological problems. *Schizophrenia Bulletin*, 11:364-379, 1985.

Spitzer, R.L.; Endicott, J.; and Robins, E. *Research Diagnostic Criteria*. New York: New York State Psychiatric Institute, 1978.

Stephens, J.H. Long-term prognosis and followup in schizophrenia. *Schizophrenia Bulletin*, 4:25-47, 1978.

Sternberg, R.J. Testing and cognitive psychology. *American Psychologist*, 36:1181-1189, 1981.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: I. Characteristics of outcome. *Archives of General Psychiatry*, 27:739-746, 1972.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia: II. Relationships between predictor and outcome variables. *Archives of General Psychiatry*, 31:37-42, 1974.

Strauss, J.S.; Carpenter, W.T., Jr.; and Bartko, J.J. The diagnosis and understanding of schizophrenia: Part III. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 11):61-69, 1974.

Taylor, M.A.; Abrams, R.; and Gaztanaga, P. Manic-depressive illness and schizophrenia: A partial validation of research diagnostic criteria utilizing neuropsychological testing. *Comprehensive Psychiatry*, 16:91-96, 1975.

Watson, C.G., and Klett, W.G. A validation of the Psychotic Inpatient Profile. *Journal of Clinical Psychology*, 28:102-109, 1972.

Wing, J.K. Clinical concepts of schizophrenia. In: Wing, J.K., ed. *Schizophrenia: Toward a New Synthesis*. London: Academic Press, Inc., 1978. pp. 1-30.

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Information-Processing Abnormalities: Trait- and State-Dependent Components

by Dennis P. Saccuzzo and David L. Braff

Abstract

Schizophrenics were compared to schizoaffective, bipolar, and nonpsychotic depressed patients in a visual masking paradigm in which an informational target stimulus was followed at varying intervals by a noninformational masking stimulus. In limiting the availability of the sensory signal provided by the target stimulus, the mask was used to probe how information from the environment enters and is processed by the central nervous system. The use of the masking paradigm was originally based on the hypothesis that thought disorder is a result of a more primary dysfunction in the processes that precede and result in thought. Results confirmed previous findings of a performance deficit in the schizophrenics when compared to nonpsychotic controls. Schizoaffective and bipolar patients also showed evidence of impaired processing, however. Results were interpreted in terms of a trait/state formulation in which impaired information processing is seen as a fundamental trait of schizophrenia spectrum disorders and as a state that can covary with psychotic illness in general. A unifying concept centers on the effects of psychopathological conditions on an individual's processing resources that results in either underprovision or overprovision of information from sensory input to complex cognitive operations dependent on the cerebral cortex. Findings from a variety of paradigms are consistent with those of the masking paradigm in revealing that the processing deficits of schizophrenics are time dependent and occur in the 500 ms following stimulus input.

Formal thought disorder is a chief defining characteristic of the group of schizophrenic disorders. In this context, information-processing approaches to schizophrenia view thought as the product of a number of stages or processes, such as the selection of relevant information from the environment, elaboration and interpretation of input, and the transfer of information throughout the nervous system. Information-processing approaches to the schizophrenias attempt to isolate an abnormality in the processes that result in thought. It is this underlying attentional or information-processing abnormality, not thought per se, that is viewed as the basic cognitive dysfunction in schizophrenia.

Venables (1964, 1973, 1984), for instance, has argued that disturbed thinking in schizophrenia can be traced to an "input dysfunction," in which, due to a disturbance in the central and peripheral mechanisms of arousal, schizophrenics take in either too much or too little information. Silverman (1964) similarly proposed that thought disorder was due to a "narrowing" or "broadening" of attention in which either too little or too much information was transferred to the higher cortical centers for analysis. Other theorists have pointed to a breakdown in a hypothetical filter that normally serves to screen out irrelevent input. The impaired filtering hypothetically causes the higher centers to become overwhelmed and results in distortions in abstraction and reasoning (Weckowicz and Blewett 1959; McGhie and Chapman 1961; Payne 1966). Still others have attributed thought disorder to an abnormally slow rate of information transfer, which results in critical losses of information and distorted interpreta-

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tions of events (Yates 1966). More recently, Callaway and Naghdi (1982) presented a model of schizophrenia as a disorder of information processing in which schizophrenics were seen as deficient in processing tasks characterized as conscious, serial, and limited in channel capacity. Processing tasks characterized as automatic, unconscious, parallel, and of very large capacity, by contrast, were hypothesized to be normal or supranormal. In this context, new evidence indicates that even apparently simple letter-detection tasks may fall into the serial-processing category (Sagi and Julesz 1985), thus accounting for the rather global deficits seen in schizophrenic patients' information-processing abilities.

The information-processing approach to the study of thought disorder in the schizophrenias is itself in the early stages of development (Kietzman, Spring, and Zubin 1980). There have been a number of important attempts to integrate existing findings along constructs such as hemispheric processing (Gur 1977, 1978; Magaro 1980), the developmental course of schizophrenia (Nuechterlein and Dawson 1984), and perceptual organization (Knight 1984; Kietzman and Zubin, in press). At present, however, there exists no fully developed or widely held model.

The current state of the information-processing approach to schizophrenia, its promise as well as its limitations and obstacles, is exemplified in studies involving the brief presentation of two or more stimuli in rapid succession, such as those involving visual masking. In a visual-masking paradigm, an informational target stimulus is presented in close temporal succession with a noninformational masking stimulus (Kahneman 1968). When a visual stimulus is presented for a short time and then is terminated, visual persistence and a host of possible individual differences in how the sensory signal might be stored and analyzed make it virtually impossible for a researcher to evaluate how the sensory impulse is experienced by the higher brain centers. The purpose of the noninformation masking stimulus is to limit the manner in which a sensory signal might vary, thus providing critical control of input. As Felsten and Wasserman (1980) state in their comprehensive review of the masking literature:

> backward masking . . . limits the duration of the sensory signal itself . . . . [The mask] can be used to control the information content of the sensory signal representing the TS [target stimulus] . . . a mask permits the cognitive investigator to do something important that cannot be done by other means, namely, to deliver a pulse of information to the central nervous system with precise control on the duration of that pulse. Without . . . masking, visual persistence will guarantee that the duration of the information pulse experienced by the central nervous system will vary in an exceedingly complex manner. [p. 350]

To evaluate possible deficits in one stage of information processing, it is necessary to either control or minimize the demands on all other stages. Such was the goal in the first effort to apply the visual masking technique to schizophrenia. Saccuzzo, Hirt, and Spencer (1974) attempted to control for possible differences in the initial strength of the sensory impulse by determining the minimal target exposure duration for criterion accuracy for groups of paranoid and nonparanoid schizophrenics, nonpsychotic psychiatric patients, and normal controls. Then, to control the duration of the signal, target presentations were followed by a noninformational mask at intervals of 50, 100, 150, 200, and 300 ms following termination of the target. To minimize the effects of short-term memory and higher cognitive processing such as organizational strategies, the task was a simple two-choice forced discrimination of a capital "A" or "T." Results showed that the two control groups achieved the no-mask condition level of accuracy at the 150 ms interval. Schizophrenics, by contrast, did not approach their no-mask performance levels until the 300 ms interval. The findings, which revealed that schizophrenics required more time than controls to process the target and/or deal with the noninformational mask, supported the notion that there is a disturbance in the processes that precede and lead to conscious thought in schizophrenic patients.

The findings of Saccuzzo, Hirt, and Spencer (1974) have subsequently been replicated and extended under a variety of conditions, and have been shown to be highly reliable (Saccuzzo and Miller 1977; Braff and Saccuzzo 1981; Saccuzzo, Braff, and Sprock 1982; Schwartz, Winstead, and Adinoff 1983; Green and Walker 1984). The use of signal-detection methodology, which takes into consideration errors as well as correct responses, has ruled out differences in visual sensitivity and response strategy in the findings, indicating that the observed schizophrenic deficit in the masking paradigm is indeed due to impaired information-processing capabilities (Saccuzzo and Braff 1981; Saccuzzo and Michael 1984). Study of the effects of medication indicated that antipsychotic drugs may actually enhance the performance of schizophrenics in the masking and related attentionally dependent paradigms

(Braff and Saccuzzo 1982). And by demonstrating quantitative as well as qualitative differences in the masking performance of schizophrenics and the elderly, who show generalized deterioration, Brody, Saccuzzo, and Braff (1980) have argued against generalized deficit as the primary factor in the poor masking performance of schizophrenics (see Saccuzzo 1977, for a discussion of generalized deficits in schizophrenia and old age).

Because no single study can reasonably be expected to cover all of the myriad possibilities, information-processing approaches to schizophrenia have required the support of a marshaling of evidence (Saccuzzo, in press). A series of studies, for example, dealt with the issue of whether the processing disturbance, as revealed by the masking paradigm, is simply the result of the disorder, or a more fundamental problem linked to its cause. Miller, Saccuzzo, and Braff (1979) studied the masking performance of remitted schizophrenics. Although free of significant thought disorder, these individuals exhibited the same poor performance as actively psychotic schizophrenics, supporting the conclusion that impaired processing is not dependent on the presence of thought disorder and, indeed, may be a more enduring feature of schizophrenia than the thinking disorder itself. In support of such a conclusion, Braff (1981) and Saccuzzo and Schubert (1981) independently demonstrated impaired masking performance in schizotypal personality disordered patients with a nonpsychotic schizophrenia spectrum disorder believed to be genetically linked to schizophrenia (Heston 1966, 1970; Meehl 1962). An important feature of Braff's (1981) study is that none of the schizotypal patients were receiving medication; yet, they were as impaired as a group of schizophrenics, most of whom were receiving antipsychotics. Such findings parallel those that emerge when related techniques are used to test remitted schizophrenics (Asarnow and MacCrimmon 1978) and children at risk for schizophrenia (Asarnow et al. 1978). The finding of impaired information-processing capabilities in schizophrenia spectrum disorders cannot be attributed to medication, hospitalization, or active thought disorder, according to Minnesota Multiphasic Personality Inventory (MMPI) studies, which have found poor masking performance in college students showing MMPI patterns associated with the schizotypic 2–7–8 code type and/or schizophrenic linked 8/9/9–8 code type (Steronko and Woods 1978; Merritt and Balogh 1984; Saccuzzo et al. 1984; Balogh and Merritt, in press; Nakano and Saccuzzo, in press).

That an information-processing disturbance, as revealed in the masking paradigm, may be a trait of certain subtypes of schizophrenics and only a state in others was revealed in a study of Saccuzzo and Braff (1981). Good prognostic schizophrenics were compared to poor prognostic schizophrenics and psychiatric as well as normal controls in eight experimental sessions during the course of their hospitalization in an acute care psychiatric hospital. The psychiatric controls consisted of a group of depressive neurotics and a group of manic controls demonstrating hypomania with no evidence of formal thought disorder. Results showed that the poor prognosis schizophrenics had a pronounced impairment in their ability to evade the effects of the mask for all eight sessions. The good prognosis schizophrenics, by contrast, showed improved masking performance as their clinical state improved. It was hypothesized that impaired information processing is a relatively pervasive and long-lasting *trait* of the poor prognosis schizophrenic patients. In addition, impaired processing was viewed as a more transient *state* of the good prognosis schizophrenic patients that covaries with their active psychosis and theoretically with their thought disorder.

If impaired information processing can be both a trait of the poor prognosis schizophrenic and schizotypal patients and a state that covaries with thought disorder, then one might expect to find evidence of impaired information processing in nonschizophrenic thought-disordered patients. Indeed, Schubert, Saccuzzo, and Braff (1985) found evidence of a continuum in the degree of information-processing impairment among schizoaffective, manic, and psychotic-depressive disorders. All three groups showed poor masking performance relative to nonpsychotic psychiatric and normal controls, yet could be distinguished among themselves. Thus, unlike the less impaired manics tested by Saccuzzo and Braff (1981), the psychotically disturbed manics tested by Schubert, Saccuzzo, and Braff (1985) did show evidence of a masking disorder. It remained to be shown, however, whether schizophrenics could be distinguished from other psychotically disturbed persons, an issue that is addressed in the present article.

Method

Subjects. Forty-four psychiatric inpatients consisting of 11 schizophrenic disorders, 11 schizoaffective disorders, 11 bipolar affective disorders, and 11 nonpsychotic depressed individuals were tested. The schizophrenic, schizoaffective,

Table 1. Mean age, Vocabulary, Global Assessment Scale (GAS), and critical stimulus duration (CSD) scores

	Schizo-phrenic	Schizo-affective	Bipolar	Non-psychotic depressed
Age				
Mean	27.18	26.82	28.73	26.64
SD	5.51	6.42	7.18	7.28
Vocabulary				
Mean	10.27	11.64	11.73	10.73
SD	2.87	2.94	1.74	2.05
GAS, lowest				
Mean	30.91	30.82	37.64	48.55
SD	9.40	8.80	14.14	10.87
GAS, overall				
Mean	4.91	5.27	4.73	3.36
SD	1.04	0.79	1.10	0.81
CSD				
Mean	12.18	18.18	13.09	6.36
SD	15.58	18.33	17.39	4.86

and bipolar affective disorders met the Research Diagnostic Criteria (RDC) of Spitzer, Endicott, and Robins (1977) for their respective groups. All diagnoses were determined by a senior psychiatrist (D.L.B.) and a trained research assistant. The nonpsychotic depressed group met the RDC for a minor depressive disorder. This group provided a nonpsychotic control group. The groups were matched on age and intelligence as evaluated by their scaled score on the Vocabulary Subscale of the Wechsler Adult Intelligence Scale. Single classification analysis of variance indicated no significant differences for either of these variables ($p > .05$). The groups did differ on the Global Assessment Scale (GAS) (Endicott, Spitzer, and Fleiss 1976), both in lowest GAS score ($F = 6.34$, $df = 3/40$, $p < .005$) and overall GAS score ($F = 8.55$, $df = 3/40$, $p < .001$). Table 1 provides the means for age, scaled score on Vocabulary, and GAS scores for the four groups. Newman-Keuls analysis of the lowest GAS score indicated no differences in the level of severity of impairment between the schizophrenics and schizoaffectives. Both of these groups, however, were significantly more impaired than the bipolar disorders ($p < .05$), who in turn were more impaired than the nonpsychotic depressed group. These findings were paralleled by those for the overall GAS score.

Patients were screened for a history of central nervous system (CNS) damage, mental retardation, electroconvulsive therapy, and severe polydrug use. All subjects had at least 20/30 vision.

Apparatus and Stimuli. Stimuli were presented in a Gerbrands four-field tachistoscope. Stimuli were constructed by mounting black paratype (Futura bold No. F29–14) upper case A's and T's on white stimulus cards. With a viewing distance of 78.7 cm, stimuli subtended a visual angle of .20 degrees when presented in the center of the visual field. Five T's and A's were constructed and presented in random order in blocks of 10. The mask consisted of overlapping X's, completely superimposed over the letters. Luminance of the target and masking fields was set at 15 footlamperts. A small dot presented just above the center in field 3, illuminated at 1 footlampert, provided a fixation field.

Procedure. After they provided written informed consent, subjects were escorted to a dimly lit experimental room where they were read a standard set of instructions (Saccuzzo, Hirt, and Spencer 1974). To ensure that subjects were attending to the task, the experimenter preceded the presentation of each stimulus card with the statement, "Ready, look." In phase 1 of the experiment, the minimum exposure duration for criterion identification of the test stimulus without a mask, which Saccuzzo et al. (1979) called the "critical stimulus duration" (CSD), was calculated for each subject. The procedure for calculating the CSD followed that used by Saccuzzo et al. (1979) and Braff and Saccuzzo (1981). Briefly, the target stimulus duration was initially set at 1.0 ms and increased in increments of 1.0 ms for each incorrect response until the subject reached the criterion of seven consecutive correct identifications. The CSD provides a suprathreshold exposure duration (see Saccuzzo and Schubert 1983). Targets were presented at each subject's independently determined CSDs for the masking experiment. There are advantages and disadvantages to the use of a CSD procedure (see Saccuzzo 1981; Stanovich and Purcell 1981; Saccuzzo and Schubert 1983; Schwartz 1983; Long 1984). In this experiment, the advantage of ruling out possible intergroup differences in the ability to identify unmasked targets outweighed the possibility of influencing the masking functions in different ways for different subjects. It is true that in normals longer durations shorten the range of the masking function for any given subject (Turvey 1973). Such findings, however, are based on the unlikely assumption of no individual differences of the strength of a sensory signal for any given duration.

In the present experiment, masking functions were calculated for four intervals between termination of the target and presentation of the mask (i.e., interstimulus intervals). These were 20, 60, 120, and 300 ms. A no-mask control was also used, which is an infinite interval between the two stimuli. For each interval, there were 10 consecutive presentations of the randomly presented T or A. The mask was presented for 50 ms, which provided a mask energy that always exceeded that of the stimulus regardless of a subject's CSD. A typical masking trial was as follows: The subject fixed on the dot in the center of the visual field. The stimulus was presented at the previously determined CSD. There then followed a dark interval at one of the four interstimulus intervals. Then the mask was presented. Subjects reported orally, "T" or "A." If no response was given or if they were not sure, subjects were instructed to guess T or A before the next trial began.

Results

Table 1 presents the mean CSDs for the groups. A single classification analysis of variance revealed no statistically significant differences in these means. Indeed, the range of CSDs for the three psychotic groups was less than 6 ms. However, because the mean of the nonpsychotic depressed group was more than two times smaller than that for the three psychotic groups, a series of individual t tests were conducted in which each group was compared to each of the others. Significant differences were found between the nonpsychotic depressed group and each of the other three ($p < .05$), indicating this group's target exposure duration was indeed below that of the three other groups.

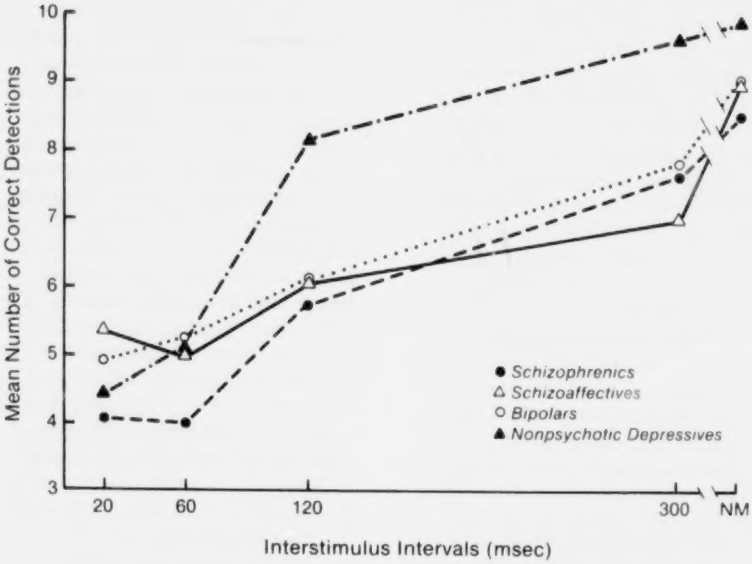
Results for the masking experiment were evaluated in a 4 (Group) × 5 (Interstimulus Interval) repeated measures analysis of variance with repeated measures on the last factor. There were significant main effects for Group ($F = 4.34$, $df = 3/40$, $p < .01$) and Interstimulus Interval ($F = 68.37$, $df = 4/160$, $p < .001$). The Group × Interstimulus Interval interaction was also significant ($F = 1.85$, $df = 12/160$, $p < .05$). Newman-Keuls analysis of the main effects indicated no statistically significant differences among the schizophrenic ($M = 6.00$), schizoaffective ($M = 6.49$), and bipolar ($M = 6.64$) disorders ($p > .05$). All three of these groups, however, were significantly below the nonpsychotic depressed group ($p < .01$). (See figure 1.) Newman-Keuls analysis of the main effect for Interstimulus Interval revealed the typical finding that as the interval between the stimulus and mask increased, so did performance. Newman-Keuls analysis of the interaction indicated that at the 20 ms, 60 ms, and no mask intervals, when subjects were performing approximately at chance or ceiling, none of the groups differed significantly ($p > .05$). At the 120 ms and 300 ms intervals, the depressed group was significantly better than all other groups ($p < .01$), who did not differ significantly from each other.

Discussion

The data revealed that schizophrenic patients could not be distinguished from the schizoaffective and bipolar affective patients on a cross-sectional basis, with all three groups showing evidence of a masking deficit. Consistent with Schubert, Saccuzzo, and Braff (1985), the data indicate an information-processing dysfunction in actively symptomatic schizoaffective and bipolar-disordered patients. These results, in conjunction with our previous findings, are consistent with an interpretation of information-processing dysfunction as both an enduring trait of patients with schizophrenia spectrum disorders and as a state that probably covaries with the presence of thought disorder and psychotic symptoms in psychotically disordered patients.

From a theoretical standpoint, there remains the question of why actively psychotic patients have difficulty with the masking task. One possibility, which we have favored (Saccuzzo, Hirt, and Spencer 1974; Saccuzzo and Braff 1981), is that the poor masking performance reflects abnormally slow information processing. Presumably, the schizophrenic does not rapidly transfer input to more complex cognitive operations that theoretically are dependent on cerebral cortical mechanisms. This information transfer problem results in *underprovision* of accurate information due to temporal processing limitations and ultimately leads to distortions of reality testing. It is also possible, however, as suggested by Knight (1984), that at least some schizophrenics process too much infor-

Figure 1. Backward-masking performance of schizophrenic, schizoaffective, bipolar, and nonpsychotic depressed inpatients



mation because they allocate unnecessary resources to the processing of an irrelevant, noninformation mask. In this case, the problem would be *overprovision* of information, a formulation in accord with impaired filter theories of schizophrenia. A unifying concept is that psychotic states deplete an individual's processing resources, putting an additional strain on an already limited system that leads to impaired performance for all tasks that require the full use of processing capabilities (Nuechterlein and Dawson 1984).

The idea that cognitive dysfunction in schizophrenia is due to alternating underprovision (or underutilization) or to overprovision (or overutilization) of input is consistent with the traditional theories of schizophrenia advocated by Venables (1964), Silverman (1964), McGhie and Chapman (1961), Weckowicz (1958), Yates (1966), and others. Cast in the context of psychophysiological (Venables 1984) and psychopharmacological (Matthysse 1974) formulations, information-processing studies of schizophrenia support a general theory in which poor prognosis/poor premorbid schizophrenics and genetically linked spectrum-disordered patients can be characterized by underprovision or overprovision of cortically dependent operations, caused by an imbalance in normal disinhibitory or inhibitory processes. According to the ideas of Venables (1964) and Mednick (1958), an affected individual might be plagued by overprovision at one point in the disorder that is followed at another point by overcompensation and then underprovision at some later time. The reverse might also be true, with some individuals beginning with an imbalance that leads to underprovision followed by overcompensation. Still others might be afflicted with a lifetime imbalance in only one direction. Unfortunately, such complex and variable formulations seem somewhat inconsistent and are not easily disconfirmed. However, this sort of formulation does lend itself to a nonlinear topographic analysis (Winfree 1983) that may ultimately prove of great utility in analyzing data from schizophrenic-disordered patients. Such nonlinear approaches also encourage researchers to obtain multiple, detailed dependent measures on schizophrenic patients in order to chart the time course of schizophrenic deficits. This type of formulation is quite consistent with Bleuler's (1950) original idea that there is a heterogeneous and complex group of schizophrenias.

Based on the ideas of Meehl (1962) and those of Zubin and Spring (1977), it seems reasonable to assume that an information-processing dysfunction may vary on a continuum of severity across afflicted individuals. If it is extreme, then the impaired processing will almost certainly lead to the development of a thought disturbance. At less severe levels, the processing impairment might affect personality and social functioning but not necessarily, or by itself, cause thought disorder. Individuals whose inhibitory-disinhibitory imbalance leads to information overutilization will manifest hypersensitivity and painful anxiety, especially in social situations. When the imbalance leads to underutilization, the individual becomes grossly insensitive to social stimuli. Missing many of life's subtleties, the individual fails to experience its rewards and there is a strong tendency to social withdrawal. Whether the individual experiences a full-blown schizophrenic disorder depends on a number of factors including the stability of the personality organization that evolves, the flexibility and effectiveness of learned coping strategies, and, as suggested by Zubin and Spring (1977), the severity of the processing impairment in relation to the degree of environmental stress.

When a high-risk (i.e., severely impaired information-processing) individual with an unstable personality organization confronts stressful events that overtax existing coping skills, limited processing resources become further strained. A vicious cycle may ensue in which strained resources further limit the individual's capacity to cope, which, in turn, enhances the impact of the stressful event, causing a still further drain of resources. Eventually the individual becomes overwhelmed. Consciousness either spills over with an uncontrollable flood of input or becomes brittle and fragmented. And when the episode is over—the major symptoms brought under control by medication and therapy or by natural cyclic remission, the individual's processing capabilities revert to their previous marginal levels while the sequelae of a schizophrenic episode may linger on. Once this path has been traveled, it is somehow facilitated so that it becomes more easily followed when the next processing storm occurs.

Current Status of the Icon and Visual Masking Mechanisms. Interpreting the results of visual information-processing experiments is far from easy. Allik and Bachmann (1983) have pointed out that 50 to 100 experiments are published each month about visual information storage, iconic memory, visual persistence, and related issues. Since Sperling (1960) originally argued that there is a large capacity, short duration store of visual information, thousands of studies have been completed on the issue of initial visual storage as high capacity, evanescent, information store, termed iconic storage by Neisser (1967). Related to iconic storage, the phenomenon of visual persistence has been unequivocally demonstrated, but the theoretical significance and anatomical locus of such persistence has been debated, dissected, and disputed. Is there a retinally or centrally based informational store? Is there a need to deify such a storage capacity with a special place in a sequential information-processing theory? After hundreds of studies and countless hours of debate, researchers have reached no clear consensus about this issue. Haber (1983) has commented extensively on the "demise of the icon" and the "sharp empirical debates" that this issue has engendered. To illustrate the sheer weight of this literature, Haber (1983) notes that Long (1980) and Coltheart (1980) have produced two lengthy reviews of over 60,000 words and that neither of these authors "ever raises any of the questions or concerns posed by theorists such as Neisser, Hochberg, Eriksen and Schultz or Turvey" (Haber 1983, p. 3). Haber's 11-page article received "open peer commentary" from *32* authors, and there was wide disagreement about the utility and accuracy of theories of iconic memory. If the issue of iconic memory is controversial when measured in small groups of normal subjects, this complexity is greatly increased when a masking paradigm is used in groups of psychopathological subjects.

Still, we can summarize the state of the literature in this area:

- A visual stimulus creates a retinal response that decays over time.
- This retinal response has central correlates.
- The visual response *persists* after stimulus termination, and this persistence probably has retinal and central components.
- The visual response can be analyzed for information content and also is subject to interference by masking.
- Theories of iconic memory as a static, purely retinally based "photograph" are outdated and not particularly useful.

Unfortunately, this evaluation leads to a more complicated question. How does the mask act in a visual masking paradigm? Is its major effect retinal or central? Is the masking mechanism integrative or interruptive? The answer is that masking can act retinally or centrally. It also can probably act via the "merging" of target and mask stimuli (integration) or via the interruption of target processing via the action of the mask (interruption). All of these mechanisms are possible, depending on the specific conditions of the experiment (Felsten and Wasserman 1980).

Visual Processing in Schizophrenic Patients. For our purposes, we should examine the conditions of our typical visual masking paradigm using schizophrenic patients. In our studies of psychopathological subjects, we have used a dark adaptation period that tends to increase the duration of visual persistence to stimuli. We have also used a target duration (whether fixed for all subjects or at the CSD) close to the visual identification threshold and a powerful (bright and long duration) mask to limit visual persistence and to control the pulse duration of information delivered to the subject. Under these conditions, we initially felt that at longer ISIs (> 100 ms), masking acts through central interruptive mechanisms. According to this formulation, the masking paradigm performance deficits of schizophrenic patients reflect a slowness of information processing. In addition some schizophrenics, as we have noted, also show deficient processing of unmasked stimuli, as revealed by increased CSDs (Saccuzzo, Hirt, and Spencer 1974; Braff and Saccuzzo 1981). Initially we interpreted such CSD findings as evidence for a deficit in input capabilities in some schizophrenics. Additional studies have not definitively resolved this issue.

Knight and colleagues (e.g., Knight, Sherer, and Shapiro 1977; Knight and Sims-Knight 1980) used a picture integration technique and concluded that schizophrenics had normal information-processing functions in the first 100 ms following stimulus input. Using a dot integration procedure, Spaulding et al. (1980) reached similar conclusions. In related work, however, Schwartz, Winstead, and Adinoff (1983) specifically evaluated speed of information processing in schizophrenic patients and normal controls using a backward-masking paradigm with varying target stimulus durations and other paradigmatic manipulations to evaluate iconic and visual persistence factors. They concluded that the retarded processing shown by schizophrenics is due, at least in part, to visual signal abnormalities caused by an instability or a slower than normal rate of decay of visual stimuli. Schwartz, Winstead, and Adinoff (1983) interpret their findings as suggesting that temporal integration is defective in schizophrenia, perhaps due to increased target stimulus visual persistence. Thus, the status of information processing at the icon level has yet to be fully understood.

With debates raging over the theoretical interpretations of iconic and masking factors, a relatively atheoretical and clear descriptive approach seems warranted. Varying interpretations of masking experiments abound. For example, it is quite clear that if schizophrenics show deficits in visual persistence, their masking deficits may occur via integrative (versus interruptive) mechanisms at retinal or CNS levels. In normals, there is evidence that masking by integration yields complex stimuli that register event related potentials (ERPs) intermediate between target and mask ERPs (Schwartz, Whittier, and Schweitzer 1979). This "mixed" ERP occurs even when the target cannot be consciously identified. When a *second* mask is used, the original mask becomes ineffective and the target is once again correctly identified, but the ERP to the target-mask stimulus is unchanged. Perhaps the defect shown by schizophrenics in the visual masking paradigm results from their inability to screen out the mask features from the target-mask stimulus and to identify the critical target-identifying features from the montage of a fused or complex target-mask stimulus. The physiological basis for this phenomenon would be slowed rates of resolution of the visual response to the target and mask stimuli. Thus, the visual information-processing data in schizophrenics could be reinterpreted along the lines of impaired filters as well as slowness of processing.

Lest we confound issues further, suffice it to say that the visual masking paradigm has usefully and reliably identified a time-linked deficit in schizophrenic patients. This deficit occurs in acutely psychotic schizophrenics and seems to persist across time in poor prognosis schizophrenics, in schizotypal patients, and so-called vulnerable but asymptomatic schizotypes identified by 2-7-8 MMPI profiles. In this current study, the visual-masking abnormality is reported to be state as well as trait dependent. Schizoaffective and thought-disordered bipolar patients show visual processing deficits similar to those of schizophrenics. These results support the idea of both state- and trait-dependent deficits in visual information processing by schizophrenic and other psychotic patients.

Related Time-Linked Findings. It is important to note that various information-processing paradigms have identified schizophrenic patients' deficits in the epoch of 500 ms after stimulus presentation (Braff and Saccuzzo 1984). Thus, a number of related paradigms, using auditory and visual processing systems, show strikingly similar time-linked deficits in schizophrenia spectrum patients (Braff 1985). For example:

- Braff, Callaway, and Naylor (1977) examined self-stimulated auditory ERPs where the subject triggers the ERP-eliciting stimulus after a delay. Normally, ERP amplitude is inhibited in the self-stimulated versus randomly presented conditions. Schizophrenics, though, seemed to show loss of normal amplitude inhibition in the 250 ms "ISI" condition (the ISI being the delay between key release and stimulus presentation). These results were interpreted as supporting a loss of normal inhibitory or sensory gating influences in information processing in schizophrenic patients.
- Freedman's group has used an ERP paradigm that reflects sensory gating. In a series of systematic and revealing studies, this group has

examined the ERP P_{50} wave to paired stimuli with a 500 ms conditioning-testing ISI. The second stimulus-elicited P_{50} wave is normally inhibited by the effects of the first stimulus. Schizophrenic patients and their first degree relatives share a loss of normal inhibitory influences as reflected by increased P_{50} waves to the second stimulus (Adler et al. 1982; Freedman et al. 1983; Siegel et al. 1984). Interestingly, Freedman's group also reports that this gating or inhibitory abnormality seems related to a trait-dependent schizotaxic factor and a transient, state-dependent factor that occurs in bipolar manic patients (Franks et al. 1983). These results provide a striking parallel to the visual masking trait/state formulation. Adler, Rose, and Freedman (1985) have also recently reported success in creating an animal (rodent) model of their P_{50} gating paradigm which will allow this group to test specific neurobiological hypotheses about the observed P_{50} abnormalities.

- A third related line of evidence utilizing human and animal data in the 50 to 500 ms interval following stimulus input has evolved from experiments using the prepulse inhibition (PPI) of the acoustic startle response (ASR). This paradigm uses the blink reflex (in humans) or whole body startle (in rats) to strong auditory stimuli. Normally, weak prestimulation at certain ISIs inhibits the ASR amplitude in normal humans. Braff et al. (1978) reported the loss of this normal prepulse inhibitory (PPI) effect in the 60 ms ISI condition in a group of schizophrenic patients. This time-linked deficit is parallel to the findings of Freedman and associates. To assess the possibility that this loss of PPI is due to dopaminergic dysfunction, Swerdlow et al. (1986), using stimulus parameters similar to those used in humans, stimulated rats' supersensitive dopamine receptors with low-dose apomorphine and found a loss of normal PPI of the ASR with mesolimbic (nucleus accumbens) but not frontal cortical supersensitive dopamine stimulation. This result was also time dependent and loss of normal inhibition was maximal in the 60 and 120 ms ISI conditions. These findings added significant support to the idea that the observed time-linked abnormalities in schizophrenic performance may well be linked to the putative underlying increase in mesolimbic dopaminergic tone in schizophrenic patients.

- There are a host of other, less time-linked deficits in auditory and visual information processing in schizophrenic patients. These include deficits in tests of distractibility (Oltmanns and Neale 1975), dichotic listening (Straube and Germer 1979), the continuous performance task (CPT) (Wohlberg and Kornetsky 1973), skin conductance orienting response (SCOR) (Spohn and Patterson 1979), startle response habituation (Geyer and Braff 1982), smooth pursuit eye movements (SPEM) (Holzman, Levy, and Proctor 1976), conventional cortical ERPs (Shagass 1976), and other measures (see Braff 1985). All these experiments reveal deficits in the ability of schizophrenic patients to maintain normal attentional and information-processing functions. These results are compatible with the idea that the information-processing deficit accrues in schizophrenia from an inability to process stimuli serially. None of these approaches are as time-based specific as are the visual masking, ERP, P_{50}, and ASR studies cited above, but future research may well examine the interrelationships of these attention-dependent measures in schizophrenic patients. We may also see the effects of increasing dopaminergic tone and of using antipsychotic medications on the information-processing performance of schizophrenic patients.

Summary

In sum, the viability of an information-processing approach to schizophrenia and psychotic illnesses is supported by numerous interlocking studies, such as those involving visual masking. While a complete model has yet to be developed, the information-processing perspective provides a way of conceptualizing trait/state and other major theoretical formulations of the psychological and neurobiological deficits in the group of schizophrenias and related psychotic states.

References

Adler, L.E.; Pachtman, E.; Franks, R.D.; Pecevich, M.; Waldo, M.C.; and Freedman, R. Neurophysiological evidence for a defect in neuronal mechanisms involved in sensory gating in schizophrenia. *Biological Psychiatry*, 17:639–654, 1982.

Adler, L.E.; Rose, G.M.; and Freedman, R. Sensory gating in rats: Parallels with psychosis. *New Research Program & Abstracts*, American Psychiatric Association Meeting, 1985. p. 28.

Allik, J., and Bachmann, T. How bad is the icon? *Behavior and Brain Science*, 6:12–13, 1983.

Asarnow, R.F., and MacCrimmon, D.J. Residual performance deficits in clinically remitted schizophrenics: A marker of schizophrenia? *Journal of Abnormal Psychology*, 87:597–608, 1978.

Asarnow, R.F.; Steffy, R.A.; MacCrimmon, D.J.; and Cleghorn, J.M. An attentional assessment of foster children at risk for schizophrenia. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment*. New York: John Wiley & Sons, 1978. pp. 339–358.

Balogh, D.W., and Merritt, R.D. Susceptibility to type A backward pattern masking among hypothetically psychosis-prone college students. *Journal of Abnormal Psychology*, in press.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias*. (1911) Translated by J. Zinkin. New York: International Universities Press, Inc., 1970.

Braff, D.L. Impaired speed of information processing in nonmedicated schizotypal patients. *Schizophrenia Bulletin*, 7:499–508, 1981.

Braff, D.L. Attention, habituation and information processing in psychiatric disorders. In: *Psychiatry III*. Philadelphia: Lippincott Co., 1985. pp. 1–12.

Braff, D.L.; Callaway, E; and Naylor, H. Very short term memory (VSTM) in schizophrenia. *Archives of General Psychiatry*, 34:25–30, 1977.

Braff, D.L., and Saccuzzo, D.P. Information processing dysfunction in paranoid schizophrenia: A two factor deficit. *American Journal of Psychiatry*, 138:1051–1056, 1981.

Braff, D.L., and Saccuzzo, D.P. The effects of antipsychotic medication on speed of information processing in schizophrenic patients. *American Journal of Psychiatry*, 139:1127–1130, 1982.

Braff, D.L.; Stone, C.; Callaway, E.; Geyer, M.; Glick, I.; and Bali, L. Prestimulus effects on human startle reflex in normals and schizophrenics. *Psychophysiology*, 15:339–343, 1978.

Brody, D.; Saccuzzo, D.P.; and Braff, D.L. Information processing for masked and unmasked stimuli in schizophrenia and old age. *Journal of Abnormal Psychology*, 89:617–622, 1980.

Callaway, E., and Naghdi, S. An information processing model for schizophrenia. *Archives of General Psychiatry*, 39:339–347, 1982.

Coltheart, M. Iconic memory and visual persistence. *Perception and Psychophysics*, 27:183–228, 1980.

Endicott, J.; Spitzer, R.L.; and Fleiss, J.L. The Global Assessment Scale: A procedure for measuring overall severity of psychiatric disturbance. *Archives of General Psychiatry*, 33:766–771, 1976.

Franks, R.; Adler, L.E.; Waldo, M.; Alpert, J.; and Freedman, R. Neurophysiological studies of sensory gating in mania: Comparison with schizophrenia. *Biological Psychiatry*, 18:989–1005, 1983.

Felsten, G., and Wasserman, G.S. Visual masking: Mechanisms and theories. *Psychological Bulletin*, 88:329–353, 1980.

Freedman, R.; Adler, L.E.; Waldo, M.C.; Pachtman, E.; and Franks, R.D. Neurophysiological evidence for a defect in inhibitory pathways in schizophrenia: Comparison of medicated and drug-free patients. *Biological Psychiatry*, 18:537–551, 1983.

Geyer, M.A., and Braff, D.L. Habituation of the blink reflex in normals and schizophrenics. *Psychophysiology*, 19:1–6, 1982.

Green, M., and Walker, E. Susceptibility to backward masking in schizophrenic patients with positive or negative symptoms. *American Journal of Psychiatry*, 141:1273–1275, 1984.

Gur, R.E. Motoric laterality imbalance in schizophrenia: A possible concomitant of left hemisphere dysfunction. *Archives of General Psychiatry*, 44:33–37, 1977.

Gur, R.E. Left hemisphere dysfunction and left hemisphere overactivation in schizophrenia. *Journal of Abnormal Psychology*, 87:226–238, 1978.

Haber, R.H. The impending demise of the icon: A critique of the concept of iconic storage in visual information processing. *The Behavioral and Brain Sciences*, 6:1–11, 1983.

Heston, L.L. Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry*, 112:819–825, 1966.

Heston, L.L. The genetics of schizophrenic and schizoid disease. *Science*, 167:249–256, 1970.

Holzman, P.; Levy, D.; and Proctor, L. Smooth pursuit eye movements, attention, and schizophrenia. *Archives of General Psychiatry*, 33:1415–1420, 1976.

Kahneman, D. Method, findings, and theory in studies of visual masking. *Psychological Bulletin*, 70:404–425, 1968.

Kietzman, M.L.; Spring, B.; and Zubin, J. Science of human behavior: Contributions of the psychological sciences. In: Kaplan, H.L.; Freedman, A.M.; and Sadock, B.W., eds. *Comprehensive Textbook of Psychiatry III*. Baltimore: Williams & Wilkins Co., 1980. pp. 334–371.

Kietzman, M.L., and Zubin, J. Sensory and perceptual behavioral markers in schizophrenia. *Psychopharmacology Bulletin*, in press.

Knight, R.A. Converging models of

cognitive deficit in schizophrenia. In: Spaulding, W.D., and Cole, J.K., eds. *Nebraska Symposium on Motivation, 1983: Theories of Schizophrenia and Psychosis.* Lincoln: University of Nebraska Press, 1984. pp. 93-156.

Knight, R.A.; Sherer, M.; and Shapiro, J. Iconic imagery in overinclusive and nonoverinclusive schizophrenics. *Journal of Abnormal Psychology,* 86:242-255, 1977.

Knight, R.A., and Sims-Knight, J.E. Integration of visual patterns in schizophrenia. *Journal of Abnormal Psychology,* 89:623-634, 1980.

Long, G.M. Iconic memory: A review and critique of the study of short-term visual storage. *Psychological Bulletin,* 88:785-820, 1980.

Long, G.M. The pretreatment of weighting technique. *Journal of Experimental Psychology: Human Perception and Performance,* 10:140-143, 1984.

Magaro, P.A. *Cognition in Schizophrenia and Paranoia: The Integration of Cognitive Processes.* Hillsdale, NJ: Lawrence Erlbaum, 1980.

Matthysse, S. A theory of the relation between dopamine and attention. In: Wynne, L.C.; Cromwell, R.L.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment.* New York: John Wiley & Sons, 1978. pp. 307-310.

McGhie, A., and Chapman, J. Disorders of attention and perception in early schizophrenia. *British Journal of Medical Psychology,* 34:102-116, 1961.

Mednick, S.A. A learning theory approach to research in schizophrenia. *Psychological Bulletin,* 55:316-327, 1958.

Meehl, P.E. Schizotaxia, schizotypy, schizophrenia. *American Psychologist,* 17:827-838, 1962.

Merritt, R.D., and Balogh, D.W. The use of a backward masking paradigm to assess information-processing deficits among schizotypics: A reevaluation of Steronko and Woods. *Journal of Nervous and Mental Disease,* 172:216-224, 1984.

Miller, S.; Saccuzzo, D.P.; and Braff, D.L. Information processing deficits in remitted schizophrenics. *Journal of Abnormal Psychology,* 88:446-449, 1979.

Nakano, K., and Saccuzzo, D.P. Schizotaxia, visual information-processing, and the MMPI 2-7-8 code type. *British Journal of Clinical Psychology,* in press.

Neisser, U. *Cognitive Psychology.* New York: Appleton-Century-Crofts, 1967.

Nuechterlein, K.H., and Dawson, M.E. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin,* 10:160-203, 1984.

Oltmanns, T., and Neale, J. Schizophrenic performance when distractors are present: Attention deficit or differential task difficulty? *Journal of Abnormal Psychology,* 34:205-209, 1975.

Payne, R.W. The measurement and significance of overinclusive thinking and retardation in schizophrenic patients. In: Hoch, P., and Zubin, J., eds. *Psychopathology of Schizophrenia.* New York: Grune & Stratton, 1966.

Saccuzzo, D.P. Bridges between schizophrenia and gerontology: Generalized or specific deficits. *Psychological Bulletin,* 84:595-600, 1977.

Saccuzzo, D.P. Input capability and speed of processing in mental retardation: A reply to Stanovich and Purcell. *Journal of Abnormal Psychology,* 90:172-174, 1981.

Saccuzzo, D.P. An information-processing interpretation of theory and research in schizophrenia. In: Ingram, R., ed. *Information Processing Approaches to Psychopathology.* New York: Academic Press, in press.

Saccuzzo, D.P., and Braff, D.L. Early information processing deficits in schizophrenia: New findings using schizophrenic subgroups and manic controls. *Archives of General Psychiatry,* 38:175-179, 1981.

Saccuzzo, D.P.; Braff, D.L.; and Sprock, J. The effect of mental retardation and schizophrenia on information-processing. *Journal of Nervous and Mental Disease,* 170:102-106, 1982.

Saccuzzo, D.P.; Braff, D.L.; Sprock, J.; and Sudik, N. The schizophrenia spectrum: A study of the relationship among the Rorschach, MMPI, and visual backward masking. *Journal of Clinical Psychology,* 40:1288-1294, 1984.

Saccuzzo, D.P.; Hirt, M.; and Spencer, T.J. Backward masking as a measure of attention in schizophrenia. *Journal of Abnormal Psychology,* 83:512-522, 1974.

Saccuzzo, D.P.; Kerr, M.; Marcus, A.; and Brown, R. Visual information processing in mental retardation. *Journal of Abnormal Psychology,* 88:341-345, 1979.

Saccuzzo, D.P., and Michael, B. Speed of information-processing and structural limitations in retarded and dual diagnosed retarded-schizophrenic persons. *American Journal of Mental Deficiency,* 89:487-495, 1984.

Saccuzzo, D.P., and Miller, S. The critical interstimulus interval in delusional schizophrenics and normals. *Journal of Abnormal Psychology*, 86:261–266, 1977.

Saccuzzo, D.P., and Schubert, D.L. Backward masking as a measure of slow processing in schizophrenic spectrum disorders. *Journal of Abnormal Psychology*, 90:305–312, 1981.

Saccuzzo, D.P., and Schubert, D.L. Information-processing in the schizophrenia spectrum: A reply to Schwartz. *Journal of Abnormal Psychology*, 92:503, 1983.

Sagi, D., and Julesz, B. "Where" and "what" in vision. *Science*, 228: 1217–1219, 1985.

Schubert, D.L.; Saccuzzo, D.P.; and Braff, D.L. Information-processing in borderline patients. *Journal of Nervous and Mental Disease*, 173:26–31, 1985.

Schwartz, B. Backward masking as a measure of slow processing in the schizophrenia spectrum: A reply to Saccuzzo and Schubert. *Journal of Abnormal Psychology*, 92:504, 1983.

Schwartz, B.D.; Winstead, D.K.; and Adinoff, B. Visual processing deficits in acute and chronic schizophrenics. *Biological Psychiatry*, 17:1377–1387, 1983.

Schwartz, M.; Whittier, O.M.; and Schweitzer, P.K. Evoked responses in retroactively masked stimuli. *Physiological Psychology*, 7:107–111, 1979.

Shagass, C. An electrophysiological view of schizophrenia. *Biological Psychiatry*, 11:3–29, 1976.

Siegel, C.; Waldo, M.; Mizner, G.; Adler, L.E.; and Freedman, R. Deficits in sensory gating in schizophrenic patients and their relatives. *Archives of General Psychiatry*, 41:607–612, 1984.

Silverman, J. The problem of attention in research and theory in schizophrenia. *Psychological Review*, 71:352–379, 1964.

Spaulding, W.; Rosenzweig, L.; Huntzinger, R.; Cromwell, R.L.; Briggs, D.; and Hayes, T. Visual pattern integration in psychiatric patients. *Journal of Abnormal Psychology*, 89:635–643, 1980.

Sperling, G. The information available in a brief visual presentation. *Psychological Monographs*, 74 (11, Whole No. 498), 1960.

Spitzer, R.L.; Endicott, J.; and Robins, E. *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders*. 3rd ed. New York: New York State Psychiatric Institute, 1977.

Spohn, H., and Patterson, T. Recent studies of psychophysiology in schizophrenia. *Schizophrenia Bulletin*, 5:581–611, 1979.

Stanovich, K.E., and Purcell, D.G. Comment on "Input Capability and Speed of Processing in Mental Retardation" by Saccuzzo, Kerr, Marcus, and Brown. *Journal of Abnormal Psychology*, 90:168–171, 1981.

Steronko, R.J., and Woods, D.J. Impairment in early stages of visual information processing in nonpsychotic schizotypic individuals. *Journal of Abnormal Psychology*, 87:481–490, 1978.

Straube, E.R., and Germer, C.K. Dichotic shadowing and selective attention to word meaning in schizophrenia. *Journal of Abnormal Psychology*, 88:346–353, 1979.

Swerdlow, N.R.; Braff, D.L.; Geyer, M.A.; and Koob, G.F. Central dopamine hyperactivity in rats mimics abnormal acoustic startle response in schizophrenics. *Biological Psychiatry*, 21:23–33, 1986.

Turvey, M.T. On peripheral and central processes in vision: Inferences from an information-processing analysis of masking with patterned stimuli. *Psychological Review*, 80:1–52, 1973.

Venables, P.H. Input dysfunction in schizophrenia. In: Maher, B.A., ed. *Progress in Experimental Personality Research*, Vol. I. New York: Academic Press, 1964. pp. 1–47.

Venables, P.H. Input regulation and psychopathology. In: Hammer, M.; Salzinger, K.; and Sutton, S., eds. *Psychopathology: Contributions From the Social, Behavioral, and Biological Sciences*. New York: John Wiley & Sons, 1973. pp. 261–285.

Venables, P.H. Cerebral mechanisms, autonomic responsiveness, and attention in schizophrenia. In: Spaulding, W.D., and Cole, J.K., eds. *Nebraska Symposium on Motivation, 1983: Theories of Schizophrenia and Psychosis*. Lincoln: University of Nebraska Press, 1984. pp. 47–91.

Weckowicz, T.E. Autonomic activity as measured by the mecholyl test and size constancy in schizophrenic patients. *Psychosomatic Medicine*, 20:66–71, 1958.

Weckowicz, T.E., and Blewett, D.B. Size constancy and abstract thinking in schizophrenic patients. *Journal of Mental Science*, 105:909–934, 1959.

Winfree, A.T. Sudden cardiac death: A problem in topology. *Scientific American*, 248(5):144–161, 1983.

Wohlberg, G., and Kornetsky, C. Sustained attention in remitted schizophrenics. *Archives of General Psychiatry*, 28:533–537, 1973.

Yates, A.J. Psychological deficit. *Annual Review of Psychology*, 17:111–144, 1966.

Zubin, J., and Spring, B. Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, 86:102–126, 1977.

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Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work, in cooperation with several community and mental health organizations, has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside."

For more information about the rental or purchase of these videotapes, please contact: **Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.**

Thought Disorder in Schizophrenia: Cognitive and Neuroscience Approaches

by Terry Patterson, Herbert E. Spohn, Douglas P. Bogia, and Kenneth Hayes

Abstract

Research with schizophrenics is reviewed in the areas of the startle reflex, subject-initiated evoked potentials, and two-stimulus evoked potentials. A hypothesis of an early (pre-100 ms) information-processing deficit is posited and further refined by results presented from brain evoked potentials during a backward-masking experiment. The implications of these findings for the speech component of schizophrenic thought disorder are speculated upon, with specific reference to failures of temporal sequencing control that may underlie schizophrenic speech dysfunction and may be attributable to defective thalamic control. Recent neuroanatomical findings of abnormalities in the hippocampus and thalamus of schizophrenics are presented, and considered as likely contributors to the early information-processing deficits posited.

A defect may exist in the early (less than 100 ms) information-processing ability of schizophrenics. This hypothesis, we believe, is supported by preliminary findings (reported below) from our work with topographical mapping of the brain electrical activity observed during a backward-masking experiment with schizophrenics and controls. Further support for this hypothesis may be found in paradigms that have explored the startle reflex, subject-initiated evoked potentials, and two-stimulus evoked potentials in schizophrenics. This hypothesized defect may constitute a fundamental element in schizophrenic thought disorder when viewed from the perspective of current models of information processing. It may explain certain aspects of dysfunctional speech in schizophrenics.

Johnson (1985) has advanced a hypothesis of an early information-processing defect in schizophrenia. While our hypothesis is similar, it is elaborated somewhat by inclusion of brain potentials in backward masking and by consideration of recent neuroanatomical and electrophysiological findings which allow hypotheses to be formulated concerning the brain mechanisms that may underlie the early information-processing defect.

A formal theoretical framework for information processing (from the psychological or cognitive science perspective) dates back to Broadbent (1958). In simplistic terms, this theoretical framework views information processing in the brain as taking place in a series of somewhat discrete stages. An initial sensory store (sometimes called the "icon" store or "iconic" image) is posited to last at most a second and, more likely, a few hundred milliseconds. This stage is believed to have a vast storage capacity but very limited duration. A subset of this information is thought to be extracted from the sensory store, transformed into a short-term memory store, and relayed through a central processing stage at which point decisions are made about the degree of further processing needed for the new information. The central processing stage is believed to be the point at which resource allocation is made toward further information processing. One of the resources available through the hypothetical central processor is the lexicon or language faculty. Up to this point we are describing mostly the "input" stage of information processing. The "output" stage in information-processing theory is

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usually referred to as "response organization" and is conceptualized to follow decisions as to the category membership of the information and the response requirements pertinent to it. It is implicitly assumed that the response requirement (which may reflect the current "state" of the organism) feeds forward in time to determine which features will be extracted from the next sensory store. Thus, a dynamic and on-going information processing system is in perpetual operation, sensitive to and interactive with the needs of the organism and changes in the environment.

This account of information-processing theory is highly oversimplified, and the concept of an "iconic" stage has been strongly criticized as unnecessary by Haber (1983), although this view is clearly not shared by some other investigators in their commentary on Haber (1983). It is both impractical and unnecessary for our purposes, in this article, to delve further into this controversy, but it is worthwhile to note that Broadbent's (1984) reformulation of his earlier outlook still includes the construct of a sensory store which possesses many of the attributes ascribed to the "icon" above. Perhaps the most important conceptualization which we would like to adduce is that information may be processed in brains (in the cognitive sense) by a process of changing one form of representational code into another form of representational code. In all likelihood, the processes whereby the code is *represented* are different from the processes whereby the code is *changed* from one form to another (see Broadbent 1984, for further discussion of this point).

For many years the language and concepts of information-processing theory have been used to "dissect" the cognitive abilities of schizophrenics, and the process continues with some controversy. Thus, deficits have been posited at the level of the sensory store or the speed/efficiency of feature extraction from it (Saccuzzo, Hirt, and Spencer 1974; Braff, Callaway, and Naylor 1977; Saccuzzo and Schubert 1981; Braff and Saccuzzo 1985). Other theorists have attributed schizophrenic information-processing deficits to slowed central processing (Yates 1966; Yates and Korboot 1970) or to failures in response organization (Hemsley and Richardson 1980). It is, of course, perfectly possible that defects in schizophrenic information processing may be manifest at each of these stages, but from our point of view, the intactness or otherwise of the earliest stages must be fully ascertained, because defects at this point may well also account for later (e.g., response organization) deficits. This proposal is justified in that information-processing theory posits to some considerable extent a "serial" process. It is therefore a logical corollary of this theoretical framework that an inadequately constituted sensory code or feature extraction from it will result in a short-term memory (or working memory—Broadbent 1984) that is an inadequate representational code of the stimulus events and their potential significance or response organization demands.

Adler et al. (1982) and Freedman et al. (1983) have reported data which indicate that when two auditory (click) stimuli are presented with an interstimulus interval (ISI) varying from 500 ms to 2 seconds, the response of normal subjects is to attenuate the P_{50} component of the evoked potential (EP) to the second stimulus. The amount of attenuation varies from 90 percent at 500 ms (ISI) to 30–50 percent at 2 seconds (ISI). Schizophrenics, on the other hand, do not attenuate the P_{50} component by more than 15 percent at 500 ms (ISI) and may even show an increased amplitude to the second stimulus at 2 seconds (ISI). The authors of these reports have argued strongly for an interpretation that involves defective input or "gating" control of structures which should selectively attenuate input to the cortex. However, these data may also speak to the concept of defective control of temporal sequencing and integration in the brains of schizophrenics. If we posit (as do Adler et al. 1982, and Freedman et al. 1983) that the attenuation of aspects of brain response to secondary input following a primary event is a "normal" function in the processing of information, then one possible implication of such a formulation is that the brain structures which control such events are acting like the absolute and relative refractory periods which follow the discharge of single neuronal cells. The further implication is that in schizophrenics, failures in inhibitory processes which allow for such refractoriness are implicated in early information processing. If we restate the argument in terms of its consequences for schizophrenia, we can hypothesize a situation in which the processing of a primary event is "overwritten" by a secondary input because of a failure to attenuate the strength of the afferent volley generated by the second event.

Such a formulation has important implications for hypotheses concerning the malfunction of early "preattentive" store in schizophrenia. It suggests that the actual *formation of the sensory store* could be defective apart from additional malfunctions in feature extraction from it. In short, the data of Adler et al. and Freedman et al. mentioned above could indicate some malfunction in the "time constant" with which information is handled by the schizophrenic brain. This conceptualization is also consistent with previous work by Shagass (1976) and Shagass et al. (1979), who observed enlarged early components of EPs in schizophrenics to be followed by diminished later components. A plausible explanation offered by these investigators is that inadequate attenuative processing is occurring in the early handling of information in the schizophrenic brain and that this stage is followed by an active inhibitory process on the part of the patient to reduce the consequences of this early failure. However, as the later components of the EP are indicative of higher order categorical decision process and response organization stages, then it is highly likely that the schizophrenic is sacrificing the richness of later stage information processing in an attempt to compensate for early registrative malfunction.

The startle reflex, which has been studied in both human and nonhuman species, is also of relevance to our discussion for two reasons. First, it is organized at a level below the cortex and involves the lateral tegmental area of the midbrain reticular formation (Leitner et al. 1981) and, more particularly, the nucleus reticularis pontis caudalis (Leitner, Powers, and Hoffman 1980). Second, it displays modification by a weak "leading" stimulus which precedes the startle-evoking stimulus (Graham 1975; Hoffman and Ison 1980). The leading stimulus is most effective in reducing the startle response in human subjects if it precedes the startle-producing stimulus by about 100 ms.

This reduction, however, is not so great in schizophrenics as in controls (Braff et al. 1978) and, as such, may reflect temporal sequencing failure in schizophrenics, as hypothesized above, but involving in this instance primarily lower brain structures. The implications for schizophrenia are that available information cannot be temporally organized to protect the schizophrenic from the startling effects of subsequent stimulus events.

> There is a short time constant information processing system acting up to 240 ms intervals which is associated with blink amplitude inhibition and latency facilitation. This system is thought to serve a preattentive filtering function by allowing rapid information processing. Since blink (and startle) reactions are inhibited by antecedent stimulation, they are less distracting and more quickly elicited (latency facilitation), allowing for rapid processing of stimuli. The deficit we see in schizophrenics is consistent with a dysfunction in this early protective mechanism which would correlate with information overload and subsequent cognitive disruption in schizophrenia. Braff et al. (1977) have described "over-reactivity" with cortical evoked potentials in similarly brief interstimulus intervals. The two findings are syntonic: when perceptual filters are impaired, as indexed by either blink reflexes or evoked potentials, there is a tendency to be flooded by environmental cues. [Braff et al. 1978, p. 343]

The EP findings of Braff, Callaway, and Naylor (1977) are also of importance to our discussion. Schafer and Marcus (1973) demonstrated that normal subjects showed a reduction of EP amplitude when they were allowed to initiate the onset of the EP stimulus themselves. This reduction in amplitude still held with delays of up to 4 seconds between subject initiation of the stimulus and its occurrence. Braff, Callaway, and Naylor (1977) showed that the amplitude reduction effect was less adequately displayed by schizophrenics with short delays of 250 ms. Again, these data indicate that short time constant information handling is defective in schizophrenics, who appear to need a longer interval to organize accommodations to the strength of incoming information.

Data from the startle reflex and self-stimulation EP paradigm cited above have been combined with the work of Adler et al. (1982) and Freedman et al. (1983) in a most thought-provoking paper by Johnson (1985). It is Johnson's conclusion that there is an overactivation of sensory input in schizophrenia which is directly due to the failure of an inhibitory mechanism, some of the attributes of which he described as follows:

> it is proposed that a mechanism exists which will respond with inhibition to any small change in stimulus intensity, the amount of inhibition corresponding to the amount of change. The inhibition begins within 100 ms of the change and continues for more than two seconds. It attenuates arousal created by external stimulation during this time, at an early or low level in the brain, before the activity has been relayed to higher neural systems. [Johnson 1985, p. 432]

Much of the data mentioned above might be accounted for *if* in some fashion a primary stimulus fails to "engage the attention" of the schizophrenic. Thus, the leading stimulus in the startle paradigm carries rapidly processable data (to the normal brain) that a startling event will take place. We suggest that in the schizophrenic brain the significance of the primary stimulus is either lost or the information it conveys cannot be used in time to mobilize adequate defenses. In Schneider and Shiffrin's (1977) formulation, this may indicate that an "automatic" (i.e., fast and involving minimal cognitive effort) information-processing system is available to the normal subjects but not available to the schizophrenics, who must rely on a slower and more cognitively effortful "controlled" information processing. In short, the primary stimulus event engages some form of attentional process in normals but fails to do so in schizophrenics.

Such a conceptualization might also be applied to the Adler et al. (1982) and Freedman et al. (1983) paradigms with the following results: If the first stimulus engages (in an "automatic" manner) the attention of the normals, then the attenuation of the P_{50} EP component to the second stimulus event may indicate a protective inhibition that will ensure adequate processing of the primary event. In the schizophrenics, however, if the attentional engagement did *not* take place, then there would be no need for an attenuation of the EP events associated with the second stimulus, leading to the observed results.

A subtle but perhaps important question raised by these studies is the distinction between the early engagement of attentional processes (and therefore the speed and accuracy with which the early sensory store is formed and information extracted from it) versus the vulnerability of the early sensory store to disruption and distortion by subsequent stimulus events. To examine this question, we have measured the EPs associated with performance on a backward-masking task. The backward-masking paradigm by which a brief stimulus event is presented to a subject (and its afterimage obliterated) has consistently shown a schizophrenic deficit (Saccuzzo, Hirt, and Spencer 1974; Braff, Callaway, and Naylor 1977; Saccuzzo and Schubert 1981; Braff and Saccuzzo 1985).

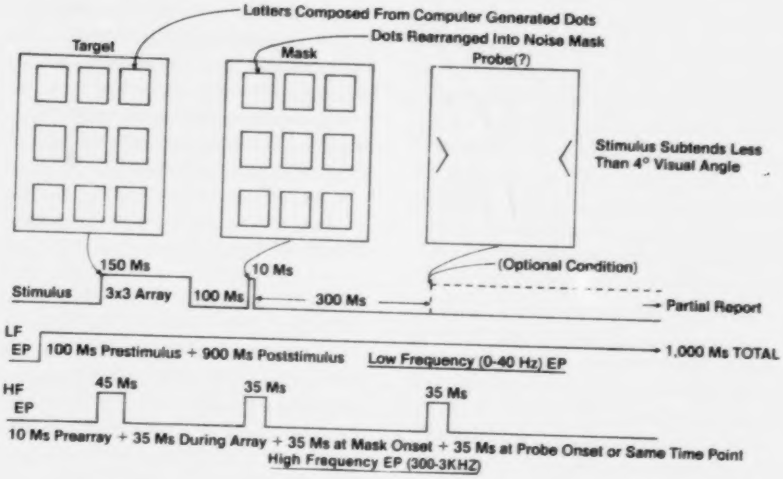
In our adaptation of this paradigm, a fixed stimulus exposure duration (of nine letters in a rectangular 3 x 3 array) is followed by a noise mask, and a partial report (of the middle line) is required from the subject. As the EP is measured throughout, we can observe the brain electrical events temporally concurrent with both the target stimulus and the masking stimulus. The brain electrical response to the target stimulus should therefore index some aspects of the early engagement of attentional processes when the subject is placed under an information-processing load. The EP events following the onset of the masking stimulus should indicate something of the brain events associated with the vulnerability of the early sensory store to disruption. To elaborate more fully on this reasoning: If the brain electrical activity were identical (in schizophrenics and controls) during the appearance of the *target stimulus*, then we would conclude that the early attentional engagement did not differ between the groups. We would then hypothesize that the *masking stimulus* produced a differential effect and would expect to see this reflected in differential brain electrical activity following the masking stimulus. If, on the other hand, a difference in brain electrical activity were observed between schizophrenics and controls *during* the target stimulus (and before the mask appeared), then we would conclude that the engagement of attentional processes was different between the groups even at this early stage. If this latter result were found along with a similar brain response to the mask in both schizophrenics and controls, then we would conclude that the deficits behaviorally observed in schizophrenics (in this task) might have been due to a failure of the target stimulus to engage the attentional processes of schizophrenics fully and that their performance deficit was not due to excessive vulnerability of their early sensory store to destruction by a secondary event (a mask in this case).

Methods

Subjects. All experimental subjects have been recruited and diagnosed (by Research Diagnostic Criteria) using identical instruments and personnel as reported in this issue by Spohn et al. (in fact, a number of the subjects are common to both studies). The reader is referred to that article for a more exhaustive description. Briefly, 19 schizophrenics (5 unmedicated and 14 medicated), 15 patient controls (4 unmedicated and 11 medicated) with the Research Diagnostic Criteria diagnosis of bipolar illness or schizoaffective disorder were compared with 31 normal controls. All groups were equated as far as possible for age (in the 18- to 55-year range) and all were male. All subjects consented to participation after full disclosure of the protocol. The patient control group allows for exploration of the specificity of findings to schizophrenia.

Backward Masking. Full technical details of our computer-controlled methodology will be reported elsewhere. Briefly, as shown diagramatically in figure 1: A target 3 × 3 array of letters was presented (within 4 degrees visual angle) to both eyes. This stimulus followed a dim fixation point, which disappeared 1 second

Figure 1. Schematic representation of stimuli and timing for a backward-masking paradigm, incorporating measurement of both high and low frequency evoked potentials


Letters Composed From Computer Generated Dots
Dots Rearranged Into Noise Mask
Target
Mask
Probe(?)
Stimulus Subtends Less Than 4° Visual Angle
150 Ms
10 Ms
(Optional Condition)
Stimulus
3x3 Array
100 Ms
300 Ms
→ Partial Report
LF EP
100 Ms Prestimulus + 900 Ms Poststimulus
Low Frequency (0-40 Hz) EP
1,000 Ms TOTAL
HF EP
45 Ms
35 Ms
35 Ms
10 Ms Prearray + 35 Ms During Array + 35 Ms at Mask Onset + 35 Ms at Probe Onset or Same Time Point
High Frequency EP (300-3KHZ)

before presentation. The letters of the array were easily readable and composed of dots, computer generated, and displayed on a fast-decay-phosphor oscilloscope screen. The entire 3 x 3 array can be created on the screen in less than 1 ms. Stimulus duration was a fixed 150 ms. The screen was blank for a further 100 ms, allowing a total stimulus availability of 250 ms including afterimage. A masking stimulus composed of exactly the same number of dots then appeared for 10 ms. The dots occupied the same block space as the letters on the screen and appeared as a 3 x 3 array of small masks. Thus, the perceived brightness of the mask was identical to that of the 3 x 3 array of letters, being composed of the same number of elements (dots) with the same brightness (Z axis gain) but rearranged into a meaningless "noise" array within the same area occupied previously by a letter. The 10 ms mask produced a 95-99 percent correct detection rate in normal subjects.

Increasing the mask duration to 15 ms produced a decrement in normals to about 70 percent correct detection, as revealed by pilot experimentation. The 10 ms mask duration was selected to allow normal subjects to perform close to ceiling scores. Subjects gave a *partial report* and were trained (before EP recording) to report only the middle line and never any other. Letters were arranged for each presentation according to a computer-controlled random sequence generator. The experiment required 50 artifact-free trials under each of two conditions (on separate days). In the first condition, the subject performed as described above; in the second, a small set of horizontally oriented chevrons "> <" appeared at the outer edges of the center line. These chevrons constituted a form of postmask probe and appeared (dim but clearly visible) 300 ms following the offset of the mask. When the probe was used, it remained visible until the experimenter reoriented the subject that a new trial would begin in about 10 seconds at which time it would disappear to be followed by the fixation spot at the beginning of a new trial. Intertrial interval was variable from 45-65 seconds as the experimenter waited until the subject was correctly head inclined (monitored by closed-circuit video) and the on-line electroencephalogram (EEG) monitor showed no artifacts and no eye movement. Each individual trial was repeated (but the letters changed) if it produced a computer-detected brain wave artifact. The subjects sat in a reclined (but not recumbent) position in a dimly lit room, which was quiet but not acoustically isolated.

Brain Wave Recording. The EEG was recorded from the full 10/20 system referenced to linked ears and with eye movement detection set at a very high gain. The latter is necessary to detect blinks and more subtle eye movements, and to reject any trial in which they occur. As this paradigm involves detection and report of letter strings, there is a danger that eye movements may cause distortion of later components of the EP. Stringent precautions were taken to avoid this. The 20 channels (19 of the 10/20 system plus eye movements) were recorded with a bandwidth of 0-10 kHz, and the 20 channels were then filtered into two separate outputs from each channel. Data reported here are from the low frequency bandwidth (.5-40 Hz-3 dB points). Data recording began 100 ms prestimulus and continued 900 ms poststimulus (for a total of 1000 ms). In addition, a high frequency bandwidth was recorded (300-3 kHz-3 dB points), beginning 10 ms prestimulus and continuing for 35 ms into the 3 × 3 array. Recording then terminated in this bandwidth and began again (for 35 ms) at the onset of the mask and again (for 35 ms) at the onset of the postmask probe or at exactly the same time point if the probe was not used. The low frequency channels were digitized at 400 Hz/channel, and the high frequency channels were digitized at 20,000 Hz/channel. All channels were recorded in *pure parallel* with a separate analog-to-digital converter on each. The low frequency and high frequency data streams were interleaved after digitation and relayed by direct memory access to the core memory of a PDP 11/34 computer (Digital Equipment Corporation). When low frequency and high frequency were recorded together, the total data input speed was 400,000 samples/second. These methods are highly technically demanding. High frequency data from this experiment will be reported elsewhere.

Data Reduction. Each trial was stringently checked by on-line computer for artifact. The artifact rejection was exceptionally conservative, and overvoltage detection on any one channel rejected the entire trial. The summed EPs for all channels and the sums of squares for each channel (trial by trial) were computed on line and the data stored on disk. The sums of squares data will not be reported here. The summed EPs (for each subject) were graphically displayed and checked visually for any possible artifacts. Data from normal subjects, schizophrenics, and patient controls were separately summed to produce grand mean profiles and also subjected to *t*-test statistical probability mapping (SPM) as recommended by Duffy, Bartels, and Burchfield (1981). The SPM maps are the only analyses presented here. In the future, we shall be reporting results acquired by multivariate approaches.

Results

The postmask probe stimulus did not produce any within-subject changes in the EP waveforms (or in correct responses). This finding is of interest and will be discussed below. The backward-masking paradigm produced a difference in performance range in the groups. Normals performed in the range of 95–99 percent correct responses, schizophrenics performed in the range of 50–70 percent correct responses, and, on the basis of preliminary analyses, patient control subjects performed at 55–90 percent correct responses. We may therefore tentatively conclude that the backward-masking paradigm differentiates schizophrenics from normals but not necessarily from patient controls, and that the postmask probe does not affect the EP waveform on a within-subject basis.

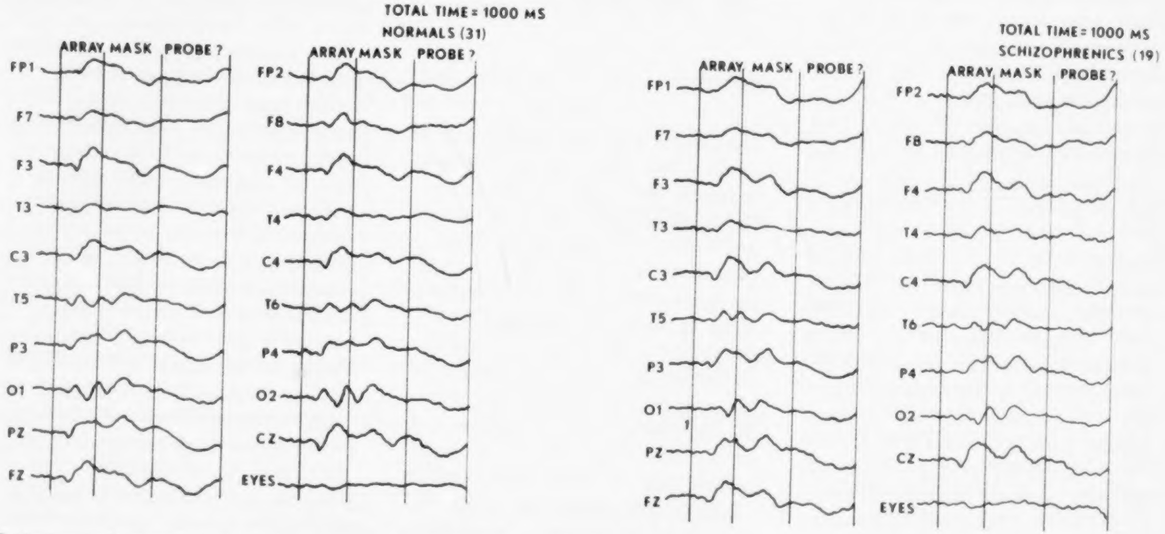
The most interesting finding is that the schizophrenics are differentiated from normal by an EP epoch which extends from approximately 80 ms to about 103 ms poststimulus. Figure 2 shows the grand mean EP waveforms for schizophrenics and normals, and figure 3 shows the SPM of comparison of brain topography of both groups at the 95 ms poststimulus point, where the differences are maximal between the schizophrenics and normals. (Figures 2 and 3 are redrawn by hand from color graphic computer topographical maps.) No other epoch of the EP waveform shows reliable differences between schizophrenics and normals. In addition, the schizophrenics depart from the patient control sample at 80–100 ms in the same way that they depart from normals. The patient control sample did not differ from normals at this epoch or at other times in the EP waveform.

The aforegoing results presentation is both partial and brief as befits reports from work in progress. The conclusions drawn must therefore be highly tentative, and may change with the addition of more subjects and more rigorous (multivariate) analysis. If this reservation is held firmly in mind, the following comments may be in order.

The addition of the postmask probe was incorporated to test, in a simple manner, the possibility that a small low-information-carrying event might differentially alter the backward-masking performance (or the EPs) of the schizophrenics and normals. The tentative conclusion is that it does not alter the performance of any group; thus, simple formulations of schizophrenics' vulnerability to disruption by *any* stimulus events occurring while information is being processed are not upheld.

One of the hypotheses under test here is that the brain potentials evoked by the mask may be of greater amplitude, regional distribution, or duration in the schizophrenics and, as such, may be "overwriting" the ongoing processing of the 3 x 3 array, hence demonstrating neurologically a possible mechanism for schizophrenic deficit in this task. This hypothesis is not borne out, and we find no evidence so far to this effect. From figure 2, however, we can see that a small amplitude but clearly discernible waveform in the 80–105 ms epoch in normals is not apparent in schizophrenics. This waveform is temporally coincident with the target stimulus presentation, which continues until 150 ms. A tentative conclusion might be that these EP waveform differences speak to early

Figure 2. Low frequency evoked potentials (50 artifact-free trials) from normals and schizophrenics



Evoked potentials were obtained from the paradigm shown in Figure 1.

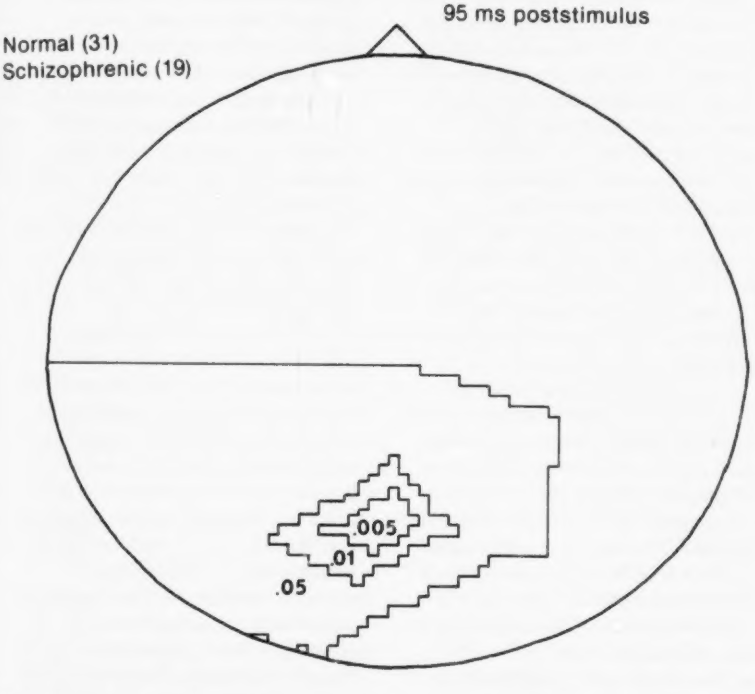
information registration differences between schizophrenics and normals or, more particularly, to the time period during which the sensory store is actually being formed. Alternatively, we might say that the target stimulus has engaged some attentional process in the normals but failed to do so in the schizophrenics.

Discussion

At this juncture we again call attention to the tentative nature of our data (due to the small number of subjects involved) and consequently the tentative nature of our conclusions and interpretations. Nonetheless, some interesting points can be made about the potential significance of the findings. The lack of a well-defined negative-going wave in the 80–105 ms epoch in the schizophrenics appears to be a reasonably strong finding and favors an interpretation of the nonengagement of some early attentional component in these subjects. Perhaps the data also speak to the fact that this early attentional dysfunction may take the form of an inadequacy in forming the early sensory store in the sense of Broadbent (1984). The failure to observe any significant EP differences (between schizophrenics and normals) at later time epochs than approximately 100 ms is perhaps a much weaker finding for the following reason: The early (pre-100 ms) EP events have a much smaller peak amplitude/latency variability (on a between-subjects, within-groups basis) than events in the later epochs (200 ms and beyond). In other words, those subjects who display an N_{100} wave will perhaps have a within-group variability of 10 ms, and thus a peak amplitude of the component somewhere between 95 and 105 ms. This is not the case with later components, where the "time window" within which the P_{300} EP component has its peak amplitude, for example, may range from 280 ms to 400 ms depending to some extent on the experimental paradigm but *also* on a substantial between-subjects, within-groups variability. For these reasons, although our 80–100 ms negative wave is quite small in amplitude, due to its lesser peak amplitude/latency variability, it may be "easier" (i.e., require fewer subjects) to demonstrate a significant between-groups effect predicated upon a small within-groups variability.

By the same logic, it will be more difficult (i.e., require more subjects) to demonstrate a later EP epoch (between-groups) difference (if it exists) due to the greater variability of these components as described above. For these reasons, we feel much more confident that an early (pre-100 ms) group difference exists than we do that a later EP epoch

Figure 3. Statistical probability map



Statistical probability mapping follows the procedure of Duffy, Bartels, and Burchfield (1981). The significance of *t*-test comparison between schizophrenics and normals is shown. See figure 2 for evoked potential waveforms.

difference does *not* exist. Larger subject numbers in each group should increase this confidence in the future.

Before we proceed to more speculative discussion in the language and neurophysiological areas of schizophrenia, this may be an appropriate place to make two additional observations: the first concerning the N_{100} EP wave form, and the second concerning a neurophysiological finding that may have some importance for early information processing.

A negative-going wave peaking at approximately 100 ms (latency largely determined by experimental conditions) has been recorded in human subjects in a wide variety of experimental paradigms. Recent data from Baribeau and Laurent (1985) suggest that this wave is indexing some aspect of "input channel selectivity" or stimulus filtering set (largely based on the work of Parasuraman 1978). The wave that we have observed to differentiate schizophrenics from normal *may* be the same wave or closely related. Accordingly, the attribution of this wave as indicating "predecision processing of signals in conjunction with target identification and categorization" (Desmedt, Huy, and Bourguet 1983, p. 272) agrees closely with our interpretation. Findings which indicate that the N_{100} is sensitive to manipulation of directed attention (Parasuraman 1980) and to processing demand (Callaway and Halliday 1982) may further substantiate this claim. Schizophrenic children have been examined by Strandburg et al. (1984) and were found to deviate from normal children in failing to increase the N_{100} component as a function of information-processing demand. The authors interpret this finding (along with other EP findings) as indicative of a failure "to regulate processes involved in the mobilization and direction of attention and the discrimination of target stimuli" (p. 251).

With such considerations in mind, we have begun to contemplate a situation wherein target stimuli (in the experimental laboratory setting) have some "inherent" or "automatic" attribution of potential significance assigned to them in the brains of normal subjecs but not in the brains of schizophrenics. One possible implication of such an outlook is that the "automatic" or "inherent" significance is attributable to the functioning of a very fast (but crudely analyzing) information-processing system which gets some representation of the target stimulus all the way from the input sensors, through the brainstem, and into the cortex in a very preliminary representational form. This "fast" input could then be used to mobilize attentional resources faster than a finer grained input-analyzing system. The credibility of such an outlook is largely predicated on its neuroanatomical feasibility. Recent data (Van der Kooy and Kolb 1985) indicate that an important brainstem structure, the globus pallidus, has axonal projections directly to the cortex as well as projections to the subthalamic nucleus and the paraventricular thalamic nucleus. Our suggestion is therefore that two routes of information relay to cortex are available: a faster (but minimally analyzing) system from brainstem structures and a slower (but more "neural-integrative") route through the thalamus and, hence, by thalamic relay to the cortex. The implication is that the "fast" route of information "preprocessing" is either not used or *cannot* be used by the schizophrenics. Although these ideas are highly speculative, they represent a preliminary attempt to account for fairly widespread findings by other investigators as well as ourselves in schizophrenic adults and children. In any event, the globus pallidus and thalamus have demonstrably important functions in language information processing, and we will return to this aspect of our discussion below.

The thematic orientation of this issue of the *Schizophrenia Bulletin* is thought disorder, and it is therefore appropriate to make more explicit the links between thought disorder as seen clinically and information processing as viewed in experimental psychopathology.

The crucial element here is schizophrenic speech, itself the subject of considerable recent controversy (Schwartz 1982; Chaika and Lambe 1985; Lanin-Kettering and Harrow 1985). We do not intend to join this controversy but rather to bring to it some concepts from the work presented above and suggest at least one method to test these concepts.

Thought disorder as diagnosed clinically is almost totally dependent upon examination of the speech of schizophrenics. It is our assumption that deviant schizophrenic speech is not predicated upon defects in the language faculty of schizophrenics, but instead reflects defects in their information-processing abilities as indicated by the material presented above. This assumption shares some ground with recent work from Grove and Andreasen (1985) where certain aspects of language were found to be intact in schizophrenia but a short-term memory deficit was strongly indicated. We therefore propose a trichotomous division between speech (which can be observed and recorded), language (the "knowledge" component that cannot be observed but is inferred from behavior), and thinking (which is loosely equated with information processing in our exposition).

From Schwartz (1982), it may be concluded that schizophrenics do not have difficulties at the level of the meaning of words but may have severe difficulties at the level of sentence use and sentence comprehension. The important addition in the transition between use of words and use of sentences is that a "time buffer" has been added in the case of the sentence. In other words, the early material in the sentence only acquires "meaning" in the *context* of the later material in the sentence (and vice versa), and the early material must be held in some temporary store or "buffer" until the later material arrives.

Recent EP evidence shows very clearly (Kutas and Hillyard 1984; McCallum, Farmer, and Pocock 1984) that a semantically incongruous ending to a sentence will produce a characteristic EP waveform (the N_{400}). The data of McCallum, Farmer, and Pocock (1984) further show that this N_{400} ("incongruous" response) starts to develop very early in time relative to the onset of the incongruous word. This strongly suggests that a *contextually driven expectancy* is set up by the subjects, as a function of the early material in the sentence. This expectation may form a (forward-projected) semantic "template" for an expected ending to the sentence; if it does not occur, a very rapid detection of the incongruity results, faster than would be possible if the later material in the sentence required more complete processing. In all likelihood, this is a normal and essential function for conversational ability in humans and is a likely candidate for defect in schizophrenics in the light of the information-processing defects posited above. We would argue that early components in the sentence may not engage sufficient attentional resources (in the schizophrenic) so that a contextually driven expectancy is inadequately generated with which to ascertain the meaning of later sentencial elements. Such a process would also be held to occur in "reverse," whereby an inadequately represented early sentence component may not be amenable to semantic modification in the light of a later sentence component. In this formulation, we contend that we are directly addressing the phenomenon of thought disorder (as diagnosed clinically) and that schizophrenics' difficulties at the level of sentence use and comprehension may be predicated upon early information-processing dysfunction.

To test this possibility, replications of the Kutas and Hillyard (1984) and McCallum, Farmer, and Pocock (1984) experiments are needed with schizophrenics. In such paradigms, we would strongly hypothesize that the N_{400} EP will not appear (as in normals) to semantic incongruity or, alternatively, that the N_{400} will be of reduced amplitude and the point at which the "incongruous" EP waveform begins to depart from the semantically congruous waveform will be later in time. In the latter case, we would interpret such results to indicate that a contextually driven expectancy is not being used to enable rapid detection of incongruity in schizophrenics and, consequently, that a more complete (and slower) processing is required for the incongruous material. If such results are observed, they may well underlie failure of "discourse editing" in schizophrenia discussed by Schwartz (1982) and Grice (in commentary to Schwartz, 1982, in the same article). We shall return to this material below in relation to recent studies of the thalamus in language and thalamic post-mortem findings in schizophrenia.

In light of recent findings, it is possible to speculate on the neurological underpinnings to the early information-processing defect posited above. Findings with various forms of EPs, as well as recent post-mortem findings in schizophrenics, are pertinent here.

The brainstem EP shows a series of small waves within about 10 ms that index the earliest stages of stimulus input to the brain, mostly below the level of the thalamus.

A small number of studies have examined the brainstem EP in schizophrenics (see Brecher and Begleiter, 1985, for a brief review as well as new data from nine schizophrenic patients). The general consensus is that schizophrenics do not differ from normal in this earliest stage of information input. However, the brainstem EP is notoriously variable, even in normal subjects, and most studies have not tested an adequately large patient or control population to make a definitive statement possible. Patterson et al. (1985) have topographically mapped the auditory brainstem EP and preliminary data show subtle topographical differences between schizophrenics and normals which may lead, with greater subject numbers, to the identification of a subpopulation of schizophrenics outside the normal range on this measure.

Interestingly, the patient control population of Patterson et al. (1985) (schizoaffectives and bipolars) showed much of the same subtle aberrations as the schizophrenic population. It is therefore possible that subtle deviations of the brainstem EP are not specific to schizophrenics, but may be sensitive to a more generalized psychotic or affective component.

If we accept for the moment that the electrophysiological indicators of stimulus input at the brainstem level do not indicate pathology, then we must ask why abnormal EPs are observed in schizophrenics at 50 ms (Adler et al. 1982; Freedman et al. 1983) and beginning at 70 ms, peaking at approximately 95 ms in the data that we have reported above. This time frame is coincident with complex interactions of the thalamus, limbic brain, and cortex. Recent research has shown direct thalamic involvement in EPs in the 18-24 ms time frame (Kudo and Yamadori 1985), as well as a specific thalamic negativity temporally coincident with the surface-recorded P_{300} waveform (Katayama, Tsukiyama, and Tsubokawa 1985). In addition, the hippocampal gyrus has been shown to be a specific dipole generating source for the 200-300 ms epoch of the EP when recorded by electromagnetic radiation (Okada, Kaufman, and Williamson 1983). It is therefore reasonable to look at possible thalamic and hippocampal sources for the underpinnings to some EP abnormalities in schizophrenia.

Kovelman and Scheibel (1984) have reported data which demonstrate profound disarray in the hippocampal CA1/CA2 layers of post-mortem schizophrenic brains. A tentative interpretation of their data suggests a pathological continuum from normal subjects through borderline schizophrenic conditions to intractable chronic schizophrenia. For our purposes, if these data are considered along with the EP studies of Halgren et al. (1980), who directly examined hippocampal and amygdala unit discharge activity (coincident with surface-recorded activity), then they strongly suggest that abnormal hippocampal influences may be seen in the surface-recorded EP and that abnormal limbic brain components may underlie some EP findings in schizophrenia.

The complete thalamus of post-mortem schizophrenic brains has been examined by Adams and Oke (in preparation, personal communication) with specific reference to its regional neurochemical constituents. Their data show a particularly marked alteration of the noradrenalin-to-dopamine ratio in many regions of the thalamus, with a considerably greater concentration of dopamine than is seen in the normal post-mortem brain. The importance of these data is heightened by the fact that unusually accurate and detailed clinical records are available for these subjects.

These data are again tentative indications that the thalamus may be contributing to neuroanatomical underpinnings of abnormal surface-recorded EPs in schizophrenia.

Both the (various) thalamic nuclei and the hippocampus and amygdala in the limbic brain are crucially important neurointegrative centers controlling the interactions of brainstem and cerebral cortex (for a more complete discussion, see Pay 1980, 1981, 1982). Certain aspects of thalamic function may be directly observable with the 40 Hz EP technique (Spydell, Pattee, and Goldie 1985), and replication of such experiments may demonstrate the intactness or otherwise of these functions of the thalamus in schizophrenia.

Perhaps a more important consideration is that certain aspects of schizophrenic dysfunction are currently attributed to the frontal cortex. For example, Levin (1984*a*, 1984*b*) has elegantly argued the case for a dysfunction in frontal eye-field control of smooth pursuit eye tracking in schizophrenia. While such attribution may be partially correct, it must be borne in mind that the exceptionally complex internal structure of the frontal cortex is partly defined by the pathways that project to it from the anterior and mediodorsal thalamus and from the limbic brain. A fine-grained analysis of these interactions has recently been reviewed and integrated by Reep (1984). It may therefore be concluded that defects in the anatomical structure of hippocampus and neurochemical abnormalities in the thalamus, as previously discussed, may be playing some part in what appears to be an abnormality in various frontal cortex functions in schizophrenia.

On a more behavioral level and following our previous discussion of abnormalities of speech in schizophrenia, a most elegant and provocative theoretical model of "spoken language formulation" is given by Crosson (1985). In this model, the thalamus plays a central role in relaying arousal information (received from the basal ganglia and, most particularly, the globus pallidus) to the cortex and also in timing the release of formulated language (Wernicke's area) for motor programming (Broca's area). Crosson's model allows for intactness in the classical language and speech areas while still permitting speech defects to occur through the type of abnormal temporal sequencing (thalamically mediated) that we have posited in schizophrenia on the basis of EP and startle reflex data.

In conclusion, we have presented preliminary findings from a backward-masking experiment in which the EP data are consistent with an interpretation of an early information-processing dysfunction in schizophrenic patients which may account for their reported deficit behaviorally in this task. When such findings are considered along with data from startle reflex experiments, self-initiated EP paradigms, and findings in paired stimulus EPs, then a plausible hypothesis of abnormalities (of a generalized nature) in temporal sequencing control of information processing in schizophrenia is generated. Such a hypothesis, when considered in the light of recent reports of anatomical abnormalities in the hippocampus and thalamus of schizophrenics, produces in our opinion a compelling theoretical framework within which abnormalities in schizophrenic verbalization can be understood both behaviorally and neurologically. Such a formulation is not inconsequential as it strikes at the heart of the verbal behavior of schizophrenics, upon which the clinical diagnosis of thought disorder is fundamentally based.

References

Adler, L.E.; Pachtman, E.; Franks, R.D.; Pecevich, M.; Waldo, M.C.; and Freedman, R. Neurophysiological evidence for a defect in neuronal mechanisms involved in sensory gating in schizophrenia. *Biological Psychiatry*, 17:639–654, 1982.

Baribeau, J., and Laurent, J.P. "Neurophysiological Indices of Two Distinct Attentional Dysfunctions in Schizophrenia With and Without Formal Thought Disorder." Presented at the 7th International Symposium on Clinical Neurophysiological Aspects of Psychiatric Conditions, Philadelphia, September 1985.

Braff, D.L.; Callaway, E. III; and Naylor, H. Very short-term memory dysfunction in schizophrenia. *Archives of General Psychiatry*, 34:25–30, 1977.

Braff, D.L., and Saccuzzo, D.P. The time course of information-processing deficits in schizophrenia. *American Journal of Psychiatry*, 142:170–174, 1985.

Braff, D.L.; Stone, C.; Callaway, E. III; Geyer, M.; Glick, I.; and Likh, B. Prestimulus effects on human startle reflex in normals and schizophrenics. *Psychophysiology*, 15:339–343, 1978.

Brecher, M., and Begleiter, H. Brain stem auditory evoked potentials in unmedicated schizophrenic patients. *Biological Psychiatry*, 20:199–202, 1985.

Broadbent, D.E. *Perception and Communication*. London: Pergamon Press, 1958.

Broadbent, D.E. The Maltese cross: A new simplistic model for memory. *Behavioral and Brain Sciences*, 7:55–94, 1984.

Callaway, E. III, and Halliday R. The effect of attentional effort on visual evoked potential N_1 latency. *Psychiatry Research*, 7:299–308, 1982.

Chaika, E., and Lambe, R. The locus of dysfunction in schizophrenic speech. *Schizophrenia Bulletin*, 11:8–15, 1985.

Crosson, B. Subcortical functions in language: A working model. *Brain and Language*, 25:257–292, 1985.

Desmedt, J.E.; Huy, N.T.; and Bourguet, M. The cognitive P_{40}, N_{60},

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and P_{100} components of somatosensory evoked potentials and the earliest electrical signs of sensory processing in man. *Electroencephalography and Clinical Neurophysiology*, 56:272–283, 1983.

Duffy, F.H.; Bartels, P.H.; and Burchfield, J.L. Significance probability mapping: An aid in the topographic analysis of brain electrical activity. *Electroencephalography and Clinical Neurophysiology*, 51:455–462, 1981.

Freedman, R.; Adler, L.E.; Waldo, M.C.; Pachtman, E.; and Franks, R.D. Neurophysiological evidence for a defect in inhibitory pathways in schizophrenia: Comparison of medicated and drug-free patients. *Biological Psychiatry*, 18:537–551, 1983.

Graham, F.K. The more or less startling effects of weak prestimulation. *Psychophysiology*, 12:238–248, 1975.

Grove, W.M., and Andreasen, N.C. Language and thinking in psychosis. *Archives of General Psychiatry*, 42:26–32, 1985.

Haber, R.N. The impending demise of the icon—A critique of the concept of iconic storage in visual information processing. *The Behavioral and Brain Sciences*, 6:1–54, 1983.

Halgren, E.; Squires, N.K.; Wilson, C.L.; Rohrbaugh, J.W.; Babb, T.L.; and Crandall, P.H. Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. *Science*, 210:802–805, 1980.

Hemsley, D.R., and Richardson, P.H. Shadowing by context in schizophrenia. *Journal of Nervous and Mental Disease*, 168:141–145, 1980.

Hoffman, H.S.; and Ison, J.R. Reflex modification in the domain of startle. *Psychological Review*, 87:175–189, 1980.

Johnson, J.D. A mechanism to inhibit input activation and its dysfunction in schizophrenia. *British Journal of Psychiatry*, 146:429–435, 1985.

Katayama, Y.; Tsukiyama, T.; and Tsubokawa, T. The thalamic negativity associated with the endogenous late positive component of cerebral evoked potentials (P_{300}): Recordings using discriminative aversive conditioning in humans and cats. *Brain Research Bulletin*, 14:223–226, 1985.

Kovelman, J.A., and Scheibel, A.B. A neurohistological correlate of schizophrenia. *Biological Psychiatry*, 19:1601–1621, 1984.

Kudo, Y., and Yamadori, A. Somatosensory evoked potentials in patients with thalamic lesions. *Journal of Neurology*, 232:61–66, 1985.

Kutas, M., and Hillyard, S. Brain potentials during reading reflect word expectancy and semantic association. *Nature*, 307:161–163, 1984.

Lanin-Kettering, I., and Harrow, M. The thought behind the words: A view of schizophrenic speech and thinking disorders. *Schizophrenia Bulletin* 11:1–7, 1985.

Leitner, D.S.; Powers, A.S.; and Hoffman, H.S. The neural substrate of the startle response. *Physiology & Behavior*, 25:291–297, 1980.

Leitner, D.S.; Powers, A.S.; Stitt, C.L.; and Hoffman, H.S. Midbrain reticular formation involvement in the inhibition of acoustic startle. *Physiology & Behavior*, 26:259–268, 1981.

Levin, S. Frontal lobe dysfunctions in schizophrenia—I. Eye movement impairments. *Journal of Psychiatric Research*, 18:27–55, 1984*a*.

Levin, S. Frontal lobe dysfunctions in schizophrenia—II. Impairments of psychological and brain functions. *Journal of Psychiatric Research*, 18:57–72, 1984*b*.

McCallum, W.C.; Farmer, S.F.; and Pocock, P.V. The effects of physical and semantic incongruities on auditory event-related potentials. *Electroencephalography and Clinical Neurophysiology*, 59:477–488, 1984.

Okada, Y.C.; Kaufman, L.; and Williamson, S.J. The hippocampal formation as a source of the slow endogenous potentials. *Electroencephalography and Clinical Neurophysiology*, 55:417–426, 1983.

Parasuraman, R. Auditory evoked potentials and divided attention. *Psychophysiology*, 15:460–465, 1978.

Parasuraman, R. Effects of information processing demands on slow negative shift latencies and N_{100} amplitude in selective and divided attention. *Biological Psychology*, 11:217–233, 1980.

Patterson, T.; Bogia. D.P.; Campbell, E.W.; and Hartzell, R. "Topographical Mapping of the Brainstem Auditory Evoked Potential: Schizophrenics and Control Subjects." Presented to the 25th Annual Meeting of the Society for Psychophysiological Research, Houston, TX, October, 1985.

Pay, R.G. Contextual organization of unitary information processes in the cortex by the thalamus and basal ganglia and the central control of attention. *International Journal of Neuroscience*, 11:249–278, 1980.

Pay, R.G. Control of complex operations and emotion in the neocortex by the limbic entorhinal subicular, and cingulate cortices and the hypothalamus, mammillary body and thalamus. *International Journal of Neuroscience*, 15:1–30, 1981.

Pay, R.G. Behavioral steering in dual and social states of conation by the amygdala, hypothalamus, ventral striatum, and thalamus. *International Journal of Neuroscience*, 16:1–40, 1982.

Reep, R. Relationship between prefrontal and limbic cortex: A comparative anatomical review. *Brain, Behavior and Evolution*, 25:5–80, 1984.

Saccuzzo, D.P.; Hirt, M.; and Spencer, T.J. Backward masking as a measure of attention in schizophrenia. *Journal of Abnormal Psychology*, 83:512–522, 1974.

Saccuzzo, D.P., and Schubert, D.L. Backward masking as a measure of slow processing in schizophrenia spectrum disorders. *Journal of Abnormal Psychology*, 90:305–312, 1981.

Schafer, E.W.P., and Marcus, M.M. Self-stimulation alters human sensory brain responses. *Science*, 181: 175–177, 1973.

Schneider, W., and Shiffrin, R.M. Controlled and automatic human information processing: I. Detection, search and attention. *Psychological Review*, 84:1–66, 1977.

Schwartz, S. Is there a schizophrenic language? *Behavioral and Brain Sciences*, 5:579–626, 1982.

Shagass, C. An electrophysiological view of schizophrenia. *Biological Psychiatry*, 11:3–30, 1976.

Shagass, C.; Roemer, R.A.; Straumanis, J.J.; and Amadeo, M. Temporal variability of somatosensory, visual and auditory evoked potentials in schizophrenia. *Archives of General Psychiatry*, 36:1341–1351, 1979.

Spydell, J.D.; Pattee, G.; and Goldie, W.D. The 40 hertz auditory event-related potential: Normal values and effects of lesions. *Electroencephalography and Clinical Neurophysiology*, 62:193–202, 1985.

Strandburg, R.J.; Marsh, J.T.; Brown, W.S.; Asarnow, R.F.; and Guthrie, D. Event-related potential concomitants of information processing dysfunction in schizophrenic children. *Electroencephalography and Clinical Neurophysiology*, 57:236–253, 1984.

Van der Kooy, D., and Kolb, B. Non-cholinergic globus pallidus cells that project to the cortex but not to the subthalamic nucleus in the rat. *Neuroscience Letters*, 57:113–118, 1985.

Yates, A.J. Data processing levels and thought disorders in schizophrenia. *Australian Journal of Psychology*, 18:103–117, 1966.

Yates, A.J., and Korboot, P. Speed of perceptual functioning in chronic nonparanoid schizophrenics. *Journal of Abnormal Psychology*, 76: 453–461, 1970.

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Scale for the Assessment of Thought, Language, and Communication (TLC)

by Nancy C. Andreasen

The following set of definitions was developed to improve the reliability of assessments of "formal thought disorder." In the past such assessments have been unreliable at least in part because clinicians and researchers have not agreed upon the meaning of the terms which they have used. It is hoped that the following definitions will provide a common and reliable stock of terms to describe the language and cognitive behaviors which can be observed in psychiatric patients.

This set of definitions began with a clinical recognition that the concept of "formal thought disorder" has often been misused and misunderstood. First, it has often been treated as if it were unitary, but in fact it is composed of a number of different language behaviors which are conceptually divergent and not always correlated in the same patient, such as "poverty of thought" and "loose associations." The recognition of the diversity of concepts and terms has led to the specification of 18 different types of "formal thought disorder."

Second, it has been assumed that "formal thought disorder," or at least "thought disorder," is pathognomonic of schizophrenia and omnipresent within schizophrenic patients. Clinical experience contradicts both assumptions. Language behaviors such as associative loosening, clanging, blocking, overconcrete or repetitive speech, and poverty of speech also occur in other psychiatric disorders such as mania or depression, and they also occur in the speech of people who do not meet the criteria for any psychiatric diagnosis, particularly when they are fatigued or stressed. Furthermore, some schizophrenic patients seem to speak and think normally, with only specific delusions or hallucinations (i.e., disorders of content of thought or perceptual disorders) as manifestations of their schizophrenia.

Because the term "formal thought disorder" has been so misunderstood and misused, it is recommended that it no longer be used. The various disorders which comprised the concept of "formal thought disorder" can be better conceptualized as "disorders of thought, language, and communication." If viewed from an empirical perspective, most of them are in fact disorders of communication, and the notion of thought need only be invoked to explain a few of them.

That is, the following set of definitions began with the idea that the reliability of assessments could be improved if "thought disorder" were defined in terms of language behavior, and only behavior which could be directly observed would be evaluated. Most of the time, the language behavior involves a dyadic interaction between a speaker and a listener, and the disorder occurs because the speaker fails to follow a set of rules which are conventionally used to make it easier for listeners to understand. When the speaker fails to take the various needs of the listener into account, the result is usually a communication disorder. According to this definition, the following items from the scale are "communication disorders": poverty of content of speech, pressure of speech, distractible speech, tangentiality, derailment, stilted speech, echolalia, self-reference, circumstantiality, loss of goal, perseveration, and blocking. The concept of language disorder should be invoked for those specific disorders in which

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the speaker violates the syntactical and semantic conventions which govern language usage: incoherence, clanging, neologisms, and word approximations. The concept of thought disorder comprises only those disorders in which thinking alone seems aberrant: poverty of speech (aberrant because thought seems not to occur) and illogicality (aberrant inferential processes). These various disorders can be referred to collectively as thought-language-communication disorders, or TLC disorders.

Experience in using this scale has indicated that some TLC disorders are more suggestive of severe psychopathology than others. These have been grouped together at the beginning of the scale and consist of the first 11 items. The data collected to date concerning the frequency of these various TLC disorders in mania, schizophrenia, and depression are summarized in the appendix. As those data indicate, some TLC disorders considered to be very important in the past, such as neologisms or blocking, are in fact so infrequent as to be of little use in assessing most patients.

In choosing which disorders to cover in the scale and how to define them, decisions were sometimes made to redefine, combine, or delete older concepts in order to enhance reliability. For example, the term "loose associations" has not been used because it is based on an outdated associationist psychology and because it has been used so loosely as to be nearly meaningless. The term "derailment" has been substituted because it carries a minimum of baggage and because it is graphically descriptive. Four other terms which also may be at times equivalent to the older concept of associative loosening are used in the scale: tangentiality, incoherence, illogicality, and clanging. Since it is probably impossible to achieve good reliability when clinicians must make judgments on how close relationships are between various ideas, definitions which must turn on this judgment have generally been eliminated. Therefore, for example, the term "flight of ideas" has been dropped and is now subsumed under the concept of derailment. In order to permit an assessment of thought, language, and communication in a wide range of patients, the definitions have not been limited to disorders customarily considered to be characteristic only of schizophrenia. Definitions of two terms which are drawn from aphasiology, semantic and phonemic paraphasia, are also included so that they can be distinguished from incoherence.

Most of the ratings can be made after a patient has been evaluated with an ordinary psychiatric interview, since this is a good vehicle for eliciting typical patterns of speech using relatively standardized questions. During some time the patient should be permitted to talk as long as possible to observe his speech during this condition. The patient should be interrupted at some time in order to see how he responds to this.

Most of the ratings are described quantitatively, i.e., how often they occur during an interview. These ratings are based on the assumption that most interviews take about 50 minutes. For longer or shorter interviews, the values should be adjusted accordingly.

The interrater reliability of these definitions has been carefully evaluated and found to be very good. For a phenomenon such as TLC disorder, which depends on subjective judgments about phenomena which may change dramatically over a few days, interrater reliability is more meaningful than test-retest reliability. The data in the appendix concerning reliability are based on live interviews of 113 patients (32 manics, 36 depressives, 45 schizophrenics).

1. Poverty of Speech (Laconic Speech, Poverty of Thought)

Restriction in the *amount* of spontaneous speech, so that replies to questions tend to be brief, concrete, and unelaborated. Unprompted additional information is rarely provided. For example, in answer to the question, "How many children do you have?", the patient replies, "Two. A girl and a boy. The girl is 13 and the boy 10." "Two" is all that is required to answer the question, and the rest of the reply is additional information. Replies may be monosyllabic, and some questions may be left unanswered altogether. When confronted with this speech pattern, the interviewer may find himself frequently prompting the patient in order to encourage elaboration of replies. To elicit this finding, the examiner must allow the patient adequate time to answer and to elaborate his answer.

Example: Interviewer: "Do you think there's a lot of corruption in government?" Patient: "Yeah, seem to be." Interviewer: "Do you think Haldeman and Erlichman and Mitchell have been fairly treated?" Patient: "I don't know." Interviewer: "Were you working at all before you came to the hospital?" Patient: "No." Interviewer: "What kind of jobs have you had in the past?" Patient: "Oh, some janitor jobs, painting." Interviewer: "What kind of work do you do?" Patient: "I don't. I don't like any kind of work. That's silly." Interviewer: "How far did you go in school?" Patient: "I'm still in the 11th grade." Interviewer: "How old are you?" Patient: "Eighteen."

0 No poverty of speech. A substantial and appropriate number of replies to questions include additional information.

1 Slight poverty of speech. Occasional replies do not include elaborated information even though this is appropriate.

2 Moderate poverty of speech. Some replies do not include appropriately elaborated information, and many replies are monosyllabic or very brief ("Yes." "No." "Maybe." "Don't know." "Last week.").

3 Severe poverty of speech. Answers are rarely more than a few words in length. Questions may be left unanswered.

4 Extreme poverty of speech. Patient is essentially mute.

2. Poverty of Content of Speech (Poverty of Thought, Alogia, Verbigeration, Negative Formal Thought Disorder)

Although replies are long enough so that speech is adequate in amount, it conveys little information. Language tends to be vague, often overabstract or overconcrete, repetitive, and stereotyped. The interviewer may recognize this finding by observing that the patient has spoken at some length but has not given adequate information to answer the question. Alternatively, the patient may provide enough information, but require many words to do so, so that a lengthy reply can be summarized in a sentence or two. Sometimes the interviewer may characterize the speech as "empty philosophizing."

Exclusions. This finding differs from circumstantiality in that the circumstantial patient tends to provide a wealth of detail.

Example. Interviewer: "Ok. Why, why is it do you think that, people believe in God?" Patient: "Well, first of all because, he uh ly, he are the person that, is their personal savior. He walks with me and talks with me. And, uh, the understanding that I have, um, a lot of peoples, they don't really, uh, know they own personal self. Because, uh, they ain't, they all, just don't know they own personal self. They don't, know that he, uh, seemed like to me, a lot of 'em don't understand that he walks and talks with them. And, uh, show them their way to go. I understand also that every man and every lady, is just not pointed in the same direction. Some are pointed different. They goes in their different ways. The way that, uh, Jesus Christ wanted 'em to go. Me myself I am pointed in the ways of, uh, knowing right from wrong and doing it. I can't do no more, or no less, than that."

0 No poverty of content of speech.

1 Mild poverty of content of speech. Occasional replies are too vague to be comprehensible or can be markedly condensed.

2 Moderate poverty of content of speech. Replies which are vague or can be markedly condensed make up at least a quarter of the interview.

3 Severe poverty of content of speech. At least half the interview is composed of vague or incomprehensible replies.

4 Extreme poverty of content of speech. Most of the interview is vague, incomprehensible, or can be markedly condensed.

3. Pressure of Speech

An increase in the amount of spontaneous speech as compared to what is considered ordinary or socially customary. The patient talks rapidly and is difficult to interrupt. Some sentences may be left uncompleted because of eagerness to get on to a new idea. Simple questions which could be answered in only a few words or sentences are answered at great length so that the answer takes minutes rather than seconds and indeed may not stop at all if the speaker is not interrupted. Even when interrupted, the speaker often continues to talk. Speech tends to be loud and emphatic. Sometimes speakers with severe pressure will talk without any social stimulation and talk even though no one is listening. When patients are receiving phenothiazines or lithium, the speech is often slowed down by medication, and then it can be judged only on the basis of amount, volume, and social appropriateness. If a quantitative measure is applied to the rate of speech, then a rate greater than 150 words/minute is usually considered rapid or pressured. This disorder may be accompanied by derailment, tangentiality, or incoherence, but it is distinct from them.

0 No pressure of speech.

1 Slight pressure of speech. Some slight increase in amount, speed, or loudness of speech.

2 Moderate pressure of speech. Usually takes several minutes to answer simple questions, may talk when no one is listening, and/or speaks loudly and rapidly.

3 Severe pressure of speech. Frequently takes as much as 3 minutes to answer simple questions, sometimes begins talking without social stimulation, and/or difficult to interrupt.

4 Extreme pressure of speech. Patient talks almost continually, cannot be interrupted at all, and/or may shout to drown out the speech of others.

4. Distractible Speech

During the course of a discussion or interview, the patient stops talking in the middle of a sentence or idea and changes the subject in response to a nearby stimulus, such as an object on a desk, the interviewer's clothing or appearance, etc.

Example. "Then I left San Francisco and moved to . . . Where did you get that tie? It looks like it's left over from the fifties. I like the warm weather in San Diego. Is that a conch shell on your desk? Have you ever gone scuba-diving?"

0 Absent.

1 Mild (is distracted once during an interview).

2 Moderate (is distracted from two to four times during an interview).

3 Severe (is distracted from 5 to 10 times during an interview).

4 Extreme (is distracted more than 10 times during an interview).

5. Tangentiality

Replying to a question in an oblique, tangential, or even irrelevant manner. The reply may be related to the question in some distant way. Or the reply may be unrelated and seem totally irrelevant. In the past tangentiality has been used as roughly equivalent to loose associations or derailment. The concept of tangentiality has been partially redefined so that it refers only to replies to questions and not to transitions in spontaneous speech.

Example. Interviewer: "What city are you from?" Patient: "Well, that's a hard question to answer because my parents . . . . I was born in Iowa, but I know that I'm white instead of black so apparently I came from the North somewhere and I don't know where, you know, I really don't know where my ancestors came from. So I don't know whether I'm Irish or French or Scandinavian or I don't, I don't believe I'm Polish but I think I'm, I think I might be German or Welsh. I'm not but that's all speculation and that, that's one thing that I would like to know and is my ancestors, you know, where did I originate. But I just never took the time to find out the answer to that question."

0 No tangentiality.

1 Mild (occurs once during an interview).

2 Moderate (occurs from two to four times).

3 Severe (occurs from 5 to 10 times).

4 Extreme (occurs more than 10 times, or so frequently that the interview is incomprehensible).

6. Derailment (Loose Associations, Flight of Ideas)

A pattern of spontaneous speech in which the ideas slip off the track onto another one which is clearly but obliquely related, or onto one which is completely unrelated. Things may be said in juxtaposition which lack a meaningful relationship, or the patient may shift idiosyncratically from one frame of reference to another. At times there may be a vague connection between the ideas, and at others none will be apparent. This pattern of speech is often characterized as sounding "disjointed." Perhaps the commonest manifestation of this disorder is a slow, steady slippage, with no single derailment being particularly severe, so that the speaker gets farther and farther off the track with each derailment without showing any awareness that his reply no longer has any connection with the question which was asked. This abnormality is often characterized by lack of cohesion between clauses and sentences and by unclear pronoun referents.

Although less severe derailments (i.e., those in which the relationship between juxtaposed ideas is oblique) have sometimes been referred to in the past as tangentiality or as flight of ideas when in the context of mania, such distinctions are not recommended because they tend to be unreliable. Flight of ideas is a derailment which occurs rapidly in the context of pressured speech. Tangentiality has been defined herein as a different phenomenon in that it occurs as the immediate response to a question.

Example. Interviewer: "Did you enjoy doing that?" Patient: "Um-hm. Oh, hey, well, I, I, oh, I really enjoyed some communities I tried it, and the next day when I'd be going out, you know, um, I took control like, uh, I put, um, bleach on my hair in, in California. My roommate was from Chicago and she was going to the junior college. And we lived in the Y.W.C.A. so she wanted to put it, um, peroxide on my hair, and she did, and I got up and looked at the mirror and tears came to my n eyes. Now do you understand, I was fully aware of what was going on but why couldn't I, why, why the tears? I can't understand that, can you?" Interviewer: "No." Patient: "Have you experienced anything like it?" Interviewer: "You just must be an emotional person, that's all." Patient: "Well, not very much I mean, what if I were dead? It's funeral age. Well, I, um? Now I had my toenails, uh, operated on. They're, uh, um, got infected and I wasn't able to do it but they wouldn't let me at my tools. Well."

0 No derailment.

1 Mild (occurs once during an interview).

2 Moderate (occurs from two to four times).

3 Severe (occurs 5 to 10 times).

4 Extreme (occurs more than 10 times, or so often that the interview is incomprehensible).

7. Incoherence (Word Salad, Jargon Aphasia, Schizophasia, Paragrammatism)

A pattern of speech which is essentially incomprehensible at times. The incoherence is due to several different mechanisms, which may sometimes all occur simultaneously. Sometimes portions of coherent sentences may be observed in the midst of a sentence which is incoherent as a whole. Sometimes the disturbance appears to be at a semantic level, so that words are substituted in a phrase or sentence so that the meaning seems to be distorted or destroyed; the word choice may seem totally random or may appear to have some oblique connection with the context. Sometimes "cementing words" (coordinating and subordinating conjunctions such as "and," "although"; adjectival pronouns such as "the," "a," and "an") are deleted.

Incoherence is often accompanied by derailment. It differs from derailment in that the abnormality in incoherence occurs *within* the level of the sentence or clause, which contains words or phrases that are joined incoherently. The abnormality in derailment involves unclear or confusing connections between larger units, such as sentences or clauses.

This type of language disorder is relatively rare. When it occurs, it tends to be severe or extreme, and mild forms are quite uncommon. It may sound quite similar to a Wernicke's aphasia or jargon aphasia, and in these cases the disorder should only be called incoherence (thereby implying a psychiatric disorder as opposed to a neurological disorder) when history and laboratory data exclude the possibility of a known organic etiology and formal testing for aphasia is negative.

Exclusions. Mildly ungrammatical constructions which occur when a person is searching for the right word, phrase, or idea should not be rated as incoherence. (For example, "My father, he, for a long time, well, he just started . . . he joined the church and became a, I say he's a Christian now because he used to lie and run around a lot.") Idiomatic usages characteristic of particular regional or ethnic backgrounds, lack of education, or low intelligence should also not be rated as incoherence. ("He ain't got no family." "That there was no good." "The lawn needs mowed." "He took the tools down cellar.")

Examples. Interviewer: "Why do you think people believe in God?" Patient: "Um, because making a do in life. Isn't none of that stuff about evolution guiding, isn't true anymore now. It all happened a long time ago. It happened in eons and eons and stuff they wouldn't believe in him. The time that Jesus Christ people believe in their thing people believed in, Jehovah God that they didn't believe in Jesus Christ that much."

Interviewer: "Um, what do you think about current political issues like the energy crisis?" Patient: "They're destroying too many cattle and oil just to make soap. If we need soap when you can jump into a pool of water, and then when you go to buy your gasoline, m-my folks always thought they should, get pop but the best thing to get, is motor oil, and, money. May-may as well go there and, trade in some, pop caps and, uh, tires, and tractors to grup, car garages, so they can pull cars away from wrecks, is what I believed in. So I didn't go there to get no more pop when my folks said it. I just went there to get a ice-cream cone, and some pop, in cans, or we can go over there to get a cigarette. And it was the largest thing you do to-to get cigarettes 'cause then you could trade off, what you owned, and go for something new, it w-it was sentimental, and that's the only thing I needed was something sentimental, and there wasn't anything else more sentimental than that, except for knick-knacks and most knick-knacks, these cost 30 or 40 dollars to get, a good billfold, or a little stand to put on your desk." Interviewer: "How do you think President Carter's doing?" Patient: "Far as I'm concerned he's probably doing all right as an individual but, he's making too many mistakes, uh, not intentional, he just, uh, w-searching for the right loopholes, when he claims a, response."

0 No incoherence.

1 Mild (occurs once during an interview).

2 Moderate (occurs from two to four times).

3 Severe (occurs 5 to 10 times).

4 Extreme (occurs more than 10 times, or so frequently that the interview is incomprehensible).

8. Illogicality

A pattern of speech in which conclusions are reached which do not follow logically. This may take the form of *non sequiturs* (= it does not follow), in which the patient makes a logical inference between two clauses which is unwarranted or illogical. It may take the form of faulty inductive inferences. It may also take the form of reaching conclusions based on faulty premises without any actual delusional thinking.

Exclusions. Illogicality may either lead to or result from delusional beliefs. When illogical thinking occurs within the context of a delusional system, it should be subsumed under the concept of delusions and not considered a separate phenomenon representing a different type of thinking disorder. Illogical thinking which is clearly due to cultural or religious values or to intellectual deficit should also be excluded.

Example. "Parents are the people that raise you. Anything that raises you can be a parent. Parents can be anything, material, vegetable, or mineral, that has taught you something. Parents would be the world of things that are alive, that are there. Rocks, a person can look at a rock and learn something from it, so that would be a parent."

0 No illogicality.

1 Mild (occurs once during an interview).

2 Moderate (occurs from two to four times).

3 Severe (occurs 5 to 10 times).

4 Extreme (occurs more than 10 times, or so frequently that the interview is incomprehensible).

9. Clanging

A pattern of speech in which sounds rather than meaningful relationships appear to govern word choice, so that the intelligibility of the speech is impaired and redundant words are introduced. In addition to rhyming relationships, this pattern of speech may also include punning associations, so that a word similar in sound brings in a new thought.

Example. "I'm not trying to make noise. I'm trying to make sense. If you can make sense out of nonsense, well, have fun." "I'm trying to make sense out of sense. I'm not making sense [cents] anymore. I have to make dollars."

0 No clanging.

1 Mild (occurs once during an interview).

2 Moderate (occurs from two to four times).

3 Severe (occurs 5 to 10 times).

4 Extreme (occurs more than 10 times, or so frequently that the interview is incomprehensible).

10. Neologisms

New word formations. A neologism is defined here as a completely new word or phrase whose derivation cannot be understood. Sometimes the term "neologism" has also been used to mean a word which has been incorrectly built up but with origins which are understandable as due to a misuse of the accepted methods of word formation. For purposes of clarity, these should be referred to as word approximations (q.v.). Neologisms are quite uncommon.

Examples. "I got so angry I picked up a dish and threw it at the geshinker." "So I sort of bawked the whole thing up."

0 Absent.

1 Mild (Using one neologism during an interview).

2 Moderate (using from two to four neologisms during an interview).

3 Severe (using more than five neologisms in an interview).

11. Word Approximations (Paraphasia, Metonyms)

Old words which are used in a new and unconventional way, or new words which are developed by conventional rules of word formation. Often the meaning will be evident even though the usage seems peculiar or bizarre (i.e., a ballpoint pen referred to as "paperskate," etc.). Sometimes the word approximations may be based on the use of stock words, so that the patient uses one or several words repeatedly in ways that give them a new meaning (i.e., a watch may be called a "time vessel," the stomach a "food vessel," a television set a "news vessel," etc.).

Exclusions. Semantic and phonemic paraphasias should be included in this category only if the results of formal testing for aphasia are negative. Sometimes incoherent speech may seem to be based on possible semantic paraphasias in the absence of positive results on formal aphasia testing. Such cases should be considered to represent incoherence if the substitutions occur frequently, and the category of word approximations should be restricted to cases where semantic substitutions occur relatively infrequently. Words used metaphorically should not be considered as word approximations (e.g., "I'm just a pin cushion or an ashtray to the rest of the world.").

Examples. "Southeast Asia, well, that's like Middle Asia now." "His boss was a seeover."

0 Absent.

1 Mild (using one word approximation during an interview).

2 Moderate (using from two to four word approximations during an interview).

3 Severe (using more than five word approximations during an interview).

12. Circumstantiality

A pattern of speech which is very indirect and delayed in reaching its goal idea. In the process of explaining something, the speaker brings in many tedious details and sometimes makes parenthetical remarks. Circumstantial replies or statements may last for many minutes if the speaker is not interrupted and urged to get to the point. Interviewers will often recognize circumstantiality on the basis of needing to interrupt the speaker in order to complete the process of history-taking within an allotted time. When not called circumstantial, these people are often referred to as "long-winded."

Exclusions. Although it may coexist with instances of poverty of content of speech or loss of goal, it differs from poverty of content of speech in containing excessive amplifying or illustrative detail and from loss of goal in that the goal is eventually reached if the person is allowed to talk long enough. It differs from derailment in that the details presented are closely related to some particular idea or goal and in that the particular goal or idea must, by definition, eventually be reached.

0 No circumstantiality.

1 Mild (occasional circumstantial reply or description during an interview, but patient can get to the point quickly if interrupted and urged to do so).

2 Moderate (several circumstantial replies or descriptions during an interview, or single replies often last at least 5 minutes, or patient continues to use circumstantial pattern sometimes if interrupted).

3 Severe (many circumstantial replies or descriptions during an interview, or any single reply of a characteristic circumstantial nature lasting more than 15 minutes, or patient usually continues circumstantial pattern even when interrupted).

13. Loss of Goal

Failure to follow a chain of thought through to its natural conclusion. This is usually manifested in speech which begins with a particular subject, wanders away from the subject, and never returns to it. The patient may or may not be aware that he has lost his goal. This often occurs in association with derailment.

0 No loss of goal.

1 Mild (one failure to follow a topic through to a logical conclusion during an interview).

2 Moderate (two to four failures to follow a topic through to a logical conclusion during an interview).

3 Severe (five or more failures to follow a topic through to a logical conclusion during an interview).

14. Perseveration

Persistent repetition of words, ideas, or subjects so that, once a patient begins a particular subject or uses a particular word, he continually returns to it in the process of speaking.

Exclusions. This differs from "stock words" in that the repeated words are used in ways appropriate to their usual meaning. Some words or phrases are commonly used as pause-fillers, such as "you know" or "like"; these should not be considered perseverations.

Examples. "I think I'll put on my hat, my hat, my hat, my hat." Interviewer: "Tell me what you are like, what kind of person you are." Patient: "I'm from Marshalltown, Iowa. That's 60 miles northwest, northeast of Des Moines, Iowa. And I'm married at the present time. I'm 36 years old. My wife is 35. She lives in Garwin, Iowa. That's 15 miles southeast of Marshalltown, Iowa. I'm getting a divorce at the present time. And I am at presently in a mental institution in Iowa City, Iowa, which is a hundred miles southeast of Marshalltown, Iowa."

0 No perseveration.

1 Mild (has a persistent repetition of one set of words or ideas).

2 Moderate (has persistent repetition of two or three different sets of words or ideas).

3 Severe (has persistent repetition of four or more different sets of words or ideas).

15. Echolalia

A pattern of speech in which the patient echoes the words or phrases of the interviewer. Typical echolalia tends to be repetitive and persistent. The echo is often uttered with a mocking, mumbling, or staccato intonation. Echolalia is relatively uncommon in adults, but more frequent in children.

Exclusions. Some people habitually echo questions, apparently to clarify the question and formulate their answer. This is usually indicated by rewording the question or repeating the last several words (i.e., from "What did you wear yesterday?" to "What did I wear yesterday?" or "Wear yesterday?").

Example. The doctor says to the patient, "I'd like to talk with you for a few minutes." The patient responds with a staccato intonation, "Talk with you for a few minutes."

0 Absent.

1 Mild (echoes words or phrases once during an interview).

2 Moderate (echoes words or phrases from two to four times during an interview).

3 Severe (echoes words or phrases five or more times during an interview).

16. Blocking

Interruption of a train of speech before a thought or idea has been completed. After a period of silence, which may last from a few seconds to minutes, the person indicates that he cannot recall what he had been saying or meant to say. Blocking should only be judged to be present either if a person voluntarily describes losing his thought or if upon questioning by the interviewer, the person indicates that that was his reason for pausing.

0 Absent.

1 Mild (occurs once during an interview).

2 Moderate (occurs two to four times).

3 Severe (occurs five or more times).

17. Stilted Speech

Speech which has an excessively stilted or formal quality. It may seem rather quaint or outdated, or it may appear pompous, distant, or overly polite. The stilted quality is usually achieved through the use of particular word choices (multisyllabic when monosyllabic alternatives are available and equally appropriate), extremely polite phraseology ("Excuse me, madam, may I request a conference in your office at your convenience?"), or stiff and formal syntax ("Whereas the attorney comported himself indecorously, the physician behaved as is customary for a born gentleman").

0 No stilted speech.

1 Mild (one or two instances of stilted speech during an interview).

2 Moderate (frequent instances of stilted speech).

3 Severe (most answers to questions and spontaneous speech are stilted).

18. Self-Reference

A disorder in which the patient repeatedly refers the subject under discussion back to himself when someone else is talking and also refers apparently neutral subjects to himself when he himself is talking. This finding usually cannot be evaluated on the basis of a psychiatric interview, since the subject is then asked to talk about himself. It may be observed during the tests of the sensorium or informal conversation about neutral subjects and should be rated only in that context.

Example. Interviewer: "What time is it?" Patient: "Seven o-clock. That's my problem. I never know what time it is. Maybe I should try to keep better track of the time."

0 Absent.

1 Mild (self-reference occurs once during a 15-minute discussion of a neutral subject).

2 Moderate (self-reference occurs two to four times during a 15-minute discussion of a neutral subject).

3 Severe (self-reference occurs five or more times during a 15-minute discussion of a neutral subject).

19. Paraphasia, Phonemic

Recognizable mispronunciation of a word because sounds or syllables have slipped out of sequence. Severe forms occur in aphasia, but milder forms may occur as "slips of the tongue" in everyday speech. The speaker usually recognizes his error and may attempt to correct it.

Example. "I sipped on the lice and broke my arm while running to catch the bus."

0 Absent.

1 Mild (one instance of phonemic paraphasia during an interview).

2 Moderate (two to four instances of phonemic paraphasia during an interview).

3 Severe (five or more instances of phonemic paraphasia during an interview).

20. Paraphasia, Semantic

Substitution of an inappropriate word when trying to say something specific. The speaker may or may not recognize his error and attempt to correct it. This typically occurs in both Broca's and Wernicke's aphasia. It may be difficult to distinguish from incoherence since incoherence may also be due to semantic substitutions which distort or obscure meaning; when this differential decision must be made, it is suggested that formal testing for aphasia be completed; if the testing is positive, then the semantic substitutions may be considered due to semantic paraphasia, and if negative to incoherence.

Example. "I slipped on the coat, on the i-i-ice, I mean, and broke my book."

0 No instances of semantic paraphasia.

1 Mild (one instance of semantic paraphasia during an interview and aphasia testing positive.

2 Moderate (2 to 10 instances of semantic paraphasia during an interview and aphasia testing positive).

3 Severe (11 to 20 instances of semantic paraphasia during an interview and aphasia testing positive).

4 Extreme (more than 20 instances of semantic paraphasia during an

interview, or so frequently that the interview is incomprehensible, and aphasia testing positive).

Global Rating of TLC Disorder (Excluding Semantic and Phonemic Paraphasias)

The global assessment of the overall severity of the TLC disorder may be approached in two ways. It may literally be rated globally, using the rating scale provided below. This global rating should reflect the recognition that some TLC disorders are more pathological than others. Circumstantiality or stilted speech are not as likely to suggest severe psychopathology as are incoherence or derailment.

An alternative method is to use the illustrated listing to summate the scores on each of the TLC ratings. Using this method, the rating for each TLC variable should be multiplied by 2 in the case of the more pathological variables and by 1 in the case of the less pathological; summing of the resulting scores will give a more quantitative measure of the severity of the TLC disorder.

Listing to summate scores

More pathological	Less pathological
Poverty of speech	Circumstantiality
Poverty of content of speech	Loss of goal
Pressure of speech	Perseveration
Distractible speech	Blocking
Derailment	Echolalia
Tangentiality	Stilted speech
Incoherence	Self-reference
Illogicality	
Clanging	
Neologisms	
Word approximations	

0 No TLC disorder. Occasional instances of the less pathological forms and no more than one instance of the more pathological (which is felt in context to be clinically insignificant).

1 Mild TLC disorder. Occasional instances of TLC disorder which are felt in context to be mild but clinically significant.

2 Moderate TLC disorder. Significant and unquestionable impaired verbal output which leads to a moderate disturbance in communication at least from time to time.

3 Severe TLC disorder. Disorder significant enough to impair communication for a substantial part of the interview; many instances of the more pathological manifestations of TLC.

4 Extreme TLC disorder. TLC disorder so severe that communication is difficult or impossible most of the time.

TLC Score Sheet

1. Poverty of speech	0	1	2	3	4
2. Poverty of content of speech	0	1	2	3	4
3. Pressure of speech	0	1	2	3	4
4. Distractible speech	0	1	2	3	4
5. Tangentiality	0	1	2	3	4
6. Derailment	0	1	2	3	4
7. Incoherence	0	1	2	3	4
8. Illogicality	0	1	2	3	4
9. Clanging	0	1	2	3	4
10. Neologisms	0	1	2	3	
11. Word approximations	0	1	2	3	
12. Circumstantiality	0	1	2	3	
13. Loss of goal	0	1	2	3	
14. Perseveration	0	1	2	3	
15. Echolalia	0	1	2	3	
16. Blocking	0	1	2	3	
17. Stilted speech	0	1	2	3	
18. Self-reference	0	1	2	3	
Global rating	0	1	2	3	4

Appendix. Kappa values of definitions of thought, language, and communication disorders in psychiatric patients (*n* = 113)

	Full scale weighted Kappa	Present/absent unweighted Kappa
Poverty of speech	.81	.75
Poverty of content of speech	.77	.62
Pressure of speech	.89	.82
Distractible speech	.78	.78
Tangentiality	.58	.49
Derailment	.83	.71
Incoherence	.88	.91
Illogicality	.80	.69
Clanging	.58	.53
Neologisms	.39	.49
Word approximations	−.02	−.02
Circumstantiality	.74	.80
Loss of goal	.70	.65
Perseveration	.74	.46
Echolalia	.59	.42
Blocking	.79	.71
Stilted speech	.70	.32
Self-reference	.50	.36

Scoring Manual for the Thought Disorder Index

Original version by Mary Hollis Johnston and Philip S. Holzman[1]

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The Thought Disorder Index (TDI) tags, classifies, and measures instances of disordered thinking. It is especially useful because it permits a trained judge, even when without knowledge of other behaviors, to derive a qualitative and quantitative assessment of cognitive slippage.

Whatever source the verbal material comes from, it is important that it be transcribed verbatim and contain a thorough inquiry of the thought slippage. The verbatim transcript is necessary to ensure that the complete verbal sample is recorded and available for assessment; the thorough inquiry is necessary to ensure that the subject has an opportunity to clarify the response and that the rater has the full information needed to classify the response.

In a scored protocol, each instance of thought slippage is characterized both quantitatively and qualitatively, and contains the following information:

- Total TDI score (which is the sum of each instance of thought disorder weighted by its severity level, corrected for response productivity, expressed as a percentage).
- Number of instances of thought disorder scored at each level.
- Number of instances of thought disorder scored for each category.

From this information, one may obtain an assessment of the severity of the subject's thought disorder and to what qualities of thinking this severity may be attributed. A high TDI score may result from a few instances of severe thought slippage, or many instances of mild slippage. Similarly, one subject's thought disorder may be characterized by problems with disorganization, whereas another's may be characterized by incongruous combinatory thinking.

Although raters should not know the diagnoses of the subjects, raters should be aware of the education, social class, and ethnic background of the subjects, since certain expressions may be evidence of disorderd thought in some circles but may be acceptable usage in others.

When a record is being assessed for thought disorder, a score is given each time thought disorder is exhibited. The question often arises as to whether a response may be given more than one thought disorder score. The rule of thumb is as follows:

In general, the score given is that which best captures the process evident in the response. For example, a subject may give a *Confabulation* response that carries within it a *Fabulized Combination*. If the *Fabulized Combination* is part of the confabulatory process, and not a separate instance of slippage, the response is given a single score of confabulation. On the other hand, if during the course of giving a *Confabulation* response, a subject produces a *Peculiar Verbalization* as well, the subject would be given a score for *Confabulation* and a score for *Peculiar Verbalization* because the *Peculiar Verbalization* is not part of the confabulatory process.

Several of the categories are broken down into more specific subcategories: *Inappropriate Distance, Peculiar Verbalization and Responses, Incongruous Combinations, Idiosyncratic Symbolism, Queer Responses*, and *Confabulations*. For the most part, subcategories are given as heuristic guides to scoring the categories and are provided solely to clarify various instances of use for that category. They are not meant to be scored in their own right. In fact, certain categories such as *Peculiar Verbalizations* contain subcategories that are difficult to distinguish reliably.

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[1] Johnson and Holzman (1979).

Subjects occasionally produce instances of thought disorder that can be characterized by a particular category, although the slippage is not quite so severe as that category warrants. In such instances, one can score a "Tendency" to the particular category, which means that the score is weighted by the immediately lower level of severity. For example, a *Tendency to Confabulation* would be scored at the 0.5 level instead of the 0.75 level given to full *Confabulations*. Obviously, one can only score a tendency for categories at the 0.5, 0.75, or 1.0 levels. Although a tendency may be scored for any of the categories, the need arises more often for certain of these categories than others. These are: *Confusion, Looseness, Fluidity, Autistic Logic, Confabulation, Incoherence,* and *Contamination*.

It is important that the examiner not deviate from the task, and generally not inquire about the subject's personal associations that do not relate to the task of responding to the test questions.

In the administration of the Wechsler Adult Intelligence Scale (WAIS), any odd verbalizations should be inquired into by the examiner.

The TDI is usually derived from verbatim responses to the Rorschach Test and at times from the verbal subscales of the WAIS. In administering the WAIS and the Rorschach, it is most important that the examiner inquire into all deviant verbalizations, percepts, slips, and reasoning.

The examiner should adopt a naive stance toward the test administration, and should not hesitate to inquire in order to clarify a point.

The examiner should not assume that s/he can figure out what a subject meant; deviant verbalizations, strange percepts, and ambiguous or elliptical responses should always be clarified through inquiry. Often a simple restatement of the subject's own words (e.g., "nose of a spider?") will be enough of a prompt to elicit an explanation from the subject. Otherwise, questions such as, "Can you say a little more about that?" or "I don't quite understand what you mean by ______," should suffice. It is better to err on the side of overinquiring than underinquiring.

List of TDI Categories (Revised)

0.25 Level

1. Inappropriate distance
    a. Loss or increase of distance
    b. Excessive qualification
    c. Concreteness
    d. Overspecificity
    e. Syncretistic response
2. Flippant response
3. Vagueness
4. Peculiar verbalizations and responses
    a. Peculiar expression
    b. Stilted, inappropriate expression
    c. Idiosyncratic word usage
5. Word-finding difficulty
6. Clangs
7. Perseveration
8. Incongruous combinations
    a. Composite response
    b. Arbitrary form-color response
    c. Inappropriate activity response
    d. External-internal response (score at 0.50 level).

0.50 Level

9. Relationship verbalization
10. Idiosyncratic symbolism
    a. Color symbolism
    b. Image symbolism
11. Queer responses
    a. Queer expressions
    b. Queer imagery
    c. Queer word usage
12. Confusion
13. Looseness
14. Fabulized combinations, impossible or bizarre
15. Playful confabulation
16. Fragmentation

0.75 Level

17. Fluidity
18. Absurd responses
19. Confabulations
    a. Details in one area generalized to larger area
    b. Extreme elaboration
20. Autistic logic

1.0 Level

21. Contamination
22. Incoherence
23. Neologisms

Description of the TDI Levels and Categories

0.25 Level

This level is characterized by minor idiosyncracies that would only rarely be noticed in ordinary conversation, although an accumulation of them would result in a lack of clarity. These are the most subtle signs of thought disorder that may be difficult for an untrained observer to detect. One may have the feeling that the speaker is not quite "with" the task, that there is some slight intrusion of an idiosyncratic set, or that the respondent has at least some difficulty in maintaining a clear unambiguous focus. Category descriptions are given to help the scorer record "minor idiosyncracies," and the definitions include some rules to help make the judgments of the scorer easier. A common reaction of beginning scorers is to assume that since most people occasionally make comments that could be scored at the 0.25 level, these comments should not be considered instances of disordered thinking. Although the categories subsumed under the 0.25 level are displayed even by the healthiest of individuals, most frequently during periods of anxiety or fatigue, they are still episodes of mild cognitive slippage and are viewed as such in this scoring system.

1. Inappropriate Distance

Inappropriate Distance refers to the psychological distance that the subject places between him/herself and a task such as the Rorschach Test. If the subject displays an *Increase of Distance*, his/her responses will show little regard for the task or will be overelaborated associatively. The point is that the responses seem to be dictated insufficiently by the task at hand; rather, they are heavily influenced by personal associations. A *Loss of Distance* is apparent when the subject responds to the task as being too real or interprets the task too literally. In other words, the subject does not maintain the appropriate perspective, and the result might be an overly concrete or emotionally charged response to the blot. There are five subcategories subsumed under *Inappropriate Distance*.

a. Loss or Increase of Distance. Responses falling under this subcategory are often but not always characterized by emotionally charged affective elaboration. A personalized response is considered a *Loss of Distance* when the task itself is taken as being too real or too personal.

> Example: (IV) I'm afraid of what else it could be . . . it scares me to think of what else it could be . . . overpowering.

A personalized response is considered an *Increase of Distance* when the affective elaboration results from a personal association to the task rather than from the task itself.

> Example: (III) It looks like a butterfly . . . it has two wings . . . sometimes they're alike and . . . I like butterflies. I love butterflies. Butterflies are beautiful.

The distinction made above regarding the *Loss* or *Increase of Distance* of these responses is not necessary to make when scoring personalized responses. The distinction that must be made is the point at which such utterances should be scored as thought disorder. At the 0.25 level of scoring *Loss* or *Increase of Distance*, the affective intrusion does not prevent the subject from performing adequately, but the personal associations tend to intermingle with adequate communication. Sometimes this intermingling causes an originally good response to be spoiled or contradicted. In scoring personally elaborated responses, the scorer must keep in mind how appropriate the final answer is to the task at hand. If there is no attempt to relate the personalizing to the task, this indicates *Inappropriate Distance*.

> Example from the WAIS (similarity between praise and punishment): Praise—like praising the Lord. And punishment when you did something wrong and you get punished for it and then you pray for the Lord to forgive you. For forgiveness. And you say, "God, I have sinned. Will you please forgive me?"

> Examples from the Rorschach: This looks like a baby's head. . . . There are two of them really. I don't know what they're doing. I just really don't like to look at this thing.
>
> (And you saw an island?) Uh-huh. I'm kind of against all this. I don't like . . . you know, letting my imagination flow unless it's with somebody I love.
>
> A bug. Oh God. I hate bugs. I hate bugs. I want to kill every one I see.
>
> These two here, I don't know. I'm hungry right now so they look like food.
>
> These look like crabs. . . . I love crabs, did you ever eat crab?
>
> It looks like a vagina because it reminds me of the way my own vagina feels.
>
> Ooh! That's too horrible, take it away, I can't stand to look at it.

In order to be scored as a *Loss* or *Increase of Distance*, an association must disrupt the task. Sometimes subjects will ask questions about the task itself or make personal comments that are still very much focused on completing the task. The following are instances when personal comments *should not be scored*: (a) comments about having taken the test before; (b) remembering prior responses; (c) asking at the end of a response what inkblot tests really mean; (d) social comments that do not occur in the middle of a response, thereby not disrupting or spoiling it;
(e) comments about actual "extratest" events (e.g., a loud noise).

> Examples of responses *not* scored: (Why does it look like a vampire?) Because it looked just like those vampires my kids watch on "Creature Features." When I was a kid, I used to be scared to death of those things.
>
> (If you were lost in the forest . . .) That happened to us once, we found a stream and followed it.

b. Excessive Qualification. In an excessively qualified response, the subject has trouble giving a response because the inkblot is not a perfect likeness of whatever percept the subject considers. A subject may offer modifications that the percept must have before it merits a given response or a subject may not give any response at all because the inkblot does not conform to his/her expectations of what it should look like. Rigid perfectionism appears to be interfering with a spontaneous response.

> Example: (I) S'like a . . . leaf. Doesn't really look like a leaf . . . I dunno. Well, actually, it couldn't be a leaf because of its color, right? I mean you don't find black leaves . . . too often, do you, unless they get older. Off'n some trees you would, but they usually turn to brown and just disintegrate. . . .
>
> Example: (VI) . . . Looked kinda feathery. Suggested Indians, a totem pole, or somethin'. (What made it look feathery?) Wh . . actually it wasn't feathery. I don't know. I don't even know why I said that. It doesn't really look feathery. If it were feathery, there would be more shading.
>
> Example: (V) Looks pretty much like a bat but got a body that looks a little bit like a mouse . . . a little bit more like an insect though, you know. (The bat?) . . . At the end of their wings they usually have claws or something, you know? The claws would probably be off in this area, but, uh, but they're misplaced you know. (Body like a mouse?) Well, I didn't really say a mouse, either, kinda like an insect again . . . can't really fit any of the three.

c. Concreteness. The response process of *Concreteness* suggests a loss of perspective in which the task takes on an apparent reality. The subject does not give a sufficiently abstract response to the task.

> Examples from the WAIS: (What direction would you go if you were going from Chicago to Panama?) If you went by airplane, I imagine you would go south.
>
> (Similarity between north-west) They're both opposite south and east.
>
> (How far is it from New York to Paris?) Oh, I don't know. Eight-hour flight.
>
> Examples from the Rorschach: (VIII) . . . an insect that's grown huge—oversized. (Okay, what made it look like an overgrown insect?) I could see the outline of an insect, but it was too big to be a normal insect so it had to be overgrown.
>
> (VII) Some kind of fancy military jet flying up the card.

d. Overspecificity. The overspecific response is similar to the excessively qualified response in that an obsessive disposition has become exaggerated in both cases. However, in an overspecific response, the scorer should look for arbitrary, irrelevant specifications that spoil a response and give it an overideational quality. The subject is attempting to be precise, but fails because of excessive obsessionality or compulsivity, and thus tends toward absurdity or bizarreness.

> Examples from the WAIS: (What is the theme of the book of Genesis?) It's, uh . . . a fairy tale . . . tryin' to describe to the . . . proletariat masses how . . . life began . . . for human beings.
>
> (What is similar about a coat and a dress?) They both hang . . . an' they're opening at the bottom, mmm . . . they could have buttons, both of them, or zippers. An' they're both worn as clothing.
>
> Examples from the Rorschach: (III) A four-legged lamb.
>
> (III) A bone of a bear.

e. Syncretistic Response. Erring in the opposite direction of *Overspecificity*, the subject is here too general and abstract; instead of restricting a conceptual class, s/he includes too much in it. The *Syncretistic Response* is seen most often on items in the Similarities subtest of the WAIS.

> Examples from the WAIS: (What is similar about a fly-tree?) They both live in the air.
>
> (What is similar about an orange-banana?) They both contain atoms.

2. Flippant Response

A *Flippant Response* also reflects a problem with distance in that the subject adopts a stance toward the testing situation that betrays an absence of seriousness or a tendency to dismiss the test-taking with wisecracks. *Flippant Responses* are often humorous, and may be very engaging in informal conversation; however, when such remarks are disruptive and inappropriate to the testing situation, they are scored. A difficulty that arises in scoring these responses is that their inappropriateness is due to the situation in which they occur, instead of to the malignancy of the response itself. Whereas there might be no indication of slippage when one makes a flippant response in ordinary conversation, it becomes a problem if one fails to inhibit these responses in the testing situation.

> Example: (IX) I see another vagina, I guess I'm a sex maniac. . . . Oh, that's getting written down? Uh-oh, they'll tell the police about me.
>
> Example: (IX) If I had known I was taking this test today, I would have boned up on this section.

3. Vagueness

The vague response conveys no clear meaning. However, *Vagueness* is sometimes used to mask an absence of information. When this is the case, it may not have the same pathological weight as it does when used in a context of confusion and autistic thinking. Because it is difficult to distinguish between the two causes of *Vagueness*, it is scored at a low level, 0.25. In these responses, the subject appears to be attempting to avoid giving inappropriate responses (in which s/he succeeds); nevertheless, s/he does not succeed in giving any real information. More serious difficulty might result if the subject failed to inhibit the irrelevant material; such failure would be *Confusion, Incoherence*, or *Absurdity*, which are scored at higher levels.

Vagueness can include both long, meandering speeches as well as excessively short, cryptic statements that carry hardly any specific information.

> Example: (How are a poem and statue alike?) Could be history.
>
> Example: (What does "sentence" mean?) It's something very short in the way of speech reading.
>
> Example: (What is the Vatican?) It's in Rome, Pope. (Explain more.) That's all connected together. The Catholic religion.
>
> Example: (Why does the state require a license in order to get married?) Legal, legalities. That's a state issue why it's necessary, legality is involved.

On the Rorschach, the subject appears unable to elaborate any clear percept:

> Example: Nothing but two figures on each side. (What made them look like that?) I don't know what kind of figures. They just looked like two smears.

The subject may give a response without communicating clearly what s/he is seeing:

> Example: Picture of like depth and stuff, like of distance . . . where your eye level would be.
>
> Example: Sort of strange and unclear . . . definition of . . . unclear . . . totally confusing . . . grotesque.
>
> Example: . . . they all could be animals, I - I, it's too much in general. I don't know . . . .

4. Peculiar Verbalizations and Responses

Three kinds of *Peculiar Responses* are included under this category:

- Peculiar Expressions
- Stilted, Inappropriate Expressions
- Idiosyncratic Word Usage

As stated earlier, distinctions among these three subtypes (especially between the first two) are not firmly established, but they are presented as heuristic guides to scoring the general class of verbalizations as *Peculiar*.

Peculiar Verbalizations include quaint, indiosyncratic, private terms or expressions in which the meaning may be clear, but the expression itself is unusual. There may be an unusual combination of words resulting in strange expressions, or incongruous modifiers. Generally, the scorer must be alert for stilted, strained, illogical, and unusual expressions that are not part of the conventional modes of response to the various test stimuli. Some pretentious or stilted expressions that are part of a cultural or ethnic mode of responding are not scored. These exceptions are noted in the examples.

a. Peculiar Expression. Odd combinations of words, perhaps with reiteration or contradiction within a phrase, result in incongruity or a peculiar, inappropriate, or anthropomorphic image.

> Examples from the WAIS: (What is the definition of breakfast?) Morning dinner.
>
> (Why are dark-colored clothes warmer than light-colored clothes?) Because the sun warms up to them . . . they retain the heat of the sun.
>
> Examples from the Rorschach: A reverse reflection
>
> Ears looking forward
>
> Bearskin rug . . . you know how they scatter out on each side.

b. Stilted, Inappropriate Expression. These are awkwardly phrased and stilted sounding expressions.

> Examples from the WAIS: (What is similar about a table and a chair?) Both room settings.
>
> (What is a sanctuary?) A place where a meeting can commence of a religious origin.
>
> (What does slice mean?) To subdivide a piece of meat or a loaf of bread into slices of thinner pieces. It could also mean a slice of bread or a slice of meat—a distribution of a larger whole.
>
> Examples from the Rorschach: (IV) It's a piece of animation.
>
> (VI) I was, my mind was, or my attention was being put on the center line.
>
> (IX) (What made it look poisonous? Most jellyfish that are poisonous . . . give off a lot of different colors. Nature put it that way, for a warning.
>
> (IV) (What made it look like an animal?) Its head features.
>
> (II) And this is a set of lungs.
>
> (III) . . . No, you can't see anymore unless you're looking at it from a perspective.
>
> (IV) What suggested it? Just its overall picture.

c. Idiosyncratic Word Usage. One example of *Idiosyncratic Word Usage* is inappropriate metonymy, in which one word is substituted for another of which the former may be an attribute.

> Examples from the Rorschach: He's all *clowned* up in some kind of suit.

> The entire shape, the perimeter, the *exterior shape*.
>
> It looks like an *inhuman* form, something that's not natural, something that's unnatural.
>
> Could be a *land*.

Another example of idiosyncratic word usage is when words or ideas that tend generally to occur together are equated and substituted for each other. In this respect, the educational and sociocultural background of the subject should be considered when scoring, because lower-class subjects and those with low levels of education frequently interchange countries, cities, and continents as well as body parts. Therefore, this kind of interchange of words should be scored only if the subject gives evidence of being confused, of blocking on the correct word, or of being aware that s/he should have known the correct word. Thus, if a subject calls antennae *antlers* at one time and *antennae* at another, the use of *antlers* is scored as peculiar. If *antlers* is the subject's usual term for *antennae*, as inferred from the educational and social class background, it should not be scored.

Some examples of common word interchange occurring in normal subjects of low social class or low education follow. *Do not score* unless the subject has at least a high school education and indicates confusion about correct terms:

> (Where is Egypt?) In Jerusalem.
>
> (What is the capital of Italy?) I was going to say Spain, but Spain is in Portugal.
>
> Hands = paws = arms
>
> Feelers = tentacles = antennae = appendages = antlers

Similarly, do not score word interchange resulting from the subject's subcultural dialect such as Black English:

> Blood = person
>
> Fang = feeler

Frequently "word play" sufficiently alters words so that they are no longer real words but are nevertheless still easily understood. The scorer should keep in mind the education and social class of the subject, as well as whether the meaning is clear. "Word play" that approximates a correct word among subjects who do not know the correct word is not considered a scorable response. When scoring verbalizations of lower-class subjects, it is sometimes impossible to know whether a word has been produced from culturally acceptable "word play" or from fluidity in thinking. In such cases, the subject should be given the benefit of the doubt and the expression should not be scored. However, idiosyncratic use of words that are *clangs* and appear to result from an attempt to impress the examiner with pretentious-sounding words or that result from blocking should be scored.[2]

> Scorable examples from the WAIS: (How are the eye and ear alike?) They're both *necessity*.
>
> (What does "repair" mean?) Get something wrong with it right . . . *rightened*.
>
> (Why are dark-colored clothes warmer than light color clothes?) They *exorb* the sun.
>
> (What does "domestic" mean?) From some *inalien* source.
>
> *film* (rather than filament)
>
> Scorable examples from the Rorschach: It looks like a bear rug . . . because of the *outstretch*.
>
> Two *charged* elephants.
>
> Two *imprint* figures of elephants.
>
> I have *self-determine*. That's what you need in this world.
>
> That's the way I *impressioned* it.
>
> Fetuses . . . it looked like there was a tightly bunched *organ*.

5. Word-Finding Difficulty

The search for a word that the subject appears to know but on which s/he is blocking is scored as (.25) *Word-Finding Difficulty*. It is important to distinguish this problem of blocking from a simple lack of knowledge of the proper word. In order to score (.25) *Word-Finding Difficulty*, the subject must either give at least two wrong alternatives in her/his search for a word (even if the correct word is then found) *or* express clearly that s/he knows the word but cannot produce it.

> Examples: (Name three types of blood vessels) Capillaries, arteries, and it begins with a v [first try], I won't say ventricle [second try] . . . veins.
>
> What do you call those hard-shelled bugs? Beetle? No, that's not it . . . I'll say beetle, I think that's the name of a bug.
>
> (What made it look like a sea animal?) It had the shape of a . . . I can't think of the name of that animal, it's an exact reproduction. I can't think of the name but it's, umm . . . it's very common.

If a (0.25) *Peculiar Response* is given while the subject is seeking to find the right word, both may be scored as in the following example.

> There again it seems to be the same picture on the other side, as it is on the other, only *reverted*, or whatever you want to call it. I can't say reverted.

Do *not score* when the subject does not know a word and simply indicates so:

> It has those, I don't know what you call them, things up there [points].

---

[2] See *Queer Responses* (11) for guidelines on distinguishing *Peculiar* (*Idiosyncratic Word Usage*), *Queer* (*Queer Word Usage*), and *Neologisms*.

In the case of blocking on finding the appropriate word, there must be more than a temporary inefficiency. *Do not score* if the subject fumbles only once and then immediately finds the word s/he is looking for:

> That looks like, uh, what do you call it, oh yeah, antenna.

6. Clangs

Clanging is scored if the response is limited to a single, clear-cut usage of rhyming or alliteration. The subject's verbalization is determined by the sound rather than by the meaning of the word(s).

> Examples from the WAIS: (What does travesty mean?) I think of the treasure and the dynasty.
>
> (What is the population of the U.S.?) . . . millions. Billions and zillions. I don't know about . . . 500 million zillions.
>
> Examples from the Rorschach: (IX) . . . And this *mist* here kind of suggests that they live in a *mystical* world. Even their form would suggest something like that, because they have more solid form here, and then a more *misty* form here, implying that they would be a more *mystical mythical* type of animal.
>
> (VII) I said *frog*, I didn't say *log* [laughs] . . . just a *clab*. A sea *clab, crab*. . . .

7. Perseveration

Perseveration may occur on both the WAIS and the Rorschach. It is revealed by a lingering, unsuppressed, compulsively repeated idea that is forced inappropriately and arbitrarily into a response.

On the Rorschach, *Perseveration* is scored if a response that is repeated *at least three times* does not have good form—that is, when the inkblot does not justify the percept:

> (One subject saw a space ship on six Rorschach cards. On card X, he had to strain to justify his response) A part of a ship, you can't see all of it.

Repetitions of either common responses (such as "butterfly"), which conform to the inkblot, or of vague responses (such as "leaf"), which fit almost any inkblot, are not perseverations. To be scored, the response must be arbitrary: the subject may have difficulty justifying it or may strain to fit the idea to the inkblot.

> Examples from the Rorschach: Just your roots inside your body (third reference to "roots inside your body").
>
> I keep seein' fur coats. Looks like fur to me (third reference to fur coats).

8. Incongruous Combinations

Incongruous Combination is scored principally on the Rorschach, where contiguous details of the inkblot or suggested images are merged into a *single* incongruous percept. Incongruous combination is scored if the activity described is unrealistic and includes only one area of the blot. Weiner (1966) described these responses as reflecting inappropriate distance from the Rorschach cards, taking the relationships between images as real, and embellishing them unjustifiably. He described four types: composite, arbitrary form-color, inappropriate activity, and external-internal responses.

a. Composite Response. A *Composite Response* combines parts of two separate percepts into a "hybrid" creature. Both parts of the percept are accurately perceived but are combined incongruously into an inappropriate unit:

> The thing on top is a person with, for some reason, two pairs of arms outstretched.
>
> A bear with a duck's face.
>
> It looks like a cross between a cat and a jet.
>
> I see a couple of horns on the bird.
>
> It looks like a mastedon wearing shoes.

Do not score images that are justified in art or mythology (for example, a centaur) or simple descriptions of parts of the blot in which the subject notices the incongruity but does not attempt to link the incongruous parts:

> Don't know what this is. It has a head like a rat and a body like a horse. But there isn't any such animal.

It is sometimes difficult to distinguish a *Composite Response* (**8a**) from a *Peculiar Verbalization* (3c). For example, if a subject says, "antlers on a butterfly," is the word antler being substituted for antennae (i.e., a *Peculiar Verbalization*), or does the subject actually see antlers on a butterfly (i.e., a *Composite*)? In ambiguous cases such as these, it is important that the examiner inquire to clarify this distinction.

b. Arbitrary Form-Color Response (FCarb). The *FCarb Response* (see Rapaport, Gill, and Schafer 1968, pp. 369–370) involves an excessively concrete, a priori decision that because the object perceived is a particular color on the card, then it must actually be that color (for example, a pink polar bear). The inkblot is more real than what the subject knows as reality distinct from the inkblot. *Composite Responses* involve combining the form qualities of the blot, while *FCarb Responses* are condensations of two modalities, form and color. The object seen is inconsistent with the color attributed to it. Inquiry can establish whether the subject intended the arbitrary description humorously, had some specific art or literary figure in mind, or can account for it in a strained but realistic manner. *FCarb* is scored only if the subject does not show awareness of the inappropriateness of the combination.

> Examples from the Rorschach: This is a little girl right here. She's on fire, too, green fire.
>
> An orange pelvic bone. Or a small guy with two orange arms.
>
> Two crabs with green appendages.

c. Inappropriate Activity Response. This response involves the attribution of an inappropriate activity to an object, usually a human or animal figure. If the resulting image is impossible and distorts reality, it should be scored.

> Examples from the Rorschach: Like two animals having their nose tied together.
>
> A beetle crying.
>
> A man shedding his skin.
>
> Caterpillars shaking hands.

d. External-Internal Response (to be scored at the 0.5 level). This response occurs when both the external and the internal parts of an object are seen at the same time, in a realistically impossible manner.

> Examples from the Rorschach: This is a woman and down here is her ovaries.
>
> Ah . . . this kinda looks like, like a person. You know? Like this would be the nostrils, and this would be the mouth. And the person's wearing the hat. And the person's hair is down like this. An' the person's collar. Person's trachea. Let's see . . . that's all.

Although *External-Internal Responses* fit conceptually into the *Incongruous Combination* category (i.e., two aspects of a percept are combined incongruously), the severity level of this subcategory is more appropriately classified as 0.5 than as 0.25. For this reason, we have chosen to categorize it as an *Incongruous Combination*, but to score it at the 0.5 level.

0.5 Level

Many 0.25 level responses are encountered with moderate frequency in everyday discourse, but they rarely convey the impression that the subject has lost contact with reality. Rather, they suggest that the subject may have trouble dealing appropriately with some tasks or may respond idiosyncratically, but not bizarrely, to some situations. Such a person may be "on the fringe" but is still in tune with her/his surroundings. People who are severely anxious, fatigued, or under some temporary systemic distress may give several .25 responses. In contrast, an accumulation of .5 responses conveys an impression of loss of mooring, shaky reality contact, emotional overreaction, and distinct oddness.

9. Relationship Verbalization

Relationship Verbalization is scored when the subject either repeats a response previously given or offers a new response, but relates the present response to the former one. The subject makes connections between responses.

> (A patient related each card to the preceding one in a kind of story sequence, carrying the story a bit further with each card.) You have the same thing here, except for the pig's being slaughtered. . . . Now the devil has destroyed the animal and the butterfly.
>
> There again you have that expansive thing . . . I find it to be prevalent in quite a few of your pictures.
>
> An allusion back to an earlier one, that being the mandibles in the very first picture.
>
> It's in pieces. That's the other part of that [points to previous card], I believe.

10. Idiosyncratic Symbolism

Two types of symbolic Rorschach responses can be distinguished (Weiner 1966; Rapaport, Gill, and Schafer 1968); one involves interpretation of the meaning of the color or shading, and the other makes use of concrete images to represent abstract ideas. We have divided *Idiosyncratic Symbolism* into two subcategories:

- Color Symbolism
- Image Symbolism

It is important to note that symbolic responses do not always represent disordered thinking and should be scored only when they take idiosyncratic rather than conventional forms.

a. Color Symbolism. Holt and Havel (1960) gave examples of conventional color symbolism that include black as evil, red as anger, green as envy, blue as coldness, and yellow as warmth. Such responses are not scored, nor are simple color responses (where color is used as a primary, secondary, or sole determinant of the response) scored as symbolism. For example, "This is a plant because it's green." Symbolic color or shading responses are scored only when the interpretation is idiosyncratic.

> Examples: . . . and, of course, the orange is symbolic of hell. The red shows action.
>
> And the fading of this dead world indicates the coming of the new world, which is highly green, symbol of a new world.

b. Image Symbolism. The use of concrete images to represent abstract ideas is scored when such symbolism is idiosyncratic and is given with an air of reality (rather than as playfully imaginative).

> Examples: Two men beating the drums . . . like Siamese twins. Their hearts would beat at the same time. This would be symbolic of their hearts beating together.
>
> Two objects right here might seem to be horns of the devil. Horns might represent evilness.
>
> This picture has a lot of meaning. It means love, peace, and happiness.

At times, highly imaginative people who are not psychotic may give symbolic responses, but they do so with an awareness of the imaginative or fantasy nature of the response. *Idiosyncratic Symbolism* is scored only when this awareness is absent and is replaced by a certainty about the symbolic representation.

11. Queer Responses. The *Queer Responses* lie on a continuum of a particular kind of bizarre expression: *Peculiar* at 0.25; *Queer* at 0.5; *Absurd* at 0.75; and *Neologism* at 1.0. Whereas *Peculiar Responses*, although deviant, could pass as unremarkable in ordinary conversation, *Queer Responses* carry an obvious ring of pathology that reflects disorganization. We have classified *Queer Responses* into three categories:

- Queer Expressions
- Queer Imagery
- Queer Word Misusage

a. Queer Expressions. These are peculiar responses carried to an extreme. The subject utters the queer expression with an air of certainty, but the listener has little idea of what is meant.

> Examples of *Queer Expressions*: Inward type of a photograph of a flower's reproductive cells.
>
> The outside lookers, the onlookers of the outside.
>
> Their feet are going together unitedly.
>
> Like the outcenter.

b. Queer Imagery. *Queer Imagery* is more than an idiosyncratic use of words; it is also a particularly distant or unrelated response to a task presented by the examiner. There is an ambiguity that exceeds vagueness. The listener has the feeling that the communication is of some private imagery, outside the realm of common discourse:

> Idealized fire. (What do you mean?) It didn't have any blue in it, so it looked like something that had been burning for a long time, or fire that wasn't lit with a match.

c. Queer Word Misusage. Word misusage which is more severe than that described for *Idiosyncratic Word Usage* (4c), and which does not appear to be the result of lack of education, is also scored as (0.50) *Queer*.

> (How are a fly and a tree alike?) A fly has *branches* like a tree.

The use of pretentious, clanging words by subjects of lower educational levels has been discussed earlier. Many such words are scored (0.25) peculiar. However, some of these words may involve more than pretentiousness, when enough fluidity in thinking occurs so that two words with separate meanings combine, and the resultant word has no clear meaning. Such words might be scored as *Neologisms* (1.0), but we believe that many may be more a result of social class and education than of a neologistic process. Unless the examiner inquires about the words, it is difficult to know what process has determined the formation of the word. Because of this difficulty, we have made a compromise rule: score such words as (0.5) *Queer*, because they seem worse than peculiar, but are perhaps not neologistic.

To clarify the distinctions among *Idiosyncratic Word Usage* (4c), *Queer Word Misusage* (10c), and *Neologism* (20), we have provided the following rules of thumb:

> Peculiar (4c): These are real words used incorrectly or awkwardly (or real words with an incorrect prefix or suffix added), although the meaning is very clear.
>
> Queer (11c): These may be real words used incorrectly where the meaning is not clear or they may be words which are not real, but whose meaning is clear. (Either they are very close to a real word or they are a clear combination of real words.)
>
> Neologisms (23): These are always invented words whose meaning or derivation is not at all clear to the listener.
>
> Example of *Queer Word Usage*: Pestals on a flower [combination of petals, pistil].
>
> Examples of *Neologisms*: *Tarangula* [perhaps a combination of tarantula and orangutan, from the context].
>
> Domestic *inquility* [although this may be a simple peculiar distortion of *tranquility*, it may also have idiosyncratic meaning].
>
> A *moliqamous* society.

12. Confusion

The subject does not appear to be sure what s/he is saying, thinking, or perceiving. *Confusion* is also scored when the subject appears to be disoriented about time, place, or person.

> Examples of confusion: This looks like some kind of insect, you know, like under the sea, some type of, not under the sea, some type of crab, yeah, under the sea.
>
> Or a milkweed butterfly, you know how those milkweed worms

> used to look, in the backyard. Oh, you weren't in my backyard.
>
> Somebody could have cut the skin off it and pulled it open and watched it, or looked at it, or something, or stripped it out or something, or pulled it out or something, or had it out straight for a minute.
>
> . . . some people smoking matches and burning cigarettes.

Sometimes a word or expression appears to be the result of a slip. If the slip is limited to a single instance, it is scored as *Tendency to Confusion* (0.25) instead of *Confusion* (0.5). Do not score a slip if the subject corrects her/himself without prompting from the examiner.

> An example of *Tendency to Confusion*: (Why are child labor laws needed?) So that they can protect the incident. (Q?) I-nocent, innocent.

13. Looseness

Looseness represents a dramatic loss of cognitive focus. The subject responds to a question or a percept with ideas that seem either unrelated or arbitrarily or tangentially related. Associations are embellished in an idiosyncratic manner and may flow rapidly with no focus of conversation. The original point is lost; the subject produces free-flowing associations that sometimes may involve serial clanging. If the subject limits her/his looseness to one association as in loose humor, or to a single clang, then it is scored at the 0.25 level (*Loss of Distance*, *Tendency to Looseness*, or *Clang*). Free-flowing, uncontrolled looseness is more pathological, although a short response should not deter one from scoring *Looseness*. We distinguish between looseness in language and conceptual activity and looseness in perceptual organization, which we call *Fluidity* (0.75).

> Examples from the WAIS: (Why should people pay taxes?) Taxation, we have representation . . . taxation without representation is treason . . . . It's frightening what Nixon has done now.
>
> (What does tranquil mean?) Peace, pleasant, something pleasant to listen to or do, or smell. A smile is pleasant.
>
> You're a pleasant dummy to be around.
>
> Examples from the Rorschach: Because it's black, dark, darkness, lovemaking
>
> It could be a bow for your hair, if you've got any. Most people do have a lot of hair, it grows, so they should know how to take care of it.
>
> (What made it look like a crab?) Cuz I'm Cancer the Crab maybe. My sign is cancer. My horoscope. And I'm thinking a lot about cancer too. God forbid if anybody is dying of cancer . . . I wish it was me . . . .
>
> Two fetuses. I love my baby. I really do. I want to go home soon so I can tell her I love her myself. You know, maybe she can come out today. Do you think I passed anything off to her, like a lot of nerves?

A *Tendency to Looseness* (0.25) is scored when the subject's loose response is limited and controlled.

> An example of *Tendency to Looseness*: These look like trousers. Trousers look better on men than women.

14. Fabulized Combinations, Impossible or Bizarre

Combinatory thinking makes use of the primary process mechanism of condensation, in which perceptions and ideas are inappropriately condensed and thus violate realistic considerations. Combinatory thinking can be seen in the language, thought, and perceptual realms.

Unrealistic relationships are inferred among images, blot qualities, objects, ideas, or activities attributed to objects and result in *Incongruous Combinations* at the 0.25 level, *Fabulized Combinations* at the 0.5 level, some *Confabulations* at the 0.75 level, and *Contaminations* at the 1.0 level.

Many subjects produce *Fabulized Combinations* that do not distort reality constraints (for example, on card II, "two women holding bowling balls"). These fabulized combinations are not scored because they result from the combination of two accurately perceived images into a realistically possible whole. *Fabulized Combination* is scored when the subject forces into an unrealistic relationship *two or more* separate and discrete percepts that are contiguous. The form quality of the separate percepts may be good, but the spatial relationship between them is taken as immutably real, and the final combined image is realistically impossible. Impossible combinations distort reality by:

- Involving impossible size discrepancies between objects or blot details;
- Combining objects that do not occur together in nature;
- Mixing natural and supernatural frames of reference.

> Examples from the Rorschach: Two crows with Afros and they're pushing two hearts together
>
> Two potatoes with eyes and a mouth trying to climb up some kind of pipe or pole
>
> Two women . . . a neck with a chest cavity . . . and looks like the two women are resting on the chest.
>
> This looks like two hippos that are climbing up some structure and stepping on a baby.

At times, raters may have difficulty distinguishing among *Incongruous Combination/Composite Response* (7a), *Fabulized Combination* (14), and *Contamination* (21). The following guidelines are suggested:

Incongruous Combination (7a). A composite response is scored when a hybrid creature (i.e., a creature comprising incongruous components) is seen in one area of the inkblot (e.g., a mouse in a coat).

Fabulized Combination (14). A fabulized combination is scored when two or more areas of the blot are combined or related inappropriately (e.g., a man being held by sea horses).

Contamination (21). A contamination is scored when two or more percepts are seen in the same area, or overlapping areas, of the blot and are merged together so that the identity of each is spoiled (e.g., an animal plant).

15. Playful Confabulation

A *Playful Confabulation* is related to the group of *Confabulation* responses (19). The subject's original percept is related to the inkblot, but the response is then overly elaborated beyond the stimulus qualities of the card. It is distinguished from a full *Confabulation* because it is not so malignant. In general, the fabulizing tendency is not so extreme, and more importantly, there is a playfulness or humorous quality to the response. There may be overelaboration of the fanciful activity, where the subject seems to be playing with the percepts and images, usually injecting some humor into the response. Because a *Playful Confabulation* does not indicate the severe slippage that a full *Confabulation* does, it is scored at the 0.5 level. A *Playful Confabulation* may sometimes be difficult to distinguish from a fabulized combination (0.5) because it may also include an inappropriate combination of percepts; however, a *Playful Confabulation* is a more fully elaborated response that conveys more of a story.

> Examples from the Rorschach: (IX) A witch on a broom stick. And it looked like an electric broom stick or something . . . gas powered because she had a cloud of smoke.
>
> (IX) The middle part looks like an evil witch doing a square dance . . . . She had her dress like this and she was doe-see-doeing.

16. Fragmentation

Fragmentation is characterized by an inability to organize and integrate information appropriately. Two kinds of fragmentation are scored:

1. *Fragmentation* is scored when a subject is unable spontaneously to verbalize an integration of several aspects of a percept into a single coherent response. Additionally, there are times when a subject implies a relationship between percepts or blot areas, but articulates this relationship in an unclear, choppy, or disjointed manner.

This response represents a deficit in the capacity to synthesize individual percepts into an integrated response. The fragmentation response should be differentiated from responses that are organized perceptually in a clear, understandable way but are communicated in a disorganized manner. These latter responses, which are marked by disorganized presentation, are more appropriately scored *Confusion*, or, in very severe cases, *Incoherent*.

> Examples: A masquerade party costume. Cha cha. Clap hands. Let's dance. (Inquiry) Partly the color . . . partly the contour of the . . . that's a dance. And . . and you stump . . . stump . . . you stump your feet [score stump peculiar] . . . clap your hands . . . together . . . with your partner.
>
> They . . . act . . . just like just friends. Act like friends, children. (Inquiry) Their feet. That would be two feet. That's when they are close together . . . because they seem so playful.
>
> . . . because of the closeness of their parts. Looks like . . . a centipede. (Inquiry) He's put together right there. Two different parts. This part . . . and this part.

2. *Fragmentation* is also scored when a subject verbalizes a part of a larger percept or discrete response without regard to the larger percept, although it may be inferred that the subject intends to verbalize the larger object. This process is usually discerned either on inquiry or in subsequent verbalizations. These responses, too, are marked by an inability to integrate or synthesize information, and should be distinguished from those responses characterized by constriction or poverty of content. The impoverished responses are generally scored *Vagueness*.

> Examples: I can't see much on this. I can see that it's dark . . . that looks like a handle. That's about all I can figure out of this picture. (Inquiry) Well, it looked like a handle. That's all I can say . . . . Well, I don't know . . . just looked like . . . .
>
> Couldn't make much out of this. I think these two on the sides . . . are picking at . . . they're picking at . . . at this. (Inquiry) Two claws . . . gives you an indication they might be crabs. The two claws . . . . I can't make anything else out of that.
>
> These little things . . . are like whiskers. I don't know what this can be. They certainly don't look like wings. And still that's the body. They must be wings. (Inquiry) I don't like this for wings. But I don't know what else to call them. Well . . . from what

> I've seen of butterflies . . . the body . . . and the big flying wings.

This type of *Fragmentation* response was described by Rorschach as a "Do" or "oligophrenic" response. It is applied when the subject identifies an area that is usually seen as a component of a larger area, either the whole card or a large detail. For example, a subject may see the antennae of a bufferfly on Card V, but not see the butterfly.

The disorder to be inferred when a *Fragmentation* response is given is that some process is interfering with the capacity to integrate the separate parts of the blot into a whole response. The process may reflect difficulties in perceptual organization or in the associative integration of percepts with their ideational referents. At times, the subject will verbalize several parts of the response (e.g., "wings, antennae, legs"), but will be unable, even on inquiry, to combine them into a unitary response.

0.75 Level

Responses scored at the 0.75 level represent clear thought disturbances that clinicians have associated with psychotic disruption. Instability of thinking and perceiving, absurdity, and unrestrained combinatory ideation are typical.

17. Fluidity

In *Fluidity*, the impression is that the subject perceives the world in a highly unstable way; something perceived as one thing at one instant is seen differently at the next instant. The constancy of the object is gone; things do not exist for very long. *Fluidity* is scored when:

- The subject states that one percept appears to be changing into another.
- The subject is unable to assign an explicit identity to a percept, so that the examiner remains uncertain about what has been described.
- The subject is unable to locate or remember a previously described percept because it appears that the percept is so unstable that it cannot be found again.

> Examples: Two people . . . one minute this appears like their eyes and the next this appears like their entire body holding on. This looks like a picture of, hmmm. Oh, at first it looked like a picture of, but I lost that one, so it doesn't look like that anymore.
>
> The head of a rocket or the head of a . . . bear or the head of a bird, but . . . that . . . to me . . . it looked like the top didn't go with the bottom. (Umhmm) Looked like something was becoming something else.

When the subject wavers between two percepts, unable to decide on one or the other—thus evidencing a certain fluidity, though not as clearly as in the preceding examples—(0.5) *Tendency to Fluidity* is scored.

> Examples: The two poodles and the two ladies are the same place, and I couldn't tell if they were poodles or ladies.
>
> When I first looked at it, it looked like a bat flying away, then I looked at it again, it looked like a bat coming toward me.

Do not score responses that are given as differentiated, explicit alternatives:

> It could be a bird or an insect.

It is sometimes difficult to distinguish *Fluidity* from verbal *Looseness*, where the subject's thoughts race and s/he is unable to stick to one focus. To score *Fluidity*, it is important that the subject clarify that one percept appears to be changing into another rather than just following quickly upon a previous, discrete percept. This may be especially difficult in disturbed subjects who speak very rapidly and dart from one percept to another.

18. Absurd Responses

When the examiner or scorer can form no idea about the source of a response, it is scored (0.75) *Absurd Response*. It may have meaning in relation to internal events of the subject but not in relation to the question asked or task set for the subject; therefore, it is almost wholly arbitrary.

> Example from the WAIS: (What is the "Apocrypha"?) Is that the emancipation and the proclamation?

On the Rorschach, an *Absurd Response* is one that has no objective support in the inkblot itself. The scorer should distinguish between *Absurd Responses* and *Perseveration* and *Vague* percepts.

> Examples from the Rorschach: A spider. (Do you see anything else?) No. (What suggested the spider?) The legs. (Was there anything else about it that made it look like a spider?) The nose.
>
> It looks like . . . I don't know. I can't see much in that except maybe bits and pieces of Thailand . . . shapes . . . all kinds of . . . .
>
> This is sticking out there. Remember that's the, uh, cure there. It's our cure, it's called . . . .
>
> I don't know what that is. (Why don't you look at it for a few minutes?) The only thing I can say is I haven't got a spinal tail. I never saw tail. I never saw anything outside of my own anatomy which is different from other anatomies. That's what I think.
>
> And this white space . . . looks like part of a pentagon.

19. Confabulation

The subject's original percept is

related to the inkblot or test material, and if s/he had stopped there, all would be well. Instead, the subject interprets the rest of the blot or material as though it had to belong and fit in with the original percept. *Confabulation* results from excessive distance from the inkblot or task. There are two kinds of *Confabulation*:

- Overgeneralizations from small to larger Rorschach blot areas.
- Extreme associative elaboration and interpretation of a percept or a question.

a. Details in One Area Generalized to a Larger Area. This type of confabulation occurs only on the Rorschach Test. The subject responds to a small area on the blot and overgeneralizes from that area to a larger one in a way that violates the shape of the larger area, although it may be justified for the smaller area.

> Example: The player, the basketball player. (What makes it look like a basketball player?) Here's the ball, then, it's not clear where the basketball player is, but I can see the ball, it must be somewhere in there.

b. Extreme Elaboration. *Extreme Elaboration* results from a fabulizing ideational tendency that goes too far by autistically extending the interpretation of the blot beyond the realm of reality constraints.

> Examples from the Rorschach: The light bulb is—I think it's manmade. And the pink is down here and it gives energy for the light bulb. And the light bulb and the vapors from the pink give, um, cause the green and orange to, uh, to be there. It was—it was—it had its own energy, but the pink coming up into the light bulb gave it more energy.
>
> I now see kind of an evil wizard holding two things up. And I guess they have great powers. He's got this fierce mane of hair . . . hanging down over his shoulders. Some kind of Nordic magic leader of a tribe. Oh, the two things he's holding up could be two animals . . . kind of climbing up the side of his body and his shoulders. He must have them trained somehow. They seem very ominous—it's like he was holding them up and they gave him some kind of power . . . .
>
> It looks like the meeting of two, um . . . dragons who are comparing swords . . . or who are each so self-absorbed in themselves they're ignoring each other and doing a hell-like, hellish . . . demonic . . . dance of the underworld . . . or they're just staring at each other on rollers or standing on clouds, kind of pink clouds.

20. Autistic Logic

To score *Autistic Logic* requires an explicit statement of faulty thinking or reasoning. This may occur when a subject attempts to justify a response with an explanation that includes a "because" phrase, but what follows appears illogical or based on private autistic reasoning rather than on commonly acknowledged logical sequences.

> Examples: WAIS (Picture Completion): I don't see any sails on this ship, so I would say we don't have anyone operating this ship. I see something rather like an appendix. (What made it look like an appendix?) Looked to me totally useless, then I thought of the appendix.
>
> (Gold bullion?) Everything was pointing up to that gold bullion and so I said it was the most precious metal I could think of.

Sometimes a response that appears to conform well to the details of the blot is explained autistically in the inquiry. It is difficult to know whether it is this idiosyncratic reasoning that has produced the response, whether the subject is also aware of the form but did not verbalize this awareness, or whether the language of description is itself so peculiar that the scorer cannot be sure how the subject arrived at the percept. In these cases, we score a *Tendency to Autistic Logic* (0.5).

> Examples: (What suggested a rock?) It looked like a hard, a non-hammer surface (*Peculiar*). It looked like a rock. It didn't look like a human.
>
> (Response given to detail on Card IV, which is commonly called a tree) Giant tree, a dead tree, a dead tree, its leaves falling off. (What made it look like that?) . . . looked like something that was dead. So something that towers over you and is dead to me follows it would be a dead tree.

1.0 Level

At the 1.0 (most disordered) level, reality contact appears to have broken down completely.

21. Contamination

In the *Contamination* response on the Rorschach, two separate and incompatible percepts merge into one. This is the extreme of combinatory activity, in which neither percept retains its original identity. Two overlapping images, usually of the same area of the inkblot, are fused in a single percept.

> Examples: A butterfly holding the world together. (What makes it look like that?) Because I see on both sides patterns of a map.
>
> That's an Indian god . . . jumping into the meadow. A Joe Smith drug meadow. (What is a Joe Smith drug meadow?) He works here at night and every time he hands out medication I see green in front of his eyes like a meadow in Ireland.
>
> This is definitely a man . . . a man butterfly. A butterfly with a man's face . . . could be a dark cloud that's dark because it swallows up all . . . uh . . . the dark particles.

> Man butterfly . . . it could be a cloud-like man butterfly.

22. Incoherence

An *Incoherent* response is not only unrelated to the task but is completely impossible for the examiner or scorer to understand in any context.

> Examples: (What makes it look like a duck?) Their disarrangement. They follow out together, they follow one another. The two toes together, meeting one another. They jacked up in back, like spinal cord being broken.
>
> It's all in front. It's all free hand work. Freehand. Remember freehand? Once in a while we had to freehand. So I did that 'til I ended up here for down the rest. You get crabby. And it's more than aging gracefully. For strong people.
>
> (What made it look like muscles?) It looked like the hairs. (What?) The hairs (What?) The hairs. The shoulders. (Did you say it looked like the hairs?) Yeah. (What made it look like hair?) No, not hair. It looked like, kind of . . . that's all I could think of.

23. Neologisms

Neologisms are invented words, perhaps as a result of verbal condensation, perhaps out of a private language. Sometimes a *Neologism* resembles a verbal contamination in that two words are combined and a third one emerges from the combination but still carries the meaning of both words. The subject seems unaware of any unusualness in the word uttered. It appears to have meaning to her/him. But the listener may be confused, either because the invented word carries two conflicting meanings, or because it is incomprehensible. See *Queer* (11) for guidelines to distinguish *Peculiar* (4), *Queer* (11), and *Neologism* (23).

> Examples: That's tavro or neoglyphics.
>
> It looks like a firmabone. (A firmabone?) That's right, a firmabone.

TD_W = the sum of the TDI scores on the WAIS

TD_R = the sum of the TDI scores on the Rorschach, divided by the number of Rorschach responses, multiplied by 100

$$TD_W = .25(A) + .50(B) + .75(C) + 1.00(D)$$

$$TD_R = \frac{.25(a) + .50(B) + .75(C) + 1.00(D)}{R} \times 100$$

where:

A = number of responses scored at level .25

B = number of responses scored at level .50

C = number of responses scored at level .75

D = number of responses scored at level 1.00

R = total number of Rorschach responses

References

Holt, R.R., and Havel, J. A method for assessing primary and secondary process in the Rorschach. In: Rickers-Ovsiankina, M.A., ed. *Rorschach Psychology*. New York: John Wiley & Sons, 1960. pp. 263-315.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking*. San Francisco: Jossey-Bass, 1979.

Rapaport, D.; Gill, M.M.; and Schafer, R. *Diagnostic Psychological Testing*. Edited by R. Holt. New York: International Universities Press, 1968.

Weiner, I.B. *Psychodiagnosis in Schizophrenia*. New York: John Wiley & Sons, 1966.

Evaluating Bizarre-Idiosyncratic Thinking: A Comprehensive Index of Positive Thought Disorder

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Abstract

A summary of our method of assessing positive thought disorder, or bizarre-idiosyncratic thinking, from two short verbal tests is presented. This measure provides for standardized thought disorder assessments of: (1) the overall presence and severity of thought disorder, and (2) the type of disordered thinking shown. A definition and examples of bizarre-idiosyncratic thinking are provided, along with information on the reliability and validity of the scoring system. A method of establishing subject groups based on the severity of positive thought disorder also is presented—ranging from no thought disorder, to abnormal thinking, to severe formal thought disorder. This measure has been used to assess longitudinal changes in thought disorder symptoms over time and to evaluate relationships between thought disorder, other aspects of psychopathology (such as delusions), and adjustment in other areas of functioning across diagnostic groups.

Investigators have attempted to assess disordered thinking from a number of different viewpoints (Buss and Lang 1965; Maher, McKean, and McLaughlin 1966; Gottschalk and Gleser 1969; Payne 1970; Maher 1972; Chapman and Chapman 1973; Andreasen 1979*a*, 1979*b*; Chapman 1979; Johnston and Holzman 1979). Much of our own approach to studying positive thought disorder has revolved around the construct of bizarre-idiosyncratic speech and thinking (Harrow and Quinlan 1985; Marengo and Harrow 1985). A comprehensive assessment of bizarre-idiosyncratic thinking involves an evaluation of most of the major types of phenomena usually included under the term "formal positive thought disorder" (Fish 1962; Andreasen and Olsen 1982).

Since the concept of positive thought disorder deals with a potentially important symptom of disturbance, we have focused on this feature as one major aspect of a long-term, multidisciplinary research project studying the course of psychosis and adjustment in schizophrenia (Chicago Followup Study, Michael Reese Hospital and Medical Center, Chicago, Illinois). In this context, our studies of positive thought disorder have been primarily directed toward answering the following research questions: (1) Is thought disorder characteristic only of schizophrenia, or do other psychotic and nonpsychotic populations also demonstrate this behavior? (2) What changes in positive thought disorder occur over the course of the schizophrenic disorder? (3) Is the degree of disordered speech and thinking during posthospital or postacute stages of schizophrenia an index of the severity of disturbance? (4) What is the longitudinal relationship between disturbed language and thought and other aspects of psychiatric adjustment (e.g., work and social functioning, delusions and hallucinations)?

This manual represents a portion of a larger and more detailed manual by the same authors: The assessment of bizarre-idiosyncratic thinking: A manual for scoring responses to verbal tests. In: Harrow, M., and Quinlan, D., eds. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, Copyright ©1985. This shorter and slightly modified version of the manual has been adapted for the current purpose with the kind permission of Gardner Press.

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The thought disorder measure presented here focuses on assessing the frequency and severity of positive thought disorder. While more qualitative typologies exist, it is assumed that measuring the degree of severity of thought disorder, which relates to hypotheses regarding a continuum of disturbance, will provide fruitful results.

The following system outlines the scoring of bizarre-idiosyncratic verbalizations from two short verbal tests. The complete scoring manual and further examples can be found in Harrow and Quinlan (1985). This system represents an extension of prior research efforts in the area. Some of the material from the current procedure incorporates criteria developed by D. Adler that have been used successfully in previous research (Harrow, Tucker, and Adler 1972; Adler and Harrow 1973, 1974; Harrow and Quinlan 1977, 1985). However, more specific criteria are now included for evaluating bizarre-idiosyncratic responses, and characteristic examples of such responses are offered as an aid to assessment. Analyses of disturbed speech and thinking, drawn from a variety of patient populations in various settings and contexts, have provided a framework for the current work.

Definition of Bizarre-Idiosyncratic Thinking

We have defined bizarre-idiosyncratic speech and behavior as that which is: (1) *unique to the particular subject;* (2) *deviant with respect to conventional social norms;* and (3) *frequently hard to understand, or to empathize with, in the context from which the response arose.* While these three features are central to the concept, other less frequent characteristics are verbalizations that: (4) *may appear confused, contradictory, or illogical;* (5) *may involve sudden or unexpected contrasts;* and (6) *are usually inappropriate or unresourceful in relation to the task at hand.*

The first two of the above features are directly linked to social norms, though they are viewed from different vantage points: that of the individual and that of society. The last three characteristics listed in the definition may also appear as features of bizarre-idiosyncratic thinking, but they are features that are present some of the time, instead of being invariant. It is surprisingly easy to spot bizarre-idiosyncratic statements, since the manner of presentation or the ideas themselves impress the rater (including a naive rater) as unusual, strange, odd, or inappropriate.

The above definition provides us with a broad conceptualization of what are usually considered idiosyncratic statements. It is important to emphasize that bizarre or idiosyncratic qualities may be found in abstract and concrete as well as correct and incorrect responses, but they are analyzed and scored as a dimension separate from these other aspects of response behavior.

To illustrate, suppose the examiner presents the proverb:

> Strike while the iron is hot.

And the subject responds with:

> It could mean (pause) Hercules! (Could you say more?) I saw the movie Hercules. (Yes . . .) and it means don't iron over your hands and don't strike anybody before you cast the first stone.

Although a response may be scored for positive thought disorder without meeting all of the above criteria, this particular response meets many of the criteria for bizarre-idiosyncratic thinking noted above. It represents an explicit example of disordered thinking. The evaluator first wonders, "Hercules? What is the relation of the response to the original proverb?" The response deviates in an idiosyncratic way from consensual norms for answering this question. Upon reflection, one sees some of the possible reasons for the subject's interpretation in the suggestion of striking and violent action, with this leading to a response involving Hercules. In both this and other components of the response, it is difficult for the scorer to empathize with or understand the context from which the answer arose. In addition, the idea of ironing one's hands is unexpected with respect to what has preceded it, and unusual on its own account. The entire statement appears confused, and the line of thinking that has led to the response has resulted in an idiosyncratic answer to the question.

Procedure

The procedure includes: (1) assessing subjects with two short verbal tests; and (2) assigning an overall score for the severity of thought disorder to each of the subject's responses. This procedure is outlined below. In addition, scoring criteria have been developed to characterize the subtypes of disordered thinking found in each response. Five categories and 11 subcategories of bizarre-idiosyncratic thinking have been defined and will be summarized in this article. More complete scoring criteria for these categories can be found in Harrow and Quinlan (1985). A protocol representing a range of overall and category scores for bizarre-idiosyncratic thinking is provided in Appendix I. This scored protocol may be used as a basis for practice and scoring comparisons.

Instruments and Administration of Tests. During the course of pilot work, it became clear that test materials vary in their potential for eliciting idiosyncratic verbal behavior. Even for those subjects whose speech would be labeled as odd by many, some types of verbal materials and tests do not readily elicit idiosyncratic responses. We have found that more open-ended procedures better lend themselves to eliciting this response behavior in those subjects with a potential for thought-disordered speech. Other tests prove cumbersome or unreliable. However, two verbal tests, the Gorham Proverbs Test (Gorham 1956) and the Comprehension Subtest of the Wechsler Adult Intelligence Scale (WAIS) (Wechsler 1955, 1981), are relatively short and easy to administer and were found to be satisfactory tools for evoking idiosyncratic verbal responses in those individuals with a potential for disordered thought.

The Proverbs Test. The Gorham Proverbs Test (clinical, free-answer set) consists of three parallel forms, each containing 12 proverbs. In our research, the multiple forms have been useful in the collection of longitudinal data over time from the same subject.

For purposes of standardizing subject assessment, directions for the test are read as follows:

> I am going to read you some sayings. For example, the saying, "Large oaks from little acorns grow" could mean that great things may have small beginnings. Now, please tell me what each saying means instead of just telling me more about it. Try to answer every one.

WAIS Comprehension Subtest. The Comprehension Subtest of the WAIS (Wechsler 1955) or WAIS–R (Wechsler 1981) consists of questions relating to social comprehension and judgment (e.g., Why does land in the city cost more than land in the country?). Twelve of these questions provide the materials for our evaluation of bizarre-idiosyncratic thinking (questions 3–14).

The Comprehension subtest is introduced to the subject as instructed in the WAIS manual. Encouragement toward responding is sometimes needed in this test, as well as in the proverbs test, and is appropriate to use in the manner standardized by the Wechsler manual.

Recording the data. It is essential that the verbalizations of the subject, as well as those of the examiner, be written down verbatim. The protocol also should include notations with reference to the behavior or affect stimulated by the test material or expressed during the course of testing. If it is impossible to record the verbalizations of an acutely disturbed patient, a transcribed tape recording may result in the most accurate test protocol. The examiner should inquire about all unclear or odd words or responses in a curious but nonintrusive manner, and these inquiries should be noted in parentheses. Since the object of these tasks is to obtain enough information to assess various aspects of speech and thinking that contribute to idiosyncratic responses, examiners must exercise judgment based on their own understanding of what has been said as the criteria for initiating inquiry.

Other verbal tests. Other comparable proverbs tests have been used in previous research (Benjamin 1944). Recently, we also have used a verbal test (Lanin-Berndt Communications Interview) with a series of standard questions designed to tap several important dimensions of verbal behavior (Berndt 1981; Lanin et al. 1981). Our efforts to derive a scoring system for tests of this general type led to the development of the current manual.

The Rorschach Test and free-verbalization situations have also been useful in our assessments of bizarre-idiosyncratic thought (Quinlan et al. 1972; Quinlan and Harrow 1974; Reilly et al. 1975). Both of these techniques show many valuable features. However, the Rorschach Test takes longer to administer and to transcribe. The free-verbalization technique, while sensitive to eliciting bizarre-idiosyncratic behavior, also presented some problems in its standardization and transcription.

Scoring

Overall score for bizarre-idiosyncratic responses. An overall score for bizarre-idiosyncratic thinking is assigned to each response. It assesses each response for its fit with the current verbal context and what is generally considered appropriate and understandable in our society. The extent to which a response as a whole is bizarre, as well as the extent to which it meets the criteria of any or all of the specific types of bizarre-idiosyncratic thinking, varies greatly. In the present scoring system, we evaluate degrees of thought disorder by assigning scores of 0, .5, 1, or 3, ranging from absent to severe thought disorder. Several examples of responses at each level of bizarre-idiosyncratic speech are provided here. Other examples with the appropriate overall score for bizarre-idiosyncratic thinking are presented in Harrow and Quinlan (1985).

Overall score values

0 = Idiosyncratic verbalizations are absent.

.5 = *Mild cognitive slips.* Verbalizations that contain some mildly idiosyncratic material. The response is slightly odd, but in a social situation the verbalization is not unusual enough to draw considerable attention. Two examples of responses scored .5 are presented below:

> Q: What should you do if while in the movies you were the first person to see smoke and fire?
>
> A: Go out and tell the messenger. (Q) Did I say that? I meant manager.
>
> Q: Why should people pay taxes?
> A: Taxes are necessary. Obsessive *takes* help the government. I could give a whole thesis on it.

1 = *A definite idiosyncratic or bizarre response.* This type of response is noticeably unusual or strange, but usually still understandable. Most responses with clear bizarre or idiosyncratic aspects receive this rating. These responses would definitely be noticeable for their strangeness in a social situation. Two examples are presented below:

> Q: Why should we keep away from bad company?
>
> A: They produce an aura of ill-effect. (Q) They're not—you shouldn't be "subseeded" or deceited by people who are bad. They're just no good.
>
> Q: Rome was not built in a day.
>
> A: It's love, I think of it as love. I have to work towards love and love has to work towards me. And this has to gradually come.

3 = *A very severe bizarre response.* Such responses reflect a very serious deviation from consensual statements, may contain considerable confusion, and are very socially atypical. It is often hard to understand why such a response was given to the particular question involved. This type of response is very rarely found in the normal population. Two examples are presented below:

> Q: When the cat's away, the mice will play.
>
> A: Yeah. On the earth, up at the top, in the middle. XYZ. The end, the beginning of the end of the beginning.
>
> Q: One swallow doesn't make a summer.
>
> A: Boy, that's greedy as hell, man, that's real greedy. That's like pulling my actual backwards.

Summed overall scores for the proverbs and comprehension tests range from 0 to 36. In assigning the overall score for potential bizarre-idiosyncratic thinking to each response, the rater is essentially assessing how strange or deviant the response is in relation to more conventional answers. Even responses that one can understand or empathize with will at times be scored, since they may show odd features or deviate from social convention in an unusual or unexpected manner. However, responses that deviate from the conventional answer and that are also difficult to understand are assigned more severe thought disorder ratings.

As outlined above, an overall score of 0 is assigned when the response is not bizarre or idiosyncratic in any way. An overall score of .5 is assigned when the response is slightly off, or contains cognitive slips that are not grossly deviant. In a social situation, such responses would not really startle people or raise major questions. This score is meant to capture slight deviations, some of which are expected to be found in normal records as well. An overall score of 1 is assigned to a response that is clearly idiosyncratic or bizarre. An overall score of 3 is assigned only to extremely unusual or very bizarre statements.

Note that in scoring bizarre-idiosyncratic responses, incorrect answers are not penalized, since lack of knowledge does not represent strangeness or bizarreness. However, incorrect answers in which it is difficult to understand why the particular incorrect answer was given, and incorrect answers that have no relationship at all to the question, are usually considered bizarre or idiosyncratic and scored as such.

The *overall score* has emerged in our research as the most accurate estimate of the severity of disordered thinking. It involves a judgment of positive thought disorder in the response as a whole and is based on the scorer's understanding of the definition of bizarre-idiosyncratic thinking as well as on the coherence and appropriateness of the response. The overall score is an assessment of the *degree* of idiosyncracy reflected in a response.

Establishing Subject Groups Based on the Severity of Bizarre-Idiosyncratic Thinking

Continuum model. Responses reflecting thought disorder can be placed on a continuum extending from very severe bizarre-idiosyncratic thinking to normal, socially consensual thinking, with considerably heavier weightings for very severe bizarre responses, as opposed to mild cognitive slips. It also can be useful to have some system available to facilitate placing subjects into rough categories according to whether they show severe thought pathology, moderate levels of thought pathology, or no thought pathology. Accordingly, we assign subjects' total summed overall scores from the Proverbs and Comprehension tests to categories or levels reflecting various degrees of bizarre-idiosyncratic thinking.

In this system, subjects' total overall scores have been categorized into a continuum of positive thought disorder. The five specific categories included in the current system are: (1) no thought disorder; (2) minimal to mild bizarre-idiosyncratic thinking; (3) signs of abnormal thinking or definite thought disorder; (4) severe thought disorder; and (5) very severe thought disorder. The conversion levels presented in table 1 are used.

Levels 1 and 2 of the Thought Disorder Scale are representative of little or no pathological thinking and lie well within the normal range. Level 3 (moderate level) reflects definite evidence of thought pathology or abnormal thinking. The number of patients at levels 4 and 5 provides an estimate of *severely* thought-disordered patients. The combined number of patients at levels 3, 4, and 5 gives the percentage with definite signs of positive thought disorder or abnormal thinking.

Composite index of bizarre-idiosyncratic thinking. A composite index of bizarre-idiosyncratic thinking also can be computed from both verbal tests, the Proverbs and the Comprehension tests, using the five levels of bizarre-idiosyncratic thinking described above. In this composite index, each subject is assigned to the *highest (most severe) level* into which he or she fits on either of the two tests. This composite index classifies subjects according to whether they show any signs of bizarre-idiosyncratic thinking, given opportunities on two separate tests to show such behavior.

In our research we also obtain measures of thought disorder from the Object Sorting Test (Goldstein and Scheerer 1941; Goldstein 1944) and have categorized the scores from the Object Sorting Test into the same five levels of bizarre-idiosyncratic thinking (Harrow et al. 1985). We have combined the category scores or levels from the Object Sorting Test with the parallel category scores or levels from the Proverbs and Comprehension tests to obtain our major composite index of bizarre-idiosyncratic thinking (table 1).

This composite index is based on scores from the three separate measures of thought disorder. For this composite index, each patient is assigned to one of the five categories or levels ranging from no abnormal thinking (level 1) to very severe thought disorder (level 5) *according to the most severe level attained on any of the three tests.* This classification system categorizes patients according to whether they show any positive thought disorder, given three separate opportunities to do so. The composite score encompasses a broad sample of behavior and is more comprehensive than indexes that include only one or two tests.

Relation of this System of Evaluating Bizarre-Idiosyncratic Thinking to Other Systems of Thought Disorder Assessment

Rapaport, Gill, and Schafer (1968) system. Note that in several ways the conceptual framework we employ to assess bizarre-idiosyncratic thinking is different from that used by Rapaport in his research studying disordered thinking, although the two systems are comparable in several respects. Our conceptual framework is closely tied to implicit conceptual norms that people have acquired over time about what is appropriate and what is deviant in a particular response situation. The framework used by Rapaport is based, in part, on assessing pathological verbalizations by whether they involve too much distance or a loss of distance from the original stimuli, with such judgments being based on implicit social norms (Rapaport, Gill, and Schafer 1968). However, while there is some difference in the conceptual basis of the systems, there is much similarity in the actual practice of scoring disordered verbalizations. The system we use involves assessing consensually deviant responses and behavior along dimensions of thought disorder that Rapaport might have labeled as "too much distance" or a "loss of distance" from the original stimuli.

Johnston-Holzman Thought Disorder Index (1979). Our construct of bizarre-idiosyncratic thinking and Johnston and Holzman's Thought Disorder Index also show similarity. In both systems, bizarre, strange, and deviant responses are assigned scores, and in both systems more severely deviant responses are given heavier or more pathological weighting. Johnston and Holzman's Thought Disorder Index is used with the WAIS Comprehension Subtest and with other WAIS subtests, as well as with the Rorschach Test.

Our Proverbs test measures of bizarre-idiosyncratic thinking showed significant correlations ($r = .61$) with thought disorder as measured on Johnston and Holzman's (1979) Thought Disorder Index in a sample of young schizophrenic subjects studied during postacute phases of disturbance. We have found this system valuable. We tend, however, to favor the current verbal tests and system of assessment since they are less time consuming.

Andreasen's Scale for the Assessment of Thought, Language, and Communication (TLC). Andreasen's (1979*a*, 1979*b*) TLC Scale defines different language behaviors as subtypes of thought disorder. Ratings for the severity of thought disorder are included and are based on a standardized means of

Table 1. Derivation of thought disorder groups and the Thought Disorder Scale from 3 psychological tests

Thought disorder group	Thought Disorder Scale	Thought disorder continuum	Proverb or Comprehension Test total score	Object Sorting Test total score
No abnormal thinking	1	Absent	0–.5	0
	2	Mild	1–2.5	1
Abnormal thinking	3	Definite	3–6.5	2
Severe thought disorder	4	Severe	7–11.5	3–6
	5	Very severe	12–36	7–21

assessment. In general, the types of strange and bizarre thinking tapped in our measure of positive thought disorder are also assessed in Andreasen's composite index of positive thought disorder derived from the TLC. For instance, the various types of thought disorder assessed by Andreasen (e.g., tangentiality, incoherence, and loss of goal) are also scored for bizarre-idiosyncratic thinking when they appear in the verbal tests we administer to assess positive thought disorder.

RDC and DSM-III criteria of thought disorder. The key components of bizarre-idiosyncratic thinking in the current system also can be helpful in assessing whether a patient has the type of formal thought disorder listed in major diagnostic systems such as the Research Diagnostic Criteria (RDC) and *DSM-III* (Spitzer and Endicott 1968; American Psychiatric Association 1980). Four of the five types of pathological speech and thinking outlined in the RDC as constituting formal thought disorder (impaired understandability of speech, loosening of association or derailment, illogical thinking, and neologisms) are viewed in the present system as components of bizarre-idiosyncratic thinking.

Similarly, although *DSM-III* is a little less optimistic about the concept of formal thought disorder, three of the specific types of thought disorder which, in effect, are substituted in *DSM-III* for formal thought disorder (incoherence, marked loosening of associations, and markedly illogical thinking) are included in the present system. Note that these types of thought pathology are included as symptoms that can be used in *DSM-III* as partial criteria for a diagnosis of schizophrenia.

Reliability and Validity. Evaluations of both reliability and validity of the current system of assessment have been based on normal subjects and on outpatient and inpatient psychiatric groups. These assessments have taken place in a variety of settings and have included chronic and acute, psychotic and nonpsychotic, and medicated and unmedicated patient samples.

Reliability. Interrater reliability has been obtained for: (1) the overall scores of bizarre-idiosyncratic thinking using Proverbs and Comprehension tests; and (2) for the subcategories of bizarre-idiosyncratic thinking presented in the following section. Interrater reliability estimates always have been high for the overall scores and moderate to high for scoring the specific categories. Four separate assessments of 20 patient records produced interrater reliabilities of $r = .93$, $r = .88$, $r = .67$, and $r = .91$ for total overall scores on the Proverbs and Comprehension tests. Interrater reliabilities for each category of idiosyncratic thinking were as follows:

Category	Range of r
I. Linguistic form and structure	.82–.99
II. Content of statement: Ideas expressed	.84–.99
III. Intermingling	.75–.85
IV. Relationship between response and question	.73–.99
V. Behavior	.47–.98

In addition to interrater reliability, internal consistencies for the Proverbs test using Cronbach's α produced a reliability of .85 for all possible combinations of item-by-item scores. Subjects were also tested on two parallel forms of the Proverbs test administered successively in the same test session. Gorham's (1956) Proverbs Set 1 and Proverbs Set 3 were used. The correlation of subjects' overall scores for bizarre-idiosyncratic thinking between these two sets of proverbs was $r = .79$ (Harrow and Miller 1980).

The total overall scores of Proverbs and Comprehension tests for the same subjects have also been compared to determine whether a subject with more pathological scores on one of these tests is likely to have more pathological scores on the other test. In a sample of 18 unmedicated State hospital inpatients assessed with both tests during the first week of hospitalization, the reliability coefficient was $r = .74$ between Proverbs and Comprehension total scores. A second sample of 104 medicated patients (63 schizophrenic and 41 nonschizophrenic subjects) from a private and State hospital setting provided a reliability coefficient of $r = .53$ between Proverbs and Comprehension total scores. Significant correlations between the tests emerged for: (1) the total number of thought-disordered responses, $r = .57$; (2) the number of moderate and severe bizarre responses, $r = .55$; and (3) the frequency of severely bizarre responses, $r = .47$. This indicates that the bizarre-idiosyncratic thinking found is not peculiar to a specific test.

However, a significant difference emerged between the total scores derived from the two tests, $t = 2.69$, $df = 102$, $p < .01$. The Proverbs test elicited more severely bizarre responses than the Comprehension subtest when administered at the acute phase of patients' disorders. The Proverbs test appears to be more sensitive to eliciting thought-disordered responses in acute patients who have a tendency toward bizarre thinking (Marengo 1983). This appears to be a function of differences in test stimuli and demand characteristics. The Proverbs test presents less familiar material to the patient and demands more abstraction and more complex thinking. The subject cannot rely as much on previous experience and socially stereotyped responses when responding to many of the proverbs.

Validity. We have conducted a series of investigations which bear on the construct validity of this system for assessing positive thought disorder. A number of these studies are based on our longitudinal research assessing (1) the relationship between bizarre-idiosyncratic speech and other aspects of psychopathology, and (2) the level of bizarre-idiosyncratic thinking across different stages of patients' disorders. Early results on various patient and normal samples have supported the validity of the construct of bizarre-idiosyncratic thinking.

Our research on bizarre-idiosyncratic thinking with the Proverbs and Comprehension tests has indicated the following:

- Bizarre-idiosyncratic thinking was positively correlated with linguistic errors on a structured communication task (Lanin-Kettering 1983). Thought disorder as measured on the Proverbs Test showed a significant association with an overall index of contextual coordination, $r = .48$, $p < .001$, and was also correlated with several component measures, including the ability to maintain topic, $r = .29$, $p < .03$, and to produce sentence-to-sentence coherence, $r = .43$, $p < .003$. Assessment included a separate sample of psychiatric and normal subjects.
- Schizophrenic and nonschizophrenic patients ($n = 85$) with a disturbance of associative processes on a word association test also manifested significantly more severe idiosyncratic thinking on the Comprehension Subtest of the WAIS (Silverstein, Harrow, and Marengo 1980).
- Correlations of the Proverbs and Comprehension test total scores with the Object Sorting Test measure of bizarre-idiosyncratic thinking were $r = .60$ and $r = .50$, respectively, in 50 psychiatric subjects assessed at the acute inpatient phase.
- We have assessed the relationships between earlier versions of our indexes of bizarre-idiosyncratic thinking and a measure of communication deviance employed by some theorists on schizophrenic families (Wild et al. 1965; Wild 1972), using the Object Sorting Test. In a study of 40 schizophrenic and nonschizophrenic patients, these relationships were significant. Wild and Lidz's measure of transactional thinking was significantly related to our earlier measures of bizarre-idiosyncratic thinking assessing WAIS Comprehension Subtest responses ($r = .45$), and to our similar measure assessing Object Sorting Test responses ($r = .67$) (Quinlan et al. 1978). These results suggest a significant relationship between measures of bizarre-idiosyncratic thinking or positive thought disorder and measures of communicative deviance from family studies.

In a series of studies using the composite index of positive thought disorder (including the three thought disorder measures), the system described above has proved useful.

- At the acute phase of disturbance, early schizophrenic patients showed significantly more severe bizarre-idiosyncratic thinking than other psychotic and nonpsychotic patient groups (with the exception of manic subjects), indicating the index's sensitivity to diagnostic factors (Marengo and Harrow 1985).
- Differences exist in the severity of bizarre-idiosyncratic thinking over different stages of psychiatric disturbance. A decline in the severity

of bizarre-idiosyncratic thinking is evident between acute-phase assessment and the stage of partial recovery (7 weeks after acute-phase assessment). This reduction is significantly associated with improvement in other aspects of patients' clinical conditions (Harrow et al. 1982; Harrow, Marengo, and Lanin-Kettering 1983).

- In a longitudinal study we found significant schizophrenic-nonschizophrenic differences in disordered thinking at the acute phase, and considerably smaller differences at 1.5-year followup, a postacute period (Harrow, Marengo, and McDonald 1986).
- We have found significant associations between the Thought Disorder Scale (based on the composite index) and other major psychopathology, such as delusions (Harrow, Silverstein, and Marengo 1983; Marengo and Harrow 1985; Harrow and Marengo, this issue; Harrow, Marengo, and McDonald 1986). In addition, we have found significant relationships between the Thought Disorder Index and the degree of dysfunction in current and subsequent overall adjustment shown by schizophrenic and other psychiatric groups (Harrow, Silverstein, and Marengo 1983; Marengo 1983; Harrow and Marengo, this issue; Harrow, Marengo, and McDonald 1986).

While we view the points of the thought disorder scale as lying on a continuum, the specific cutoff points that establish the thought disorder groups have proved valuable. When patients are categorized according to whether they have severe thought disorder, as opposed to signs of abnormal thinking, as opposed to little or no thought disorder on our composite index, significant differences among groups in various types of major psychopathology emerge (Harrow and Marengo, this issue). This has occurred in comparisons among schizophrenic patients with no or only mild thought disorder (Thought Disorder Scale levels 1 and 2), others showing signs of abnormal thinking (Thought Disorder Scale level 3), and those exhibiting severe or very severe thought disorder (Thought Disorder Scale levels 4 and 5). Thought disorder group differences have been found in concurrent assessments of work functioning, overall psychotic activity, and rehospitalization rates during posthospital phases of schizophrenia and major depressive disorders (Marengo 1983). These data indicate some degree of validity for the cutoff points established and listed above.

In other phases of our research, the Thought Disorder Scale and the thought disorder groups (using the composite index) were predictive of the future functioning of patients when thought disorder was assessed at an early, posthospital phase of disorder. The presence of severe thought disorder (Thought Disorder Scale level 4 or 5) is a good prognostic indicator of future sustained functioning impairments in both schizophrenic and nonschizophrenic disorders (Harrow and Marengo, this issue). By contrast, non-thought-disordered patients and those showing abnormal thinking (but not severe thought disorder) evidence a more benign or remitting subsequent course of psychosis than do severely thought-disordered patients over the longitudinal course of disturbance (Marengo 1983).

In these early studies, the results from the Thought Disorder Index have generally fit in with common clinical observations and theories about bizarre-idiosyncratic thinking and positive thought disorder.

Note that in our assessments of nonpatient control samples, some normal subjects have manifested signs of abnormal thinking (Marengo and Harrow 1985). Normal subjects have also demonstrated signs of severe positive thought disorder. From 10 to 15 percent of normal subjects showed level 4 or 5 on the Comprehension or Proverbs test when one of these tests was used alone, and 10 percent showed severely disordered thinking on the composite index (Marengo and Harrow 1985). The finding of some control subjects with severe levels of thought disorder should not be surprising, in view of various findings over the years of certain levels of psychopathology, and even severe psychopathology, in some "normals."

We should also point out that thought-disordered individuals show a great deal of normal speech. This attests to the phasic nature of thought disorder, in some subjects, even during periods of acute upset. Our measures assess the behavioral *potential* for bizarre-idiosyncratic thinking in spontaneous, open-ended speech situations.

Specific Categories and Subcategories of Bizarre-Idiosyncratic Thinking

In addition to the *overall* scores for thought disorder severity, we have outlined a system for evaluations of each response focusing on criteria constructed to delineate the anomalies of positive thought disorder. Five categories and 11 subcategories of bizarre-idiosyncratic thinking provide the criteria for evaluating components of bizarre-idiosyncratic verbalizations in greater detail. These major categories and subcategories represent various types of abnormal ideas, behavior, and language. They also provide one way of categorizing some of the different types of disordered thinking that can be seen in response to specific tasks, as well as in people's day-to-day behavior.

The presence of these components of disordered thinking can be independently studied in light of different theoretical predictions, developments at different points in the unfolding of, or recovery from, a particular disorder, and in understanding differences among clinical populations. With bizarre-idiosyncratic thinking as a more general construct, these categorical evaluations provide the opportunity to study particular kinds of thought disorder.

We have found these subtypes of bizarre-idiosyncratic verbalizations and behavior useful, and we do score them in our own research. *We should emphasize, however, that scoring or attending to these specific criteria is not absolutely necessary for attaining the overall score for bizarre-idiosyncratic thinking. One can use the overall system of assessment of thought disorder severity on the basis of the criteria outlined in the previous pages without the detailed and specific subcategories noted in the following pages.* If the following subcategories are used, the initial overall rating of bizarre-idiosyncratic thinking should be made first, before scoring these individual subcategories.

We have used five basic categories to study both the subject's linguistic form (i.e., the manner in which ideas are communicated) and the content of the responses (i.e., the ideas themselves). Detailed examples of specific responses and how they should be scored within the 5 categories and 11 subcategories of bizarre-idiosyncratic responses are provided in Harrow and Quinlan (1985). These categories are not exclusive, and many responses are scored for several types of component problems, particularly since problems in form and problems in content are often difficult to tease apart.

Five Categories of Bizarre-Idiosyncratic Thinking

I. Linguistic form and structure. Here, the structure of language within the response is under scrutiny. A problem in this area implies that it is difficult to understand the subject's statement owing to *distortions in word use, grammatical form, or the linkage of words and phrases. A response also may be communicated poorly.* Questions are raised about peculiarities in the individual's verbal style, the linguistic structure of the response, or gaps in communication that may interfere with the clear communication of meaning.

II. Content of the statement: The ideas expressed. Under primary consideration are the ideas presented within the response. This category pertains to peculiarities within a response such as *idiosyncratic reasoning, asocial attitudes, and disorganized or confused ideas.* The ideas or attitudes the subject presents are evaluated from the perspective of conventional attitudes, logical thought, or cogent explanations.

III. What is intermixed into the response. In this category, the focus is on whether the response moves away from a consensual answer owing to a shift to loosely associated ideas or the subject's personal preoccupations. The twofold emphasis of this category is on: (1) the mixing or blending into the response of personal material from the subject's past or current experience, and (2) the extensive elaboration of a theme or idea, which does not fit neatly into the structure of the response, making it appear somewhat unusual. The question raised is: Are there any *personal associations or tangential ideas* that the subject is expressing as part of his response *that are idiosyncratic or bizarre*?

IV. Relationship between question and response. Here, the focus of evaluation is on how the *ideas the subject presents relate to the question asked.* Is the subject able to address the task of interpretation, or answer a question? Is the subject inappropriately, or totally, focused on private, autistic associations or thoughts?

V. Behavior. Behavior is assessed using conventional norms for conduct in a testing situation, including physical, affective, and verbal behavior. Is the subject's behavior deviant either in its extreme expression or in its incongruity and impropriety within the testing context?

Suggested Step-by-Step Procedure for Evaluating the Individual Categories of Bizarre-Idiosyncratic Thinking. The assignment of scores within these categories is determined using the criteria listed below. The extent to which a response meets each (or none) of the relevant category criteria is evaluated and the response is assigned a score of 0, .5, 1, or 3 for each category.

In assigning category and subcategory scores, it helps to:

- Consider how the subject expresses ideas. Is the response poorly structured, composed of vague elements, or obscured by idiosyncratic terminology? What is the subject's choice of words? Is there any unusual word usage or use of artificial, pedantic, or stilted language? Does the subject communicate ideas in a reasonable fashion, or are there unexplained gaps or missing referents that make it very difficult to understand? If yes, then evaluate the response in terms of subcategory indicators and assign a score reflecting the degree of thought disorder in Category I.

- Consider next the ideas presented. Does the subject's statement make any sense at all? If the response makes no sense, score a 3 in Category II. Does the reasoning deviate from rules of logic or social convention? Does the statement involve an odd meaning or outlook? If yes, then evaluate the extent and assign an appropriate score in Category II and in its appropriate subcategories.
- Then evaluate intermingling in the response. At times a subject's answer shows intermingling of personal material from the very beginning of a response. At other times, after a smooth beginning (in which the initial thrust of the response is partially or completely correct), does the subject's response then wander onto tangential or irrelevant topics? Does the response reflect intermingled personal material? If yes, then evaluate the extent to which this additional material causes the response to appear odd and assign scores to the appropriate subcategories and to Category III.
- Now consider the relation of the responses to the question or proverb. Does the content of the response (at least initially) refer to the question asked (irrespective of whether it is correct or incorrect, concrete or abstract, and despite other aspects of bizarreness)? Is there any discernible association between the words or concepts of the question or proverb on the one hand, and the subject's statement on the other? Assign a score reflecting the distance of the response from the question or proverb in Category IV.
- Finally, consider whether the subject's physical, affective, or verbal behavior deviates from conventional norms during the test situation. If it does, then evaluate the degree and assign a score of 0, .5, 1, or 3 in Category V.

Each of the five categories is scored on the scoring sheet. The scoring of the above five categories is based primarily on the following 11 subcategories of disturbed language and thinking. These indicators have proved useful in orienting raters to the behavioral foundations of the five descriptive categories of idiosyncratic thinking. In addition, some of these subcategories may be related to specific mechanisms involved in bizarre-idiosyncratic thinking. Some bizarre-idiosyncratic responses may be influenced by, or in part be a consequence of, confusion-disorganization, a tendency to intermingle personal concerns when thinking about neutral material, a disorder in logical reasoning, or attending to part rather than all of a question. However, while these 11 subtypes of bizarre-idiosyncratic thinking may be important, they do not represent an exhaustive list of all possible types of bizarre behavior and ideas, or all the possible dimensions with which one can look at these phenomena. There are a vast number of ways responses can be strange and bizarre, and a vast number of ways that responses can deviate from social convention in an idiosyncratic manner.

Category I: Linguistic Form and Structure

1. Strange verbalizations.

a. Single words used in an unusual or peculiar manner (which are, in the rater's best judgment, not attributable to intellectual or cultural deficits).

b. Mild or moderate cognitive slippage in sentence structure, the expression of ideas, or the construction of new words (the new word is close in form to the correct word).

c. Neologisms (a new word with *private* meaning). Real neologisms (involving a private meaning) are very unusual, and are scored "3" to reflect their very severe idiosyncratic quality.

d. Artificial, pedantic, or stilted language, inappropriate to the level of discourse in the testing situation.

2. Lack of shared communication.

a. Responses that are not explicitly stated.

b. Small gaps in communication, in which words are not explained or referents are unclear.

c. Larger gaps in communication, in which phrases are not explained. Elements of private language may be apparent with unshared or unexplained concepts or ideas.

d. Disorganized or poor linkage between consecutive words, phrases, or sentences within the response.

Category II: Content of the Statement

3. Responses involving coherent but odd ideas.

4. Responses that are deviant with respect to social convention.

5. Peculiar or idiosyncratic reasoning or logic.

a. Responses that are incorrect and illogical in terms of common knowledge about people, events, or the environment.

b. Responses violating a logical paradigm, such as predicate logic.

c. Self-contradictory responses or responses with confused logic.

d. Responses with peculiar, autistic logic.

6. Confused or disorganized ideas.

a. Combinations of words put together in a manner that only dimly makes sense.

b. Grammatically correct sentences that do not hold a logical thought.

Category III: Intermixing

7. Overelaborated response.

a. Irrelevant wandering within a partially correct or correct answer.

b. Elaboration that is far too extensive, to the point where the original question is almost lost from sight.

8. Intermingling of personal concerns or associations into the response.

Category IV: Relationship Between Question and Response

9. Attending to a part of rather than the whole question: associations or interpretations of a word or phrase that suggest the subject's response is not based on the question as a conceptual whole, and also make the response appear strange or idiosyncratic.

10. Lack of a relationship between the subject's statement and the question asked—almost as if a different question is being asked.

Category V: Behavior

11. Strange behavior—including physical and affective behavior.

In scoring a response according to the list of major categories and subcategories outlined above, the scorer first analyzes the subject's response and scores it according to each of the 11 subcategories. The scorer then assigns scores on the five major categories. The presence of one type of subcategory of bizarre-idiosyncratic thinking in a response (e.g., lack of shared communication) does not exclude the simultaneous presence of another type or subcategory from that same response (e.g., intermingling or an overelaborated response).

The categories were constructed to focus on the distinct properties of a response that may contribute to an overall impression of unusual or odd verbal behavior. However, at times, although a category score is indicated (i.e., something unusual occurs in linguistic style, or in how a response is stated), the idiosyncracy is not attributable to any specific subcategory or behavioral indicator. In such instances, a category score for bizarre-idiosyncratic thinking is still justified in that particular area, while the individual behavioral descriptors are left blank.

The categories and the subcategories that they comprise are not an exhaustive list of all possible aspects of idiosyncratic thought and language. The overall response score is a general barometer of bizarre-idiosyncracy, and the categories and their foci are probes for the components of such verbal behavior. Any one response may be scored for overall bizarreness but may not fit neatly into any of the outlined categories or subcategories, with all of the categories and subcategories consequently rated as 0.

On the other hand, if a score of 1 is assigned for any of the five categories, an *overall* score of at least 1 is logically indicated, as the overall response or part of it is clearly bizarre or idiosyncratic. Although the overall score should be at least as great as that given in any individual category, the accumulation of category scores may, and often does, add up to more than the overall score. (The overall score for a response can only equal 0, .5, 1, or 3 and is *not* the summation of category scores.)

As we have indicated, many responses are scored in more than one category or subcategory. In a sample of hospitalized schizophrenic patients, for example, we found that of those responses scored for bizarre-idiosyncratic language and thought, approximately 50 percent were scored in one subcategory, 30 percent in two subcategories, and 20 percent in three or more subcategories. This ratio, however, may differ with varying populations.

Conclusion

In this article, we have summarized our method of assessing positive thought disorder in two verbal tests. This has included information on some of our conceptualizations about bizarre-idiosyncratic thinking and positive thought disorder, as well as the reliability and validity of our scoring system. While we have conveyed the essential aspects of our scoring system here, more thorough and detailed scoring procedures for the categories of bizarre-idiosyncratic thinking and additional scoring examples are presented in Harrow and Quinlan (1985).

References

Adler, D., and Harrow, M. *Manual for Assessing Components of Idiosyncratic or Bizarre Responses.* (ASIS/NAPS #02191) New York: Microfiche Publication, 1973.

Adler, D., and Harrow, M. Idiosyncratic thinking and personally overinvolved thinking in schizophrenic patients during partial recovery. *Comprehensive Psychiatry*, 15:57-67, 1974.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed.

Washington, DC: The Association, 1980.

Andreasen, N.C. The clinical assessment of thought, language and communication disorders. *Archives of General Psychiatry*, 36:1315–1321, 1979*a*.

Andreasen, N.C. The diagnostic significance of disorders in thought, language and communication disorders. *Archives of General Psychiatry*, 36:1325–1330, 1979*b*.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia. *Archives of General Psychiatry*, 39:789–794, 1982.

Benjamin, J.D. A method for distinguishing and evaluating formal thinking disorders in schizophrenia. In: Kasanin, J.S., ed. *Language and Thought in Schizophrenia*. Berkeley: University of California Press, 1944. pp. 65–90.

Berndt, D.J. "The Course of Thought Disorder at Acute and Posthospitalization Phases With an Emphasis on the Roles of Energy Level and Affective Disturbance." Ph.D. thesis, Loyola University, Chicago, IL, 1981.

Buss, A.H. and Lang, P.J. Psychological deficit in schizophrenia: I. Affect, reinforcement, and concept attainment. *Journal of Abnormal Psychology*, 70:2–24, 1965.

Chapman, L.J. Recent advances in the study of schizophrenic cognition. *Schizophrenia Bulletin*, 5:568–580, 1979.

Chapman, L.J., and Chapman, J.P. *Disordered Thought in Schizophrenia*. New York: Appleton Century Crofts, 1973.

Fish, F.J. *Schizophrenia*. Baltimore: William & Wilkins Co., 1962.

Goldstein, K. A methodological approach to the study of schizophrenic thought disorder. In: Kasanin, J.S., ed. *Language and Thought in Schizophrenia*. Berkeley: University of California Press, 1944. pp. 17–40.

Goldstein, K., and Scheerer, M. Abstract and concrete behavior: An experimental study with special tests. *Psychological Monographs*, 53: No. 2, 1941.

Gorham, D.R. A proverbs test for clinical and experimental use. *Psychological Reports*, 1:1–12, 1956.

Gottschalk, L.A., and Gleser, G.C. *The Measurement of Psychological States Through the Content Analysis of Verbal Behavior*. Berkeley and Los Angeles: University of California Press, 1969.

Harrow, M.; Grossman, D.; Silverstein, M.L.; and Meltzer, H. Thought pathology in manic and schizophrenic patients: At hospital admission and seven weeks later. *Archives of General Psychiatry*, 39:665–671, 1982.

Harrow, M.; King, G.; Marengo, J.; Rattenbury, F.; and Silverstein, M.L. "A Manual to Assess Positive Thought Disorder, Using the Object Sorting Test." Unpublished manual, 1985.

Harrow, M., and Marengo, J. Schizophrenic thought disorder at followup: Its persistence and prognostic significance. *Schizophrenia Bulletin*, 12:373–393, 1986.

Harrow, M.; Marengo, J.; and Lanin-Kettering, I. "Longitudinal Trends in Positive Thought Disorder in Schizophrenia and Psychosis." Presented at the Annual Meeting of the American Psychological Association, Anaheim, CA, August, 1983.

Harrow, M.; Marengo, J.; and McDonald, C. The early course of schizophrenic thought disorder. *Schizophrenia Bulletin*, 12:208–224, 1986.

Harrow, M., and Miller, J.G. Schizophrenic thought disorders and impaired perspective. *Journal of Abnormal Psychology*, 89:717–727, 1980.

Harrow, M., and Quinlan, D. Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry*, 34:15–21, 1977.

Harrow, M., and Quinlan, D. *Disordered Thinking and Schizophrenic Psychopathology*. New York: Gardner Press, 1985.

Harrow, M.; Silverstein, M.L.; and Marengo, J. Disordered thinking: Does it identify nuclear schizophrenia? *Archives of General Psychiatry*, 40:765–771, 1983.

Harrow, M.; Tucker, G.; and Adler, D. Concrete and idiosyncratic thinking in acute schizophrenic patients. *Archives of General Psychiatry*, 26:433–439, 1972.

Johnston, M.H., and Holzman, P.S. *Assessing Schizophrenic Thinking*. San Francisco: Jossey-Bass, 1979.

Lanin, I.B.; Berndt, D.; Harrow, M.; and Neiditz, J. "The Many Levels of Cohesive Speech Behavior: An Empirical Examination." Presented at the Parasession on Language and Behavior of the Chicago Linguistic Society, Chicago, IL, May 1981.

Lanin-Kettering, I. "Towards a Communicative Model of Discourse Cohesion." Ph.D. thesis, University of Chicago, Chicago, IL 1983.

Maher, B. The language of schizophrenia: A review and interpretation. *British Journal of Psychiatry*, 120:3–17, 1972.

Maher, B.; McKean, K.O.; and McLaughlin, B. Studies in psychotic language. In: Stone et al., eds. *General Inquirer*. Cambridge, MA: MIT Press, 1966. pp. 469–503.

Marengo, J. "The Prognosis of Thought Disordered Schizophrenics: A Followup Study." Ph.D. thesis, Northwestern University, Evanston, IL, 1983.

Marengo, J., and Harrow, M. Thought disorder: A function of schizophrenia, mania or psychosis? *Journal of Nervous and Mental Disease*, 173:35–41, 1985.

Marengo, J.; Harrow, M.; and Silverstein, M.L. "The Prognosis of Thought Disordered Schizophrenics." Presented at the Annual Meeting of the American Psychiatric Association, Toronto, Ont., Canada, May, 1982.

Payne, R. Disorders of thinking. In: Costello, G.C., ed. *Symptoms of Psychopathology: A Handbook.* New York: John Wiley & Sons, 1970. pp. 49–94.

Quinlan, D., and Harrow, M. Boundary disturbances in schizophrenia. *Journal of Abnormal Psychology*, 83:533–541, 1974.

Quinlan, D.; Harrow, M.; and Carlson, K. *Manual for Assessment of Deviant Responses on the Rorschach.* (ASIS/NAPS #02211) New York: Microfiche Publications, 1973.

Quinlan, D.; Harrow, M.; Tucker, G.J.; and Carlson, K. Varieties of "disordered" thinking on the Rorschach: Findings in schizophrenic and non-schizophrenic patients. *Journal of Abnormal Psychology*, 79:47–53, 1972.

Rapaport, D.; Gill, M.; and Schafer, R. *Diagnostic Psychological Testing.* Edited by R. Holt. New York: International Universities Press, 1968.

Reilly, R.; Harrow, M.; Tucker, G.J.; Quinlan, D.; and Siegel, A. Looseness of associations in acute schizophrenia. *British Journal of Psychiatry*, 127:240–246, 1975.

Silverstein, M.L.; Harrow, M.; and Marengo, J. "Thought Disorders: One or Many?" Unpublished manuscript, Michael Reese Medical Center, Chicago, IL, 1980.

Spitzer, R.L., and Endicott, J. *Research Diagnostic Criteria.* New York: New York State Psychiatric Institute, 1968.

Wechsler, D. *Wechsler Adult Intelligence Scale Manual.* New York: Psychological Corp., 1955.

Wechsler, D. *Wechsler Adult Intelligence Scale Manual: Revised Form.* New York: Psychological Corp., 1981.

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Appendix 1: Scored Protocol

1. Where there's a will, there's a way.

   Destiny is always truth.

2. Rome was not built in a day.

   You'd have to ask my brother that. He's an architect. In the Koran, my brother is Moses, so he's the best to answer that question. I'm studying the Romans in the Bible now. I'm studying their mental attitudes toward life. I'm in school now—University of Mozambique. It's through the mind I'm learning.[1]

3. When the cat's away, the mice will play.

   It refers to supervision—children get aggressive and selfish when their parents aren't there. Behavior is mischievous when there is no supervision.

4. Barking dogs seldom bite.

   Intimidation. (Q) A barking dog is trying to frighten or intimidate you.

5. A stream cannot rise higher than its source.

   Water doesn't run up hill.

6. Don't swap (trade) horses when crossing a stream.

   It is negative, a pyramid over water. Others out there disagree with me.

7. The used key is always bright.

   I don't know what that means in frontal knowledge, but not in lateral.

8. Gold goes in at any gate except heaven's.

   Heaven has a gate of gold. You can't buy heaven.

9. One swallow (bird) doesn't make a summer.

   Teaches patience.

10. The wife is the key to the house.

    She's the protective image of the household. She's the leader behind closed doors.

11. Riches serve a wise man but command a fool.

    That's true. (Enc.) It's the difference between sitting yoga and traveling or putting on your clothes and using transportation. (Q) It's transcendental sound.

12. Don't cast pearls before swine (pigs).

    Swine are only intended to eat ground or soil but not pearls which are the epitome of eternity.

---

[1] Subject has never attended a foreign university.

Subject I.D. ____________

Rater ____________ Date: ____________
Proverbs Form 1 Form 2 Form 3 Administration
Comprehension (circle one) Date: ____________
Scoring

Score sheet for bizarre-idiosyncratic responses

			I. Linguistic form or structure			II. Content of the statement, ideas expressed					III. Intermixing			IV. Relationship between response and question			V. Behavior
Response number		Overall score	Peculiar word form or use	Lack shared communication	Overall category I	Coherent but odd ideas	Deviant with respect to social convention	Illogic	Confused ideas	Overall category II	Overelaborated	Intermingled	Overall category III	Attention to limited part of stimulus	Lack of relationship to stimulus material	Overall cate gory IV	Overall category V
Prov/ Comp			1	2		3	4	5	6		7	8		9	10		11
1		1		1	1	1				1					1	1	
2		3									3	3	3	1		1	
3	3	0															
4	4	0															
5	5	0															
6	6	3		3	3												
7	7	3	3		3				3	3					3	3	
8	8	0															
9	9	.5		.5	.5												
10	10	0															
11	11	3	1	.5	1	3				3					3	3	
12	12	1				1				1							
	13																
	14																
Overall Sum		14.5	4.0	5.0	8.5	5.0	00	00	3.0	8.0	3.0	3.0	3.0	1.0	7.0	8.0	00
Sum. 1 & 3pt. rsp.		14	4	4	8	5	0	0	3	8	3	3	3	1	7	8	0
# of 0's		05	10	8	7	9	12	12	11	8	11	11	11	11	9	8	12
# of .5's		01	0	2	1	0	0	0	0	0	0	0	0	0	0	0	0
# of 1's		02	1	1	2	2	0	0	0	2	0	0	0	1	1	2	0
# of 3's		04	1	1	2	1	0	0	1	2	1	1	1	0	2	2	0

Note: Prov/Comp. = Proverbs test, Comprehension test; pt. rsp. = response point.

Bibliography on Schizophrenia

The bibliography that appears below is drawn from the Mental Health Abstracts database, produced by IFI/Plenum Data Company, 3202 Kirkwood Highway, Wilmington, DE 19808. The titles were selected for inclusion here on the basis of their direct relevance to schizophrenia: judgments about the quality of the article or book have been left to the reader. Due to space limitations, the *Schizophrenia Bulletin* has temporarily discontinued publishing the abstracts themselves; however, we hope to reinstitute this portion in the near future.

The articles listed below are not available from the Schizophrenia Research Branch, but are available for access through Dialog Information Retrieval Systems in reference departments of major university libraries and public libraries.

Attention, Perception, and Cognition

6747. Asarnow, R.F., and MacCrimmon, D.J. (Dept. of Psychiatry, University of California, Los Angeles, CA 90024) Span of apprehension deficits during the postpsychotic stages of schizophrenia: A replication and extension. Archives of General Psychiatry, 38:1006-1011, 1981.

6748. Cegalis, J.A.; Dellis, E.M.; and Sweeney, J.A. (Yale University, New Haven, CT 06520) Refixation saccades and attention in schizophrenia. Psychiatry Research, 7:189-198, 1982.

6749. Cegalis, J.A., and Sweeney, J.A. (Yale University, New Haven, CT 06520) The effect of attention on smooth pursuit eye movements of schizophrenics. Journal of Psychiatric Research, 16:145-162, 1981.

6750. Chattopadhyay, P.K.; Biswas, P.K.; and Mitra, A. (Dept. of Psychology, University College of Science, 92, A.P.C. Rd., Calcutta, India) Estimation of task-oriented time in schizophrenics, anxious neurotics and normals. Indian Journal of Clinical Psychology, 9:19-23, 1982.

6751. Crider, A.; McMahon, M.A.; and Solomon, P.R. (Williams College, Williamstown, MA 01267) Disruption of selective attention in the rat following chronic d-amphetamine administration: Relationship to schizophrenic attention disorder. Biological Psychiatry, 17:351-361, 1982.

6752. Iacono, W.G.; Johnson, R.A.; and Tuason, V.B. (Dept. of Psychology, University of British Columbia, Vancouver, Canada) Dissociation of smooth-pursuit and saccadic eye tracking in remitted schizophrenics: An ocular reaction time task that schizophrenics perform well. Archives of General Psychiatry, 38:991-996, 1981.

6753. Oades, R.D. (Max Planck Institut für Hirnforschung, Frankfurt, Federal Republic of Germany) Search strategies on a hole-board are impaired in rats with ventral tegmental damage: Animal model for tests of thought disorder. Biological Psychiatry, 17:243-258, 1982.

6754. Reich, S.S., and Cutting, J. (The London Hospital, Whitechapel, London, England) Picture perception and abstract thought in schizophrenia. Psychological Medicine, 12:91-96, 1982.

6755. Silverstein, M.L., and Harrow, M. (Illinois State Psychiatric Institute, 1601 W. Taylor St., Chicago, IL 60612) Inter-relationships among three measures of disordered thinking in continuous word association. Psychological Reports, 50:803-809, 1982.

6756. Walker, E., and McGuire, M. (Cornell University, Ithaca, NY 10021) Intra- and interhemispheric information processing in schizophrenia. Psychological Bulletin, 92:701-725, 1982.

Biology

6757. Baron, M.; Asnis, L.; Gruen, R.; Levitt, M.; Meltzer, H.Y.; and Perlman, R. (Columbia University College of Physicians and Surgeons, New York State Psychiatric Institute, New York, NY 10023) Plasma copper and dopamine-β-hydroxylase in schizophrenia. Biological Psychiatry, 17:115-120, 1982.

6758. Baron, M.; Cannova, G.; Levitt, M.; and Perumal, A.S. (New York State Psychiatric Institute, New York, NY 10023) Platelet monoamine oxidase in schizophrenia with b-phenylethylamine and benzylamine as substrates. Biological Psychiatry, 17:479-483, 1982.

6759. Baron, M.; Gruen, R.; Kane, J.; and Levitt, M. (New York State Psychiatric Institute, New York, NY 10023) Neuroleptic drug effect on platelet monoamine oxidase and plasma amine oxidase in schizophrenia. Psychiatry Research, 7:179-187, 1982.

6760. Cabranes, J.A.; Almoguera, M.I.; Borque, M.; Fuentenebro, F.; and Recio, M.N. (Dept. of Psychiatry and Dept. of Nuclear Medicine, Hospital Clinico de San Carlos, Universidad Complutense, Madrid, Spain). Prolactin secretion response to TRH stimulation in acute and chronic schizophrenic patients under neuroleptic treatment. International Pharmacopsychiatry, 17:1-7, 1982.

6761. DeLisi, L.E.; Goodman, S.; Neckers, L.M.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, St. Elizabeths Hospital, Washington, DC 20032) An analysis of lymphocyte subpopulations in schizophrenic patients. Biological Psychiatry, 17:1003-1009, 1982.

6762. Duncavage, M.; Luchins, D.J.; and Meltzer, H.Y. (University of Chicago, Pritzker School of Medicine, Chicago, IL 60601) Platelet MAO activity and family history of schizophrenia. Psychiatry Research, 7:47-51, 1982.

6763. Dunlop, S.R.; Hendrie, H.C.; Shea, P.A. (Indiana University School of Medicine, Indianapolis, IN 46202) The relationship between plasma and red blood cell neuroleptic levels, oral dosage, and clinical parameters in a chronic schizophrenic population. Biological Psychiatry, 17:929-936, 1982.

6764. Frith, C.D.; Crow, T.J.; Johnstone, E.C.; and Stevens, M. (Clinical Research Center, Division of Psychiatry, Norwick Park Hospital, Watford Road, Harrow, Middlesex HAI 3UJ, England) Skin conductance habituation during acute episodes of schizophrenia: Qualitative differences from anxious and depressed patients. Psychological Medicine, 12:575-583, 1982.

6765. Gillin, J.C.; Carpenter, W.T., Jr.; Hambidge, K.M.; Henkin, R.I.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, St. Elizabeths Hospital, Washington, DC 20032) Zinc and copper in patients with schizophrenia. Encephale, 8:435-444, 1982.

6766. Golden, C.J.; Berg, R.A.; Bloch, S.; Coffman, J.; Graber, B.; and Newlin, D.B. (Nebraska Psychiatric Institute, University of Nebraska Medical Center, Omaha, NE 68132) Structural brain deficits in schizophrenia: Identification by computed tomographic scan density measurements. Archives of General Psychiatry, 38:1014-1017, 1981.

6767. Hallert, C. (Linkoping University, Stockholm, Sweden) Psychiatric illness, gluten, and celiac disease. Biological Psychiatry, 17:959-961, 1982.

6768. Hallgren, R.; Venge, P.; and Wistedt, B. (No address given) Elevated serum levels of lactoferrin and eosinophil cationic protein in schizophrenic patients. British Journal of Psychiatry, 140:55-60, 1982.

6769. Ho, B.T.; Allen R.; Dejohn, C.; Khan, M.; Kralik, P.; Schooler, J.; and Smith, R.C. (Texas Research Institute of Mental Sciences, Texas Medical Center, Houston, TX 76101) Effects of neuroleptics on platelet monoamine oxidase activity. Biological Psychiatry, 17:885-895, 1982.

6770. Karson, C.N.; Apostoles, P.S.; Bridge, T.P.; Phelps, B.H.; Potkin, S.G.; Wise, C.D.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, St. Elizabeths Hospital, Washington, DC 20032) The effects of oral glucose on platelet monoamine oxidase. Biological Psychiatry, 17:1011-1015, 1982.

6771. Kleinman, J.E.; Bunney, W.E., Jr.; Davis, G.C.; Mendelson, W.B.; Rogol, A.; Shiling, D.J.; Weinberger, D.R.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, St. Elizabeths Hospital, Washington, DC 20032) Naloxone in chronic schizophrenic patients: Neuroendocrine and behavioral effects. Psychiatry Research, 7:1–7, 1982.

6772. Kozhushko, L.F.; Kozlova, I.A.; and Leibovich, F.A. (Institute of Psychiatry, Academy of Medical Sciences of the USSR, Moscow, USSR) Characteristics of the parental EEG related to the course of schizophrenia in children. Neuroscience and Behavioral Physiology, 12:101–106, 1982.

6773. Latham, C.; Holzman, P.S.; Manschreck, T.C.; and Tole, J. (Dept. of Psychiatry, Harvard Medical School, Boston, MA 02115) Optokinetic nystagmus and pursuit eye movements in schizophrenia. Archives of General Psychiatry, 38:997–1003, 1981.

6774. Malas, K.L., and van Kammen, D.P. (Biological Psychiatry Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD 20205) Markedly elevated creatine phosphokinase levels after neuroleptic withdrawal. American Journal of Psychiatry, 139:231–232, 1982.

6775. Meltzer, H.Y.; Lewine, R.R.J.; and Perline, R. (University of Chicago Pritzker School of Medicine, Chicago, IL 60637) Biological studies of DSM-III psychotic disorders. I. Platelet measures and apomorphine-induced growth hormone response. Journal of Nervous and Mental Disease, 170:758–765, 1982.

6776. Mooney, J.J.; Adler, S.A.; Chao, F.C.; Orsulak, P.J.; and Schildkraut, J.J. (Dept. of Psychiatry, Harvard Medical School, Boston, MA 02115) Platelet monoamine oxidase activity in psychiatric disorders: The application of a technique for the isolation of free platelet mitochondria from relatively small blood samples. Journal of Psychiatric Research, 16:163–172, 1981.

6777. Myslobodsky, M.S., and Kofman, O. (Tel-Aviv University, Ramat Aviv, Israel) Unilateral versus bilateral electroconvulsive shock in albino rats: Comparison of behavioral symptomatology and neocortical reactivity changes. Biological Psychiatry, 17:363–380, 1982.

6778. Nasrallah, H.A.; Damasio, H.; Jacoby, C.G.; Kuperman, S.; McCalley-Whitters, M.; and Rizzo, M. (University of Iowa, Iowa City, IA 52240) Neurological differences between paranoid and nonparanoid schizophrenia: Part II. Computerized tomographic findings. Journal of Clinical Psychiatry, 43:307–309, 1982.

6779. Nasrallah, H.A.; Kuperman, S.; and McCalley-Whitters, M. (University of Iowa, Iowa City, IA 52240) Neurological differences between paranoid and nonparanoid schizophrenia: Part I. Sensory-motor lateralization. Journal of Clinical Psychiatry, 43:305–306, 1982.

6780. Nasrallah, H.A.; Kuperman, S.; McCalley-Whitters, M.; and Tippin, J. (University of Iowa, Iowa City, IA 52240) Neurological differences between paranoid and nonparanoid schizophrenia: Part III. Neurological soft signs. Journal of Clinical Psychiatry, 43:310–312, 1982.

6781. Nyback, H.; Berggren, B.-M.; Hindmarsh, T.; and Wiesel, F.-A. (Departments of Psychiatry and Neuroradiology, Karolinska Hospital, Stockholm, Sweden) Computed tomography of the brain in patients with acute psychosis and in healthy volunteers. Acta Psychiatrica Scandinavica, 65:403–414, 1982.

6782. Propert, D.N. (University of Melbourne, Parkville, Victoria, Australia) Immunoglobulin allotypes G_m and K_m in chronic schizophrenia: No apparent association. Psychological Medicine, 13:27–30, 1983.

6783. Rothschild, A.J.; Cole, J.O.; Rosenbaum, A.H.; Schatzberg, A.F.; and Stahl, J.B. (Harvard Medical School, Boston, MA 02115) The dexamethasone suppression test as a discriminator among subtypes of psychotic patients. British Journal of Psychiatry, 141:471–474, 1982.

6784. Rotman, A.; Shatz, A.; and Szekely, G.A. (No address given) Correlation between serotonin uptake and imipramine binding in schizophrenic patients. Progress in Neuropsychopharmacology, 6:57–61, 1982.

6785. Scarone, S.; Cazzullo, C.L.; Gambini, O.; Massironi, R.; and Pieri, E. (Dept. of Psychiatry, Milan Medical School, 1,20161 Milan, Italy) The asymmetric lateralization of tactile extinction in schizophrenia: The possible role of limbic and frontal regions. British Journal of Psychiatry, 141:350–353, 1982.

6786. Schayer, R.W.; Kazimierczak, W.; Maslinski, C.; Pilc, A.; and Zaborwski, A. (Rockland Research Institute, Orangeburg, NY 10962) The histamine-induced skin reaction in schizophrenic and normal subjects.

Journal of Clinical Psychiatry, 43:7-9, 1982.

6787. Schulz, S.C.; Cohen, M.R.; Naber, D.; Pickar, D.; and van Kammen, D.P. (Medical College of Virginia, Richmond, VA 23219) Response of plasma β-endorphin immunoreactivity to *d*-amphetamine and placebo in schizophrenic patients. Psychiatry Research, 7:171-178, 1982.

6788. Smythies, J.R. (University of Alabama School of Medicine, Birmingham, AL 35233) Biological markers for the schizophrenic and atypical psychoses. Journal of Nervous and Mental Disease, 170:732-736, 1982.

6789. Sternbach, H.; Gerner, R.H.; and Gwirtsman, H.E. (University of California, Los Angeles, CA 90024) The thyrotropin releasing hormone stimulation test: A review. Journal of Clinical Psychiatry, 43:4-6, 1982.

6790. Turner, W.J. (No address given) Dexamethasone suppression test: Problems and promise. Biological Psychiatry, 17:1-2, 1982.

6791. Vanherweghem, J.-L.; Linkowski, P.; and Mendlewicz, J. (University Clinic of Brussels, Erasme Hospital, Free University of Brussels, Brussels, Belgium) Hemodialysis in schizophrenics: A double-blind study. Archives of General Psychiatry, 40:211-214, 1983.

6792. Widerlov, E.; Andersson, U.; Breese, G.R.; Haggstrom, J.-E.; Kilts, C.D.; and Mailman, R.B. (Psychiatric Research Center, Ulleraker Hospital, University of Uppsala, S-75017 Uppsala, Sweden) Serum concentrations of thioridazine, its major metabolites and serum neuroleptic-like activities in schizophrenics with and without tardive dyskinesia. Acta Psychiatrica Scandinavica, 66:294-305, 1982.

6793. Wolf, M.E.; Bowie, L.; Keener, S.; and Mosnaim, A.D. (VA Medical Center, North Chicago, IL 60064) Prolactin response in tardive dyskinesia. Biological Psychiatry, 17:485-490, 1982.

6794. Yesavage, J.A.; Becker, J.; Cohn, R.; Holman, C.A.; Mills, M.J.; and Werner, P.D. (VA Medical Center, Palo Alto, CA 94304) Serum level monitoring of thiothixene in schizophrenia: Acute single-dose levels at fixed doses. American Journal of Psychiatry, 139:174-178, 1982.

Case Studies

6795. Director, K.L., and Muniz, C.E. (University of Florida, Gainesville, FL 32601) Diazepam in the treatment of extrapyramidal symptoms: A case report. Journal of Clinical Psychiatry, 43:160-161, 1982.

6796. Gomez, E.A.; Comstock, B.S.; and Rosario, A. (Baylor College of Medicine, Houston, TX 77025) Organic versus functional etiology in catatonia: Case report. Journal of Clinical Psychiatry, 43:200-201, 1982.

6797. Flaherty, J.A. (University of Illinois Medical Center, Chicago, IL 60612) Three generations of schizophrenia. Journal of Clinical Psychiatry, 43:204-205, 1982.

6798. Jorgensen, H.A., and Cappelen, C., Jr. (No address given) Naloxone-induced reduction of schizophrenic symptoms. A case report. Acta Psychiatrica Scandinavica, 65:370-374, 1982.

Childhood Psychosis

6799. Bauer, W., and Bauer, J.L. (2727 North Mayfair Rd., Milwaukee, WI 53222) Adolescent schizophrenia. Adolescence, 17:685-694, 1982.

6800. Berlin, I.N., and Critchley, D.L. (University of New Mexico School of Medicine, Albuquerque, NM 87106) The work of play for parents of schizophrenic children. Child Psychiatry and Human Development, 13:111-119, 1982.

6801. Kydd, R.R., and Werry, J.S. (No address given) Schizophrenia in children under 16 years. Journal of Autism and Developmental Disorders, 12:343-357, 1982.

Cross-Cultural Studies

6802. Burvill, P.W.; Carlson, J.; Reymond, J.; and Stampfer, H. (Depts. of Psychiatry and Behavioural Science, University of Western Australia, Perth, Australia) Relationship between country of birth and psychiatric admissions in Western Australia. Acta Psychiatrica Scandinavica, 66:322-335, 1982.

6803. Ihezue, U.H. (Dept. of Psychological Medicine, University of Nigeria, Nigeria, Africa) Some observations and comments on the psychosocial profile of first-ever referral to the psychiatric hospital, Enugu, Nigeria. Acta Psychiatrica Scandinavica, 65:355-364, 1982.

6804. Murphy, H.B.M., and Vega, G. (Department of Psychiatry,

best copy available

McGill University, Montreal, Canada) Schizophrenia and religious affiliation in northern Ireland. Psychological Medicine, 12(3):595-605, 1982.

6805. Wiersma, D.; De Jong, A.; Giel, R.; and Sloof, C.J. (No address given) Social class and schizophrenia in a Dutch cohort. Psychological Medicine, 13:141-150, 1983.

Description

6806. Berent, S.; Boll, T.J.; and Giordani, B. (Ann Arbor VA Medical Center, Ann Arbor, MI 48105) Rod-and-frame and MMPI scores for a group of female psychiatric inpatients. Perceptual and Motor Skills, 54:907-913, 1982.

6807. Clayton, P.J. (University of Minnesota, Minneapolis, MN 55455) Schizoaffective disorders. Journal of Nervous and Mental Disease, 170: 646-650, 1982.

6808. Emery, R.; Neale, J.M.; and Weintraub, S. (State University of New York at Stony Brook, Stony Brook, NY 11790) Effects of marital discord on the school behavior of children of schizophrenic, affectively disordered, and normal parents. Journal of Abnormal Child Psychology, 10:215-228, 1982.

6809. Evenson, R.C.; Cho, D.W.; Nuttall, E.A.; and Wood, J.B. (Missouri Institute of Psychiatry, St. Louis, MO 63139) Suicide rates among public mental health patients. Acta Psychiatrica Scandinavica, 66:254-264, 1982.

6810. Fawcett, J.; Clark, D.C.; Gibbons, R.D.; and Scheftner, W.A. (Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 62290) Assessing anhedonia in psychiatric patients: The Pleasure Scale. Archives of General Psychiatry, 40:79-84, 1983.

6811. Fischer, E.H.; Council, J.R.; Eastman, A.; Farina, A.; Millard, R.; and Pitts, H. (Connecticut Valley Hospital, Middletown, CT 06457) Influence of adjustment and physical attractiveness on the employability of schizophrenic women. Journal of Clinical Child Psychology, 50:530-534, 1982.

6812. Hogan, T.P.; Awad, A.G.; and Eastwood, R. (Queen Street Mental Health Centre, Toronto, M6J 1H4, Canada) A self-report scale predictive of drug compliance in schizophrenics: Reliability and discriminative validity. Psychological Medicine, 13:177-183, 1983.

6813. Landmark, J. (No address given) A manual for the assessment of schizophrenia. Acta Psychiatrica Scandinavica, 65:1-88, 1982.

6814. Lesser, I.M.; Friedmann, C.T.H.; and Faguet, R.A. (University of California at Los Angeles, Los Angeles, CA 90024) Unusual presentations of schizophrenia: Extraordinary disorders of human behavior. In: Critical Issues in Psychiatry. New York: Plenum Press, 1982. pp. 293-306.

6815. Lindsay, W.R. (Monklands District General Hospital, Air Drie, Lanarkshire M16 OJS, Scotland) The effects of labeling: Blind and nonblind ratings of social skills in schizophrenic and nonschizophrenic control subjects. American Journal of Psychiatry, 139:216-219, 1982.

6816. McEvoy, J.P.; Abernethy, V.; Appelbaum, P.S.; and Hatcher, A. (Western Psychiatric Institute and Clinic, Pittsburgh, PA 15213) Chronic schizophrenic women's attitudes toward sex, pregnancy, birth control, and childrearing. Hospital & Community Psychiatry, 34:536-539, 1983.

6817. Mohamed, S.N.; Disney, T.F.; Kazarian, S.; and Merskey, M. (London Psychiatric Hospital, 850 Highbury Ave., P.O. Box 2532, London, Ontario, Canada N6A 4HL) An investigation of the possible inverse relationships between the occurrence of rheumatoid arthritis, osteoarthritis and schizophrenia. Canadian Journal of Psychiatry, 27:381-383, 1982.

6818. Morrison, J.R. (No address given) Suicide in a psychiatric practice population. Journal of Clinical Psychiatry, 43:348-352, 1982.

6819. Nakai, H. (Kobe University, Kobe, Japan) Longitudinal and multidimensional observation of schizophrenics in their remission. Indonesian Psychiatric Quarterly, 15:65-71, 1982.

6820. Ndetei, D.M., and Singh, A. (University of Nairobi, Dept. of Psychiatry, Medical School, P.O. Box 30588, Nairobi, Kenya, Africa) Schizophrenia with depression: Causal or coexistent? British Journal of Psychiatry, 141:354-356, 1982.

6821. Parnas, J.; Jensen, P.S.; Korsgaard, S.; and Krautwald, O. (University Department of Psychiatry, Copenhagen Municipal Hospital, Copenhagen, Denmark) Chronic psychosis in epilepsy: A clinical investigation of 29 patients. Acta Psychiatrica Scandinavica, 66:282-293, 1982.

6822. Schueler, D.E.; Herron, W.G.; Poland, H.V.; and Schultz, C.L. (St. Johns University, Jamaica, NY 11432) Defense mechanisms in reactive and process schizophrenics. Journal of Clinical Psychology, 38:486-489, 1982.

6823. Shur, E. (Maudsley and Bethlem Royal Hospitals, London, England) Family history and schizophrenia: Characteristics of groups with and without positive family histories. Psychological Medicine, 12:591-594, 1982.

6824. Soloff, P.H., and Millward, J.W. (Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213) Psychiatric disorders in the families of borderline patients. Archives of General Psychiatry, 40:37-44, 1983.

6825. Taylor, P.; Dalton, R.; and Fleminger, J.J. (No address given) Handedness and schizophrenic symptoms. British Journal of Medical Psychology, 55:287-291, 1982.

6826. Turkat, D., and Buzzel, V. (No address given) The relationship between family violence and hospital recidivism. Hospital & Community Psychiatry, 34:552-553, 1983.

6827. Brockington, I.F.; Hillier, V.E.; Kendell, R.E.; Perris, C.; and Wainwright, S. (University of South Manchester, West Didsbury, Manchester, England) The course and outcome of cycloid psychosis. Psychological Medicine, 12:97-105, 1982.

Diagnosis

6828. Andreasen, N.C., and Akiskal, H.S. (University of Iowa College of Medicine, Iowa City, IA 52242) The specificity of Bleulerian and Schneiderian symptoms: A critical reevaluation. Psychiatric Clinics of North America, 6:41-54, 1983.

6829. Ban, T.; Guy, W.; Kelwala, S.; and Wilson, W.H. (Tennessee Neuropsychiatric Institute, Clinical Research Service, Nashville, TN 37202) Psychopharmacology and Leonhard's classification of chronic schizophrenias. International Pharmacopsychiatry, 17:153-162, 1982.

6830. Grove, W.M. (No address given) Psychometric detection of schizotypy. Psychological Bulletin, 92:27-38, 1982.

6831. Gunderson, J.G.; Siever, L.J.; and Spaudling, W. (McLean Hospital, 115 Mill Street, Belmont, MA 02178) The search for a schizotype: Crossing the border again. Archives of General Psychiatry, 40:15-22, 1983.

6832. Lazar, Z.L., and Schwartz, F. (New York Hospital, Cornell Medical Center, Westchester Division, New York, NY 10021) The contaminated Rorschach response: Formal features. Journal of Clinical Psychology, 38:415-419, 1982.

6833. Ndetei, D.M., and Singh, A. (University of Nairobi, Dept. of Psychiatry, Medical School, P.O. Box 30588, Nairobi, Kenya, Africa) Study of delusions in Kenyan schizophrenic patients diagnosed using a set of research diagnostic criteria. Acta Psychiatrica Scandinavica, 66:208-215, 1982.

6834. Page, S. (Dept. of Psychology, University of Windsor, Windsor, Ontario, Canada) Psychologist and physician diagnoses of hospitalized patients with similar MMPI symptomatology. Canadian Journal of Psychiatry, 27:471-473, 1982.

6835. Pi, E.H., and Surawicz, F.G. (University of Southern California, Los Angeles, CA 90024) Schizoaffective disorder (Schneiderian positive), manic type: A comparison with mania. Journal of Clinical Psychiatry, 43:235-236, 1982.

6836. Pope, H.G., Jr.; Cohen, B.M.; Gunderson, J.G.; Hudson, J.I.; and Jonas, J.M. (Harvard Medical School, Boston, MA 02115) The validity of DSM-III borderline personality disorder. A phenomenologic, family history, treatment response, and long-term followup study. Archives of General Psychiatry, 40:23-30, 1983.

6837. Sillitti, J. (Friends Hospital, Philadelphia, PA 19124) MMPI-derived indicators of organic brain dysfunction. Journal of Clinical Psychology, 38:601-605, 1982.

6838. Tamminga, C.A., and Carpenter, W.T., Jr. (Maryland Psychiatric Research Center, P.O. Box 3235, Baltimore, MD 21228) The DSM-III diagnosis of schizophrenic-like illness and the clinical pharmacology of psychosis. Journal of Nervous and Mental Disease, 170:744-751, 1982.

6839. Templer, D.I., and Veleber, D.M. (California School of Professional Psychology, 1350 M St., Fresno, CA 93721) The decline of hebephrenic schizophrenia. Journal of Orthomolecular Psychiatry, 11:100-102, 1982.

Epidemiology

6840. Ramsay, R.A.; Ananth, J.; Engelsmann, F.; Jarosz, M.;

Krakowski, A.J.; and Rydzynski, Z. (Allan Memorial Institute, Montreal, Quebec, Canada) International comparison of the prevalence of psychosomatic disorders in schizophrenic patients. Psychotherapy and Psychosomatics, 38:206-220, 1982.

The Family

6841. Goldstein, M.J., and Doane, J.A. (University of California, Los Angeles, CA 90024) Family factors in the onset, course, and treatment of schizophrenic spectrum disorders: An update on current research. Journal of Nervous and Mental Disease, 170:692-700, 1982.

6842. Scharfetter, C. (Psychiatric University Clinic of Zurich, Lenggstrasse 31, P.O. Box 68, CH-8029 Zurich, Switzerland) Schizophrenia's classical subtypes: A family heredity study. Archiv für Psychiatrie und Nervenkrankheiten, 231:443-447, 1982.

Genetics

6843. Gershon, E.S., and Nurnberger, J.I., Jr. (Biological Psychiatry Branch, National Institute of Mental Health, Bldg. 10, Rm. 3N218, Bethesda, MD 20892) Inheritance of major psychiatric disorders. Trends in Neurosciences, 5:241-242, 1982.

6844. Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. (Dept. of Psychiatry, Bronx VA Medical Center, Bronx, NY 11209) An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia: I. The relationship between anxiety disorder and schizophrenia. Archives of General Psychiatry, 38:973-977, 1981.

6845. Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. (Dept. of Psychiatry, Bronx VA Medical Center, Bronx, NY 11209) An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia: II. The relationship between schizotypal personality disorder and schizophrenia. Archives of General Psychiatry, 38:982-984, 1981.

6846. Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. (Dept. of Psychiatry, Bronx VA Medical Center, Bronx, NY 11209) An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia: III. The relationship between paranoid psychosis (delusional disorder) and the schizophrenia spectrum disorders. Archives of General Psychiatry, 38:985-987, 1981.

6847. Lange, V. (Human Genetics, Dept. of Biology, University of Frankfurt, Frankfurt, Federal Republic of Germany) Genetic markers for schizophrenic subgroups. Psychiatria Clinica, 15:133-144, 1982.

6848. McGuffin, P.; Festenstein, H.; and Murray, R. (Institute of Psychiatry, De Crespigny Park, Denmark Hill, London, England SE5 8AF) A family study of HLA antigens and other genetic markers in schizophrenia. Psychological Medicine, 13:31-43, 1983.

6849. O'Rourke, D.H.; Gottesman, I.I.; Reich, T.; Rice, J.; and Suarez, B.K. (Washington University School of Medicine, St. Louis, MO 63110) Refutation of the general single-locus model for the etiology of schizophrenia. American Journal of Human Genetics, 34:630-649, 1982.

6850. Watt, D.C. (No address given) The search for genetic linkage in schizophrenia. British Journal of Psychiatry, 140:532-537, 1982.

High-Risk Studies

6851. Harvey, P.D.; Neale, J.M.; and Weintraub, S. (No address given) Speech competence of children vulnerable to psychopathology. Journal of Abnormal Child Psychology, 10:373-387, 1982.

Prognosis

6852. Goldstein, J.M., and Caton, C.L.M. (New York State Psychiatric Institute, New York, NY 10023) The effects of the community environment on chronic psychiatric patients. Psychological Medicine, 13:193-199, 1983.

6853. Nantonakis, J.E.; Christodoulou, G.N.; Jemos, J.J.; and Lykouras, E.P. (Dept. of Psychiatry, University of Athens, Athens, Greece) Short-term social prognosis of schizophrenia. Acta Psychiatrica Scandinavica, 66:306-310, 1982.

Psychiatry and the Law

6854. Marder, S.R.; Chien, C.-P.; Mebane, A.; Swann, E.; van Putten, T.; and Winslade, W.J. (West Los Angeles VA Hospital, Los Angeles, CA 90025) A comparison of patients who refuse and consent to neuroleptic treatment. American Journal of Psychiatry, 140:470-472, 1983.

Psychological Theory

6855. Cutting, J., and Ryan, K. (King's College Hospital, London, England) The appreciation of imagery by schizophrenics: An interpretation of Goldstein's impairment of the abstract attitude. Psychological Medicine, 12:585-590, 1982.

Treatment

6856. Bellack, A.S.; Morrison, E.L.; Hersen, M.; and Kazdin, A.E. (Medical College of Pennsylvania, Philadelphia, PA 19104) Interpersonal dysfunction. In: Bellack, A.S., ed. International Handbook of Behavior Modification and Therapy. New York: Plenum Press, 1982. pp. 717-747.

6857. Braden, W.; Fink, E.B.; Ho, C.K.; Qualls, C.B.; and Samuels, W.O. (No address given) Lithium and chlorpromazine in psychotic inpatients. Psychiatry Research, 7:69-81, 1982.

6858. Cox, M. (London Hospital, London, England) "I took a life because I needed one": Psychotherapeutic possibilities with the schizophrenic offender-patient. Psychotherapy and Psychosomatics, 37:96-105, 1982.

6859. Curran, J.P.; Kazdin, A.E.; Monti, P.M.; Bellack, A.S.; and Hersen, M. (Brown University Medical School, Providence, RI 02912) Treatment of schizophrenia. In: Bellack, A.S., ed. International Handbook of Behavior Modification and Therapy. New York: Plenum Press, 1982.

6860. Delva, N.J., and Letemendia, F.J.J. (Dept. of Psychiatry, Queen's University, Kingston, Ontario, Canada) Lithium treatment in schizophrenia and schizoaffective disorders. British Journal of Psychiatry, 141:387-400, 1982.

6861. Frye, P.E.; Kim, M.H.; O'Shaughnessy, R.W.; and Pariser, S.F. (Ohio State University, Columbus, OH 43210) Bromocriptine associated with symptom exacerbation during neuroleptic treatment of schizoaffective schizophrenia. Journal of Clinical Psychiatry, 43:252-253, 1982.

6862. Haggstrom, J.-E.; Andersson, U.; and Gunne, L.M. (Psychiatric Research Center, Ulleraker Hospital, University of Uppsala, S-75017 Uppsala, Sweden) Bromocriptine in tardive dyskinesia. Pharmakopsychiatrie/Neuro-psychopharmakologie, 15:161-163, 1982.

6863. Lehmann, E.; Brauer, H.; Kretschmar, J.H.; and Reinhardt, B. (Psychiatric Clinic, University of Dusseldorf, Rheinisches Landeskrankenhaus, Bergische Landstrabe 2,4000 Dusseldorf 12, Federal Republic of Germany) Interaction of changes in hospital environment and neurotropic co-medication in chronic psychiatric inpatients. Pharmakopsychiatrie/Neuro-psychopharmakologie, 15:57-60, 1982.

6864. Leone, N.F. (No address given) Response of borderline patients to loxapine and chlorpromazine. Journal of Clinical Psychiatry, 43:148-150, 1982.

6865. McCarron, M.M.; Boettger, M.L.; and Peck, J.J. (No address given) A case of neuroleptic malignant syndrome successfully treated with amantadine. Journal of Clinical Psychiatry, 43:381-382, 1982.

6866. McGlashan, T.H. (Chestnut Lodge, 500 W. Montgomery Avenue, Rockville, MD 20850) DSM-III schizophrenia and individual psychotherapy. Journal of Nervous and Mental Disease, 170:752-757, 1982.

6867. Malm, U. (Dept. of Psychiatry Sahlgren's Hospital, University of Gothenburg, Gothenburg, Sweden) The influence of group therapy on schizophrenia. Acta Psychiatrica Scandinavica, Suppl., 297:1-65, 1982.

6868. Neborsky, R.; Depry, D.; Hornbeck, C.; Janowsky, D.; and Munson, E. (University of California, La Jolla, CA 92037) Behavioral prediction of response to haloperidol: A test dose strategy. Journal of Clinical Psychiatry, 43:157-158, 1982.

6869. Nurnberg, H.G., and Suh, R. (Mt. Sinai School of Medicine, New York, NY 10029) Time-limited psychotherapy of the hospitalized borderline patient. American Journal of Psychotherapy, 36:82-90, 1982.

6870. Odejide, O.A., and Aderounmu, A.F. (University College Hospital, Ibadan, Nigeria, Africa) Double-blind placebo substitution: Withdrawal of fluphenazine decanoate in schizophrenic patients. Journal of Clinical Psychiatry, 43:195-196, 1982.

6871. Pickar, D.; Vartanian, F.; Bunney, W.E., Jr.; Maier, H.P.; Gastpar, M.T.; Prakash, R.; Sethi, B.B.; Lideman, R.; and Belyaev, B.S. (Biological Psychiatry Branch, National Institute of Mental Health, Bldg. 10, Room 2N210, 9000 Rockville Pike, Bethesda, MD 20892) Short-term naloxone administration in schizophrenic and manic patients: A World Health Organization collaborative study. Archives of General Psychiatry, 39:313-319, 1982.

6872. Sandel, S.L. (19 Edwards St., New Haven, CT 06511) The process of individuation in dance-movement therapy with schizophrenic patients. Arts in Psychotherapy, 9:11-18, 1982.

6873. Schneider, S.J. (VA Hospital, Montrose, NY 10548) Electrodermal activity and therapeutic response to neuroleptic treatment in chronic schizophrenic inpatients. Psychological Medicine, 12:607-613, 1982.

6874. Seeman, M.V.; Blake, P.; Denberg, D.; Freire, M.; and Pyke, J. (University of Toronto, Clarke Institute of Psychiatry, Toronto, Ontario, Canada) Co-therapy in a clinic for schizophrenia. Canadian Journal of Psychiatry, 27:296-300, 1982.

6875. Skrabanek, P. (Misericordiae Hospital, Dublin, Ireland) Hemodialysis in schizophrenia. Lancet, 1:1404-1405, 1982.

6876. Staniforth, D.H.; Yorkston, N.J.; and Zaki, S.A. (Dept. of Clinical Pharmacology, University College Hospital, London, England) Dose dependent changes in propranolol half life when used as an adjunct to neuroleptic treatment. Psychopharmacology, 76:62-65, 1982.

6877. Stanton, A.H. (McLean Hospital, 115 Mill St., Belmont, MA 02178) Frieda Fromm-Reichmann: A seminar on the history of psychiatry. VII. Frieda Fromm-Reichmann, M.D.: Her impact on American Psychiatry: Journal for the Study of Interpersonal Processes, 45:121-127, 1982.

6878. Storms, L.H.; Clopton, J.M.; and Wright, C. (Psychology Service (116B), VA Medical Center, San Diego, CA 92161) Effects of gluten on schizophrenics. Archives of General Psychiatry, 39:323-327, 1982.

6879. Takashi, R.; Asada, S.; Atsuki, S.; Inanaga, K.; Nakano, S.; Samejima, K.; and Sarai, K. (Dept. of Neuropsychiatry, Nagasaki University, Nagasaki, Japan) Comparison of efficacy of a new butyrophenone derivative, timiperone, and perphenazine in schizophrenia by a multicenter controlled study. Journal of International Medical Research, 10:257-267, 1982.

6880. van Hassel, J.H.; Bloom, L.J.; and Gonzalez, A.M. (Dept. of Psychology, Colorado State University, Andrew G. Clark Bldg., Fort Collins, CO 80523) Anxiety management with schizophrenic outpatients. Journal of Clinical Psychology, 38:280-285, 1982.

6881. van Kammen, D.P.; Docherty, J.P.; and Bunney, W.E., Jr. (National Institute of Mental Health, 9000 Rockville Pike, Bldg. 10, Room 4N214, Bethesda, MD 20892) Prediction of early relapse after pimozide discontinuation by response to d-amphetamine during pimozide treatment. Biological Psychiatry, 17:233-242, 1982.

6882. van Praag, H.M.; de Wied, D.; van Ree, J.M.; and Verhoeven, W.M.A. (Dept. of Psychiatry, University of Utrecht, Utrecht, The Netherlands) The treatment of schizophrenic psychoses with γ-type endorphins. Biological Psychiatry, 17:83-98, 1982.

6883. Waring, E.M. (Victoria Hospital, Dept. of Psychiatry, 375 South Street, London, Ontario, Canada N6A AG5) Cognitive family therapy in the treatment of schizophrenia. Psychiatric Journal of the University of Ottawa, 6:229-233, 1981.

6884. West, A.P. (VA Medical Center, Sepulveda, CA 91343) Lithium neurotoxicity in schizophrenic and schizoaffective patients: Behavioral state-related aspects of neurotoxicity. International Pharmacopsychiatry, 17:91-103, 1982.

6885. Wilson, L.G.; Gerber, C.J.; Johnson, M.H.; and Roberts, R.W. (University of Washington, Seattle, WA 98105) Pimozide versus chlorpromazine in chronic schizophrenia: A 52-week double-blind study of maintenance therapy. Journal of Clinical Psychiatry, 43:62-65, 1982.

6886. Wollersheim, J.P.; Bordewick, M.; Knapp, M.; McLellarn, R.; and Paul, W. (University of Montana, Missoula, MT 59812) The influence of therapy rationales upon perceptions of clinical problems. Cognitive Therapy and Research, 6:167-172, 1982.

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The Family Support Group of the Northeast Ohio Chapter of Alliance for the Mentally Ill is compiling a resource list of all facilities that are available to and provide services for those afflicted with schizophrenia. All information, inquiries, and cooperation are solicited. Please contact:

M. Jaffe
2447 Edgerton Road
Cleveland, OH 44118

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The Menninger Foundation announces the availability of the May issue of the *Bulletin of the Menninger Clinic.* This volume features "Update on Schizophrenia," a collection of papers from the Seventh Annual Vail Psychiatry Conference held March 16-22, 1985, in Vail, Colorado. Topics include electrophysiological studies, community residential treatment, the changing role of families, and long-term followup to assess the effects of psychotherapy.

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For further information, contact:
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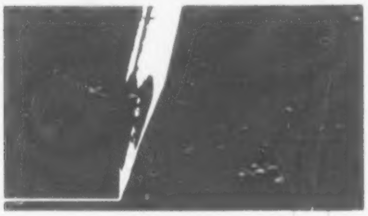

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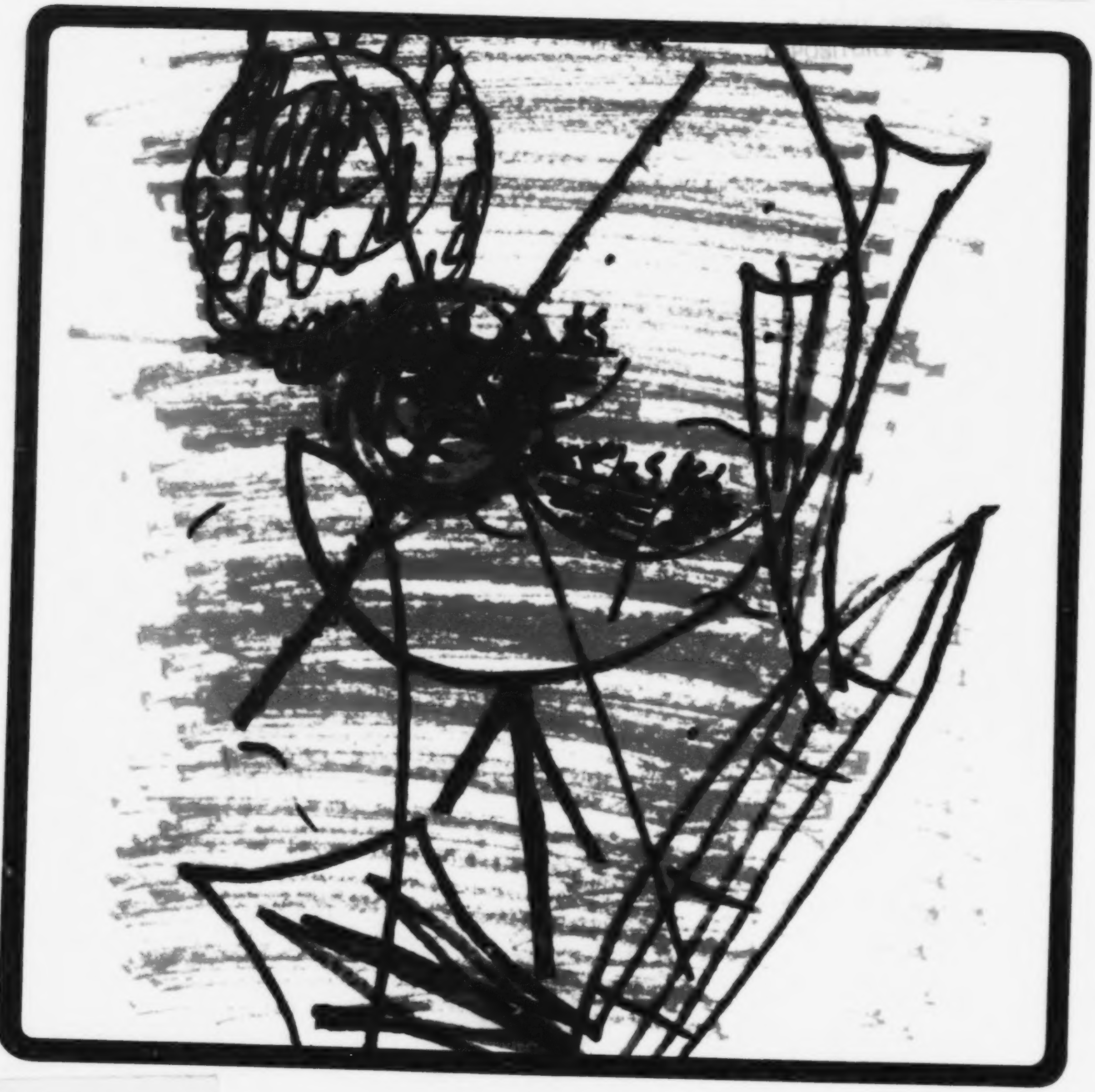

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About the Cover

The *Schizophrenia Bulletin* features art done by current and former mental patients. The drawing that appears on the cover of this issue was sent to us by Dr. Robert Paul Liberman, UCLA, Los Angeles, California. Although the artist wishes to remain anonymous, he has granted us permission to use his painting. The following background information was provided by his occupational therapist:

The individual who created the cover art participated in occupational therapy for a year during both inpatient and outpatient phases of his treatment. He refused to become involved in reality-oriented activities and focused his efforts exclusively on drawing. His conversation was limited to brief sentences, usually with bizarre content. He sat alone and worked on his drawings independently, often talking and laughing to himself. He became easily frustrated, but as time went on he appeared to gain self-confidence and his mood brightened. He took pleasure in his art and it served as the prime means of communication with others.

Thoughts on the Treatment of Schizophrenia

by William T. Carpenter, Jr.

At Issue

The At Issue section of the Schizophrenia Bulletin *contains viewpoints and arguments on controversial issues. Articles published in this section may not meet the strict editorial and scientific standards that are applied to major articles in the* Bulletin. *In addition, the viewpoints expressed in the following articles do not necessarily represent those of the staff or the Editorial Advisory Board of the* Bulletin.—The Editors.

Abstract

The author contends that the clinical relationship is the foundation for treating the person with schizophrenia. However, the models and scientific data available to the clinician have been limited by prevailing ideologies. Despite important contributions from interpersonal, pharmacologic, and rehabilitation treatment perspectives, each has curtailed the range of clinical observation and the capacity for integration of treatments. Even recent advances in clinical science methodology have contributed to a short fall in treatment knowledge. The author espouses a broad medical model for defining the range of data and integration of techniques relevant to therapeutics. He views phenomenology as the crucial concept for clinical exploration, and contrasts this approach with the shallowness of purely descriptive approaches on the one hand and the distortion imposed by theoretical presentiments of psychotherapy on the other. Continuity of care and integration of therapies can emerge from phenomenology in the context of the clinical relationship.

Clinical scientists too often find themselves so involved in methods, operations, and data interpretation that they fail to contribute adequately to the public discussion of perspective and clinical meaningfulness of the data they generate and interpret. On the other hand, clinicians often feel they cannot critically assess the scientific methods, technologies, and statistical assessments commonly employed in modern clinical studies, and thus fail to decipher the data from a clinical perspective. The "At Issue" section of the *Bulletin* provides the opportunity for me to wear both hats and express a personal view on issues relevant to the treatment of schizophrenia. The major premise of this view is that parochialism and scientific shortcomings in such areas as psychodynamically based interpersonal treatment strategies, psychopharmacotherapy, rehabilitation, and clinical trials research methodology have undermined the integration of what, in fact, are complementary treatment approaches and have thus sharply limited the informational base from which the therapeutics of schizophrenia ultimately evolve.

Interpersonal Therapeutics

Despite Freud's dictum that persons with schizophrenia could not form a transference neurosis and, therefore, were not suitable subjects for psychoanalytic inquiry (Freud 1953, 1957), the preeminence of psychodynamic psychiatry in North America at midcentury afforded the circumstances in which psychogenic theories were judged most promising and psychotherapeutic approaches were

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This is an abridged version of the lecture presented in response to the 21st Annual Strecker Award and is published with permission of Dr. Layton McCurdy, Director of the Institute of Pennsylvania Hospital, on behalf of the Strecker Award Selection Committee.

Reprint requests should be sent to Dr. W.T. Carpenter, Maryland Psychiatric Research Center, P.O. Box 21247, Baltimore, MD 21228.

presumed efficacious. Influential theories concerning familial interactions in the etiology of schizophrenia were espoused, and family therapy gained wide acceptance. These were the best of times for psychodynamically oriented clinicians, some of whom contributed substantially to our understanding of the phenomenology of schizophrenia and of the psychotherapeutic process with psychotic patients. For those interested in engaging in empirical work, research funding was readily available. Contributions from this area were embraced by both the academician and the practitioner. These favorable circumstances, however, did not prove to be an impetus to scientific development or to major advances in the clinical care of persons with schizophrenia. There were notable exceptions, of course, but the following principal consequences of this lengthy dominance in American psychiatry discouraged:

- the scientific evaluation of treatment efficacy;
- the development of interpersonal treatment strategies which did not derive from the predominant ideologic and theoretical position;
- the development of interpersonal strategies that could practically be delivered to the vast numbers of patients with schizophrenia;
- the implementation of phenomenologic investigation relatively free of theoretical presentiments;
- the integration of interpersonal therapeutic strategies with the burgeoning field of psychopharmacology; and
- a constructive collaboration between clinicians and families.

During the 1950s and 1960s, psychotherapeutic services for the schizophrenic patient were considered an ethical imperative, though this view contrasted with a reality in which significant psychotherapeutic services were available for only a few schizophrenic patients. In the mid-1980s, however, the majority of professionals seem to view psychotherapeutic intervention as irrelevant. Where an effect is noted, as many argue that it is dangerous and countertherapeutic as are prepared to argue that it is distinctly therapeutic. Even those who, by professional training and personal proclivity, are most prepared to deal intimately with the severely disturbed individual over time are unlikely to pursue such an opportunity or to be encouraged to do so by their colleagues.

This has resulted in a dearth of talented individuals capable of integrating interpersonal treatment strategies with pharmacologic treatment, or of providing the vantage of psychodynamic psychiatry in developing and interpreting clinical data germane to the care and study of persons with schizophrenia (Carpenter and Heinrichs 1980).

Pharmacotherapy

The introduction of antipsychotic drugs to the treatment of psychotic illness in the early 1950s has profoundly altered the theory and therapy of schizophrenia. Clinically effective psychoactive drugs have had an unprecedented impact on the study of brain physiology and have provided the impetus for reestablishing biology as a foundation of American psychiatry. The use of systematic descriptive methods and controlled study designs proved an efficient mechanism for demonstrating that neuroleptic drugs were capable of reducing the relapse rate in recovered or clinically stable patients and were causally associated with reduction in a broad range of psychopathologic manifestations in the acutely ill patient. Unfortunately, such results were greeted derisively in some quarters, with derogatory reference made to the virtual use of chemical lobotomies or straightjackets, the superficiality of symptom suppression, and the undermining of the patient's motivation to help himself.

Effective pharmacotherapeutics created a political, as well as a scientific, challenge to the psychodynamic leadership in American psychiatry. Unfortunately, the competition between therapies was dominated by oppositional views that were of marginal scientific validity. Given this ambiance, comparative treatment studies consistently found psychopharmacotherapy to be superior to psychotherapy (Schooler 1978; Gunderson 1979; Mosher and Keith 1980; Heinrichs and Carpenter 1981; Stanton et al. 1984). As a result, psychiatry has now become extensively reoriented, with the majority of young clinicians being trained in the use of therapeutically active drugs and espousing their role as crucial in psychiatric therapeutics. Not only has the psychodynamic sphere of influence lost preeminence, but the psychodynamic viewpoint is no longer adequately represented in the scientific academic community. The ill-conceived censure of the pharmacotherapeutics of schizophrenia has not proven valid and is now only of incidental interest. There are, however, new criticisms of a pharmacologic approach that must be addressed. Schizophrenia is manifested with considerable variation from one case to another, yet pharmacotherapeutics are used with remarkable uniformity. Inadequate attention has been given to the identification of neurolepticnonresponsive subgroups, subgroups responsive to alternative drugs, and subgroups with an unfavorable risk-benefit ratio.

The assumption that schizophrenia is a heterogeneous syndrome and that any discrete disease entity within the syndrome may have a multifactorial etiology provides a compelling rationale for seeking multiple treatment strategies and ascertaining their optimal integration. Clinical psychopharmacologists are only now beginning to recognize the necessity for implementing psychosocial approaches as a primary, rather than simply supportive and humanistic, attribute of clinical care (Schooler and Carpenter 1983; Mosher and Meltzer 1984).

The nature of schizophrenia varies within the individual according to the phase of illness. Some patients may demonstrate recovery or extensive stability after an initial episode and others, despite repeated episodes during young adulthood, may become clinically stable at a later period in life. The prevailing view calling for continuous drug treatment in all patients for maintenance and prophylactic purposes is not compatible with the nature of the disease as seen from the vantage of long-term studies (Bleuler 1972; Ciompi 1980). The field must develop methods for more highly individualized pharmacotherapeutic strategies (Davis et al. 1980; Carpenter and Heinrichs 1981; Tamminga and Carpenter 1982).

It is now evident that tardive dyskinesia occurs with alarming frequency with neuroleptic drugs. It is also possible that drugs with such varied effects on brain physiology will prove detrimental in the long term in other areas of functioning. In fact, in primates and in nonpsychotic human subjects, neuroleptic drugs induce behavioral changes which mimic many aspects of the deficit syndrome (Carpenter, Heinrichs, and Alphs 1985). It is, therefore, incumbent upon the field to develop treatment strategies which reduce the exposure of schizophrenic patients to neuroleptic drugs to an extent compatible with effective treatment of psychosis. Furthermore, the long-term effect of these drugs on core features, often referred to as negative or deficit symptoms (Strauss, Carpenter, and Bartko 1974; Andreasen and Olsen 1982; Heinrichs, Hanlon, and Carpenter 1984; Andreasen 1985; Bilder et al. 1985; Carpenter, Heinrichs, and Alphs 1985; Carpenter, Heinrichs, and Wagman 1985; Cornblatt 1985; Crow 1985; Gibbons et al. 1985; Goldberg 1985; Lewine 1985; Mayer et al. 1985; Pogue-Geile and Harrow 1985; Sommers 1985; Strauss 1985; Zubin 1985) (e.g., amotivation, anhedonia, restricted ideation, and affect), must be ascertained to determine whether there is a negative or beneficial interaction between neuroleptic drugs and personality integrity over extended periods of time.

Rehabilitation

In the long-term treatment of individuals with schizophrenia, psychiatry has relied too extensively on hospital care and asylum. We were ill-prepared for deinstitutionalization and have not provided effective leadership in the comprehensive clinical care of noninstitutionalized chronic patients (Bachrach 1983).

The void is being partly filled by social rehabilitation workers who have developed a number of models for providing work, shelter, and life support for the chronically ill (Anthony, Cohen, and Cohen 1983). While these contributions are to be applauded, they are too often associated with rejection of the medical model and a failure to solicit, or even permit, integration of medical management with psychosocial care. No doubt, this is partly the result of rarely finding the medical community available, responsive, or informed in these matters, but it is nourished by an unfortunate suspiciousness, competition for funds and influence, and rejection of the tenets of the medical model.

Research Methodology

Psychiatry has experienced an extensive development of clinical research methods over the past two decades. These methods have enhanced our ability to communicate concerning diagnosis, to distinguish between association and cause, and to obtain empiric evidence to support and refute theories rather than rely on prevailing views and personal authority. Research developments have also enabled psychiatry to contribute substantially to the study of the brain and to the integration of biologic data with clinical observation. However, there have been some unfavorable undertones.

The hypothesis-testing demand of isolating and manipulating a variable to demonstrate the existence of functional relationships often results in designs that reduce observed phenomena so extensively that clinical reality is no longer represented. The exigencies of research prompt attention to treatments that can be highly specified, delivered in short time, and easily measured with respect to outcome. The field thus shifted away from the study of complex and subtle psychopathology and toward psychopathologic attributes that can be assessed with minimal clinical involvement. Core features of illness are ignored if inconvenient (Carpenter 1980; Carpenter, Heinrichs, and Hanlon 1981), and data analysis and interpretation too often are based on computing capability and the availability of statistical programs, rather than on the utilization of statistical approaches that relate to clinical concepts and meaningfulness (Glantz 1981; Carpenter, Bartko, and St. Claire Evans 1983). As a result, little of this research is germane to the task of integrating seemingly diverse treatments in a manner appropriate to an individual patient's psychopathology, personality, and circumstances. These criticisms do not belie the impressive progress made in clinical science. Nor are they shortcomings of scientific methods per se, but rather an unnecessary side effect. Such practices persevere, in part, because clinical psychiatry has not been sufficiently critical of its research products (Strauss and Hafez 1981).

The Integration of Treatment Strategies

Integrating treatments in schizophrenia requires a conceptual approach which can define the range of data pertinent to understanding the schizophrenia syndrome and treating the individual. Although the medical model has no competitor capable of dealing with the full range of data already demonstrated to be relevant to schizophrenia, the biomedical version of the model is too narrow. Engel (1977) has pointed out that the biomedical model is preeminent in Western medicine, but its scientific validity is undermined by reductionism (attempting to account for illness and treatment in solely physico-chemical terms) and exclusiveness (considering factors not reducible to physico-chemical explanation as not relevant or trivial). The introduction of general systems theory to the study of biological organisms (Von Bertalanffy 1952, 1967) has clarified the intricate connectedness between the various levels of functioning of the human organism (Grinker 1975; Engel 1977; Marmor 1983). The resulting biopsychosocial medical model (Engel 1977, 1980) provides a model for the hierarchical organization of the individual which is heuristically valuable in the comprehensive study of health and disease. The importance of factors drawn from the biological, psychological, and sociological levels of abstraction is illustrated by the substantial contribution of behavioral variables to mortality (Hamburg 1982) and in the impossibility of accounting for a mere blush without considering social, psychological, and biological factors simultaneously.

A developmental, interactive medical model seems optimal for the study and treatment of persons with schizophrenia (Strauss and Carpenter 1981; Carpenter 1983). In this model, attention is called to etiologic factors and their interaction with environmental factors during development to determine vulnerability. The manifestation of psychotic illness in the vulnerable person may be determined by innate factors and temporal stresses. Once psychosis is manifest, various biological, psychological, and social factors determine the course and eventual outcome of the illness.

The causes of schizophrenia(s) remain an enigma, but present data on risk factors suggest contributions to etiology from genetics, gestational and birth complications, and an unknown factor associated with winter birth (Neale and Oltmanns 1980; Pulver, Sawyer, and Childs 1981; Pulver et al. 1983; Boyd, Pulver, and Stewart 1986). The role of developmental factors in shaping the degree of vulnerability in the etiologically predisposed individual has received considerable theoretical attention, but specific processes involved in pathogenesis have not yet been scientifically confirmed.

Some putative factors, such as the schizophrenogenic mother (Fromm-Reichmann 1948), were based solely on ideologically based retrospective study to which little credence can be granted. Other factors, such as parental communication deviance (Wynne and Singer 1963; Wynne et al. 1975), are firmly established in association with some forms of schizophrenia. But, it is not yet clear whether the intrafamilial communication pattern has a causative role, is reflective of the presence of a schizophrenic offspring, or is the result of parent and child sharing some third factor. These are not mutually exclusive possibilities. For example, communication deviancy could be a heritable trait linked with schizophrenia. A parent and the ill offspring may share the genetic trait—and thus the association. Furthermore, communication deviance in the home may increase vulnerability to psychosis in the etiologically prone offspring. Finally, the presence of an offspring with schizophrenia may increase the communication deviance of the parent. In any such circumstance, a disease model integrating genetic, cognitive, and social interactive variables over time is required. Those interested in the pathogenesis of schizophrenia and in prevention will surely scrutinize the many interpersonal and biologic factors that influence development (Mednick and McNeil 1968; Baumrind 1975; Reiss and Wyatt 1975; Hirsch 1981).

In addition to etiological and developmental considerations, another set of more proximal variables must be considered. Application of the stress-diathesis model (Rosenthal et al. 1974; Spring and Zubin 1977; Cancro 1980; Zubin 1981; Nuechterlein and Dawson 1984) is appealing for the clinical task faced with schizophrenia, for it is useful to conceptualize vulnerability to an illness where the timing and nature of manifestations are importantly determined by temporal factors described under the rubric of stress. This gives patient and clinician an opportunity to examine meaningful relationships between illness and the person's experience without postulating etiologic significance and provides a basis for an analysis of interpersonal dynamics which are not implicated with respect to etiology.

When we speak of the manifestation of schizophrenia, we refer to a variety of psychopathologic attributes that are present in varying degrees and mixtures in different patients. We decline to make the diagnosis of schizophrenia in the absence of positive symptom manifestations, which include hallucinations, delusions, and conspicuous disturbances in the content and flow of thought. We also observe a range of deficit or negative symptoms in which the diminution or absence of ordinary functioning is observed (Strauss, Carpenter, and Bartko 1974). These features represent the personality impairment and deterioration so devastating in the long term. It is thus useful to consider the manifestations of illness in a social context since diminished engagement in the social sphere is one of the devastating and disheartening aspects of schizophrenia.

At the turn of the century, Kraepelin (1971) described the manifestations of dementia praecox as indicative of the dissolution of the very essence of the person. Although the nature of this most devastating disease was clearly described so many years ago, our knowledge of cause, prevention, and cure remains rudimentary.

Personal Experience

In followup studies of a quasi-representative cohort drawn from various treatment facilities (Carpenter, Strauss, and Bartko 1973; World Health Organization 1974; Strauss and Carpenter 1977; Pulver and Carpenter 1983), I noted the uniformity of treatment with antipsychotic drugs despite tremendous differences in illness manifestations and course. I also noted that the majority of patients had little opportunity for treatment beyond drugs and brief visits with various clinicians. They often did not know their doctors' names and were uncertain with whom they would talk if they kept their clinic appointments. The patients with most favorable outcomes often seemed to make their adjustment "on their own." Also, many of the patients with very poor outcome eschewed the advice of doctors, failed to comply with long-term drug treatment, and had no involvement with professionals who might guide, encourage, or otherwise support realistic adjustment.

On an inpatient program at the National Institutes of Health Clinical Center designed for the care and study of schizophrenic patients in the early phases of their illness, I had the opportunity to become intimately familiar with the experiences of acutely psychotic patients during periods off medication (McGlashan, Levy, and Carpenter 1975; Sacks, Carpenter, and Richmond 1975; Wadeson and Carpenter 1976; Carpenter, McGlashan, and Strauss 1977). Therapeutics were based on a number of interpersonal techniques as well as pharmacotherapy in selected cases. I was impressed by the strength and resiliency of patients facing the most vexing and devastating mental alterations. The fact that many patients did well during hospital care and at 1-year followup attests to their favorable prognostic status, and it was always difficult to ascribe change to decisive therapeutic interventions. These patients varied considerably in which elements of treatment they found useful and meaningful. Any therapeutic intervention welcomed and used by some patients was experienced as noxious and counterproductive by others. Two lessons were evident. First, treatment approaches need to consider the qualities of each patient, and sweeping generalizations based on diagnostic class cannot be implemented successfully without this individualized context. Second, many attributes of interpersonal care are found meaningful and helpful by patients having psychotic experiences.

Treatment Relevant Subgroups. For patients with a diagnosis of schizophrenia, it is useful to determine their similarity to certain treatment relevant subgroups. Further research findings in this regard will emerge, but we can now identify subgroups with distinguishing features or patterns of illness. Among these are groups associated with neurologic impairment, computed tomography scan evidence of altered brain structure, poor premorbid development, and chronic functional deficits. Such patients may be distinguished from those with neurologically intact, better prognostic, and more episodic types of illness (Crow 1985). For the former subgroups, the therapeutic benefits of neuroleptic drugs may be weaker and the risks greater than in the latter. Similarly, patients may be categorized according to environmental circumstances (Carpenter and Heinrichs 1981; Leff, Kuipers, and Berkowitz 1983; McFarlane and Beels 1983). Those living in highly challenging, arousing circumstances require entirely different interpersonal strategies than those found in isolated, understimulating, low expectation environments. The administration of prophylactic antipsychotic drugs may be crucial in the former and deleterious in the latter. A manner of defining treatment relevant subgroups on the basis of clinical data alone is described elsewhere (Carpenter and Heinrichs 1981).

The fact that this effect is not robust on psychopathological attributes most likely to serve as a basis for judging treatment efficacy challenges psychiatry to better define the relevance of perspective, understanding, support, and placing the when and how—but not the why—of illness experience in a comprehensible personal framework. The following views on the role of diagnosis, treatment relevant subtypes, the individualization of treatment, pharmacotherapy, and interpersonal intervention are based on personal experience and a general synthesis of the literature.

Diagnosis. The introduction of psychoactive drugs with differential effects on various aspects of psychopathology made diagnosis of practical importance to therapeutics. It is not important because psychotherapeutic drugs are disease-class specific, for none are, but because purpose and choice of drug varies with classification. Both the diagnostic class and process are critical to therapeutics. Diagnostic class provides the first approximation of treatment requirements, and diagnostic process should be richly informative concerning illness manifestations, the personal strengths and weaknesses, and the circumstances of illness. A comprehensive diagnostic evaluation that includes a sufficient period of observation to become thoroughly informed as to the inner world of the patient is an indispensable starting point.

In addition to recognition of any definable illness, a careful differential diagnosis of functional psychoses is critical. The diagnosis of schizophrenia is reserved for patients with a psychotic experience which is not due to any known medical condition or to the effects of drug or toxic substance. *DSM-III* (American Psychiatric Association 1980) provides a number of alternative categories for the schizophrenia-like psychoses. These are not putative disease entities with their own set of therapeutic considerations, but they serve the useful purpose of withholding the designation of schizophrenia, which is associated with presumptions about therapy and course. Errors in the diagnosis of schizophrenia are not likely to be clinically damaging in early stages, since treatment considerations for alternative diagnostic classes may be essentially the same as for the acute phase of schizophrenia. From that point on, the patient with chronic or recurring forms of schizophrenia will provide sufficient clinical manifestations to revise the diagnosis to one of schizophrenia. With currently prevailing attitudes regarding the treatment of schizophrenia, it may be advantageous for early cases of schizophrenia to be treated as though they were an uncertain form of psychosis for which individualized, practical treatment decisions are more likely to be made. It is a more serious mistake to diagnose schizophrenia in cases with affective psychoses and organic psychosyndromes, for which treatment considerations are quite different.

More explicit guidelines for establishing treatment relevant subgroups of schizophrenia will undoubtedly be developed. Assessments of social environment, electrophysiology, psychophysiology, cognitive performance, clinical dimensions, course and drug response, biochemical markers, brain imaging, and personality will be of relevance to tomorrow's treatment planning.

Individualized Treatment Decisions. While characteristics of diagnostic and treatment relevant subgroups define the range of therapeutic considerations, the actual selection and implementation of treatment in any given case must depend on the specific evaluations of the individual patient and his or her circumstances. It is not only the nature of psychopathology but also the environmental circumstances and personality strengths and weaknesses of the patient that guide the clinician in selecting treatments. Such individualized decision-making requires a strong and continuous clinical relationship in which the clinician must be intimately informed about the patient's inner world, reactivity to various environmental circumstances, and responsiveness to various therapeutic maneuvers. The patient's ability and willingness to collaborate in the various treatment procedures is influenced by the bond that forms in this relationship.

The doctor-patient relationship affords the opportunity for phenomenologic evaluation (Jaspers 1963; Carpenter and Heinrichs 1980; Carpenter and Hanlon 1986) in which the experiences, perceptions, and behaviors emanating from illness and from adaptive response to the illness can become known to the clinician. It is crucial to engage in this process relatively free of theoretical presentiment, if the uniqueness of each patient is to be fully appreciated. The resulting longitudinal framework thus yields a data base on which treatment decisions can be validly based, regardless of modality (Carpenter and Heinrichs 1980; Strauss and Carpenter 1981).

Pharmacotherapy. It is surprising that the pharmacotherapy of schizophrenic patients is so focused on neuroleptic drugs that a full exploration of the therapeutic potential of other types of drugs is rarely undertaken. In treating such an incapacitating illness, some risk-taking concerning pharmacotherapeutics is warranted to identify those individuals who would be responsive to nonneuroleptic drugs. Also, because of the grave consequences of the long-term use of neuroleptic drugs in many patients, the assumption of some risk in seeking treatment strategies that minimize exposure to these drugs is justified.

No low-risk treatment is presently available for schizophrenia, and clinical experimentation which deviates from the standard practice of administering neuroleptics does not shift from low to high risk, but rather alters the profile of risks and benefits. The treatment of schizophrenia is now freeing itself from the Procrustean bed where uniformity reigned and individual experimentation was curbed by legal, administrative, professional, and consumer pressures.

Although we do not yet have guidelines for selecting schizophrenic patients who will prove responsive to other classes of psychoactive drugs, there is ample descriptive literature and some empirical documentation of responsiveness to lithium and antiseizure medication. Also, dysphoric affect in the schizophrenic patient may at times be alleviated by anxiolytic or antidepressant medication, and hypnotic drugs may be effective for insomnia. This entire range of clinical manifestations is usually treated with antipsychotic drugs alone even when not part of a psychotic episode. Painful anxiety, in the absence of psychotic deterioration, suggests a role for antianxiety drugs. Significant depression, in the absence of florid psychotic symptoms, may be sufficient indication for a trial with antidepressant drugs. Patients with a history of seizure disorder, inexplicable and stereotyped behavioral outbursts, head trauma, pathologic intoxication, and microamnestic eipsodes may be candidates for a trial with antiseizure medication (Monroe 1970, 1982).

Inasmuch as they are faced with a likely prospect of lifelong illness and reliance on antipsychotic drugs, even patients without clear indication of potential responsivity could be administered other types of drugs and followed on an empirical trial basis. Responsiveness to lithium is a case in point. It would seem evident that the greater the similarity with the affective disorders, the better the indication for a trial with lithium. The clinical trial literature does identify a subgroup of lithium-responsive schizophrenics, but does not confirm the expected delineation of this subgroup. Schizophrenic patients without characteristics similar to the affective disorders appear as likely to be lithium responders as those with affective features. Donaldson, Gelenberg, and Baldessarini (1983) provide an excellent literature review and discussion of nonneuroleptic pharmacotherapy in schizophrenia. The unfortunate fact that most patients with chronic schizophrenia will not be robustly responsive to these drugs is no basis for failing to find the minority who will be responsive.

New strategies with neuroleptic drugs are warranted (Schooler 1983; Mosher and Meltzer 1984) in an effort to reduce the incidence of tardive dyskinesia and minimize other long-term adverse drug effects. Efforts to reduce neuroleptic drug exposure with low dose or noncontinuous treatment are yielding favorable preliminary results in controlled studies (Carpenter and Heinrichs 1983; Donaldson, Gelenberg, and Baldessarini 1983; Falloon and Liberman 1983; Friedhoff 1983; Kane 1983; Schooler and Carpenter 1983).

Investigations concerned with pharmacologic intervention during the prodromal phase of relapse to determine if patients can remain off medication for substantial periods and still gain prophylactic benefits from an early intervention strategy require a clinical program that integrates psychosocial approaches, phenomenology, and psychopharmacotherapy. The low-dose continuous drug approach is another neuroleptic drug reduction strategy proving feasible for clinical application. It appears that dosages in the range of 10–20 percent of prevailing levels are almost as effective as these levels at reducing relapse rates, and may be associated with enhanced cognitive function and social adjustment. The psychosocial aspect of therapeutics for both the intermittent and continuous drug administration strategies emphasizes continuity of care, education of patient and relatives, clarification of the prodromal phenomenon, crisis management, environmental support, and other practical considerations. Finally, although transitory negative symptoms respond to neuroleptic treatment of acute psychoses, almost unexplored is the pharmacotherapy of long-term negative or deficit symptoms (Carpenter, Heinrichs, and Alphs 1985).

Interpersonal Strategies. Evidence to date has not established the therapeutic efficacy of exploratory, insight-oriented psychotherapy in schizophrenia. Comparative studies have failed to demonstrate that this form of treatment is superior to less theoretical, more supportive interpersonal strategies or to pharmacologic treatments (Gunderson 1979; Mosher and Keith 1980; Heinrichs and Carpenter 1981; Stanton et al. 1984), and May's (1968) seminal contribution has withstood challenge despite methodologic problems. Empirical data supporting the efficacy of more practically derived family and individual techniques are encouraging (Paul, Tobias, and Holly 1972; Falloon et al. 1982) and provide a beginning for the examination of the integration of interpersonal and pharmacologic treatment strategies (Stein, Test, and Marx 1975; Heinrichs and Carpenter 1982, 1983; Anderson 1983; Falloon and Liberman 1983).

A well-designed study of the efficacy of intensive psychotherapy in schizophrenia requires enormous effort. The equivalent of a placebo control is difficult to construct. Alternative interpersonal treatments may be usefully compared, but this addresses the secondary question of comparative efficacy rather than the primary question of efficacy per se. The goals of psychoanalytically derived therapy, however, can be clearly articulated and reliably assessed (McGlashan and Miller 1982), and the operations of such treatment for persons with schizophrenia have been recently reviewed (McGlashan 1983).

The recently completed psychotherapy of schizophrenia project (Gunderson et al. 1984; Stanton et al. 1984) illustrates the feasibility of conducting an arduous experiment, and found that exploratory psychotherapy was not superior (as hypothesized) to supportive psychotherapy. However, the primary psychotherapy efficacy issue still remains to be directly assessed (Carpenter 1984).

In clinical practice, it is time to return to a basic empirical approach. The irreducible essence of our interest in schizophrenia is the nature of another person's experience. It is in the subjective and inner world of volition, perception, cognition, and affect that schizophrenia is manifest. Empathy is crucial in discovering the subjective life of another person, and to engage in this process with the psychotic person requires skill, intuition, training, and perseverance. Jaspers recognized that knowledge of this inner world was central to diagnosis, prognosis, and treatment, and he borrowed phenomenology from Husserl's philosophy and introduced it into medicine as a scientific method (Jaspers 1963). Phenomenology defines the goals and methods used by the clinician to generate the observations upon which clinical decisions are based. When this base is weakened by narrowly defining psychopathologic attributes, or by distorted observation with premature inference and theoretically determined assumption, our view is truncated and the science of clinical care is compromised.

The Clinical Relationship

The cornerstone of treatment of the schizophrenic patient is the clinical relationship. This relationship needs personal continuity over time, not simply because this is a more sensitive delivery of care, but because the patient with schizophrenia is so likely to have difficulty establishing relationships, maintaining trust, and taking initiative. The type of relationship envisioned need not be and, for practical matters, cannot always be with a physician. I would stress, however, the importance of the primary clinician being familiar with the full range of potential treatments ranging from psychosocial rehabilitation through family and dyadic treatment strategies to pharmacologic considerations.

The initial task in the clinical relationship is phenomenologic, and does not require a theoretical or therapeutic orientation. A focused psychotherapeutic stance is thus based on observed difficulties and the discernment of potentially attainable goals. Psychotherapeutic work need not be exclusive, nor should it compete with other aspects of treatment. The reworking of personality structure no longer seems a suitable or practical goal in the treatment of most schizophrenic patients. Psychotherapeutic tasks are more promising when based on short term, highly individualistic goals, such as reducing stressful, idiosyncratic reactions to environmental events or developing insight into warning signs of impending relapse.

As part of his work, the clinician has important educational responsibilities to the patient and other household members. The patient and family may be so burdened with misconception and misinformation that an educational effort is reassuring and illuminating. To be optimally effective, such education should be personalized so that it takes on real meaning to patients and their families. Dealt with in abstraction, the information may prove difficult to incorporate and use. Furthermore, each case has specific considerations which may not be addressed in a more general educational presentation.

Present therapeutic work that involves the family has made an important shift from an etiologically oriented—often experienced as blaming—approach to one that emphasizes collaboration with those most extensively involved in caring for the patient. The importance, for example, of stress reduction and stress coping strategies in general in the management of an individual with a stress-sensitive illness is more readily understood in a benign context where collaboration is established.

Many other issues having a bearing on treatment emerge in this clinical relationship. We can, for example, counsel on the genetics of schizophrenia, but only after developing a view of the particular concerns and assumptions of each patient and family (Tsuang 1978; Kessler 1980). Also, recognition of the role of violent fantasy and primitive impulse can enable the clinician to monitor dangerousness. When the primary clinician requires the involvement of other clinicians to administer specific treatments, the medical model provides a basis for integrating the professional team. It is crucial that the patient deal with new treatments—e.g., change in drug status—with a familiar clinician. There are scores of such crucial components which can only be illuminated and responded to in the context of an ongoing clinical relationship. The establishment of a health care system that interjects a different person and place for each therapeutic shift—e.g., "Go to the emergency room and tell the doctor the voices are coming back," was designed without regard to the nature of schizophrenic illness.

The clinician's role is a challenging one. A broad knowledge of psychopathology, interpersonal skills, the medical model, and diverse treatment techniques is required. The work is frustrating, for cause and cure remain elusive. The patients are often fascinating, often fatiguing, but their courage is remarkable and their need compelling. We are presently in a position to move knowledge forward by relying more extensively on what is observable and verifiable, and by integrating clinical observation with the rapid advances in technology and science now occurring in the life sciences. We must resist the temptation of oversimplifying schizophrenia. The presentation of a medical model scientifically valid for the care and study of persons with schizophrenia may also serve as a standard for other fields of medicine where clinical observation is too narrowly defined, where biologic reductionism belies the complexity of human organization, and mindfulness of the human dimension is no longer considered essential to the role of physician (Schwartz and Wiggins, in press).

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

Anderson, C.M. A psychoeducational program for families of patients with schizophrenia. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 99–116.

Andreasen, N.C. Positive vs. negative schizophrenia: A critical evaluation. *Schizophrenia Bulletin*, 11:380–389, 1985.

Andreasen, N.C., and Olsen, S. Negative *v.* positive schizophrenia. *Archives of General Psychiatry*, 39:789–794, 1982.

Anthony, W.A.; Cohen, M.R.; and Cohen, B.F. Philosophy, treatment process, and principles of the psychiatric rehabilitation approach. In: Bachrach, L., ed. *Deinstitutionalization*. San Francisco: Jossey-Bass, 1983. pp. 67–79.

Bachrach, L.L., ed. *Deinstitutionalization*. San Francisco: Jossey-Bass, 1983.

Baumrind, D. The contributions of the family to the development of competence in children. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 14):12–37, 1975.

Bilder, R.M.; Mukherjee, S.; Rieder, R.O.; and Pandurangi, A.K. Symptomatic and neuropsychological components of defect states. *Schizophrenia Bulletin*, 11:409–419, 1985.

Bleuler, M. *The Schizophrenic Disorders: Long-term Patient and Family Studies*. Translated by Siegfried M. Clemens. New Haven and London: Yale University Press, 1972.

Boyd, J.H.; Pulver, A.E.; and Stewart, W. Season of birth: Schizophrenia and bipolar disorder. *Schizophrenia Bulletin*, 12:173–186, 1986.

Cancro, R. Overview of schizophrenia. In: Kaplan, H.A.; Freedman, A.M.; and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry*. Vol. II. 3rd ed. Baltimore, MD: Williams and Wilkins, 1980. pp. 1093–1104.

Carpenter, W.T., Jr. Clinical research methods applicable to the study of treatment effects in chronic schizophrenic patients. In: Baxter, D.F., and Melnechuk, T., eds.

Perspectives in Schizophrenia Research: Presentations and Sessions of the V.A. Advisory Conference on Chronic Schizophrenia, Harpers Ferry, W. Va., 1979. New York: Raven Press, Inc., 1980. pp. 297-311.

Carpenter, W.T., Jr. What is schizophrenia? *Schizophrenia Bulletin*, 9:9-10, 1983.

Carpenter, W.T., Jr. A perspective on the psychotherapy ot schizophrenia project. *Schizophrenia Bulletin*, 10:599-603, 1984.

Carpenter, W.T., Jr.; Bartko, J.J.; and St. Claire Evans, M. Multivariate statistics and meaning in the study of psychopathology: Focus on a specific problem. *Schizophrenia Bulletin*, 9:1-5, 1983.

Carpenter, W.T., Jr., and Hanlon, T.E. Clinical practice and the phenomenology of schizophrenia. In: Burrows, C.D., ed. *Handbook of Studies on Schizophrenia.* Part I. Amsterdam: Elsevier, 1986. pp. 123-130.

Carpenter, W.T., Jr., and Heinrichs, D.W. The role for psychodynamic psychiatry in the treatment of schizophrenic patients. In: Strauss, J.S.; Fleck, S.; Bowers, M.; Levine, I.; Downey, T.W.; and Jackson, S., eds. *Psychotherapy of Schizophrenia: Current Status and New Directions.* New York: Plenum Press, Inc., 1980. pp. 239-255.

Carpenter, W.T., Jr., and Heinrichs, D.W. Treatment relevant subtypes of schizophrenia. *Journal of Nervous & Mental Disease*, 169:113-119, 1981.

Carpenter, W.T., Jr., and Heinrichs, D.W. Early intervention, time-limited, targeted pharmacotherapy of schizophrenia. *Schizophrenia Bulletin*, 9:533-542, 1983.

Carpenter, W.T., Jr.; Heinrichs, D.W.; and Alphs, L. Treatment of negative symptoms. *Schizophrenia Bulletin*, 11:440-452, 1985.

Carpenter, W.T., Jr.; Heinrichs, D.W.; and Hanlon, T.E. Methodologic standards for treatment outcome research in schizophrenia. *American Journal of Psychiatry*, 138:465-471, 1981.

Carpenter, W.T.; Heinrichs, D.W.; and Wagman, A.M.I. On the heterogeneity of schizophrenia. In: Alpert, M., ed. *Controversies in Schizophrenia.* New York: Guilford Press, 1985. pp. 25-37.

Carpenter, W.T., Jr.; McGlashan, T.H.; and Strauss, J.S. The treatment of acute schizophrenia without drugs: An investigation of some current assumptions. *American Journal of Psychiatry*, 134:14-20, 1977.

Carpenter, W.T., Jr.; Strauss, J.S.; and Bartko, J.J. A flexible system for the indentification of schizophrenia: A Report from the International Pilot Study of Schizophrenia. *Science*, 182:1275-1278, 1973.

Ciompi, L. Three lectures on schizophrenia: The natural history of schizophrenia in the long term. *British Journal of Psychiatry*, 136:413-420, 1980.

Cornblatt, B.A.; Lenzenweger, M.F.; Dworkin, R.H.; and Erlenmeyer-Kimling, L. Positive and negative schizophrenic symptoms, attention, and information processing. *Schizophrenia Bulletin*, 11:397-408, 1985.

Crow, T.J. The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin*, 11:471-485, 1985.

Davis, J.M.; Schaffer, C.B.; Killian, G.A.; Kinard, C.; and Chan, C. Important issues in the drug treatment of schizophrenia. *Schizophrenia Bulletin*, 6:70-87, 1980.

Donaldson, S.R.; Gelenberg, A.J.; and Baldessarini, R.J. The pharmacologic treatment of schizophrenia: A progress report. *Schizophrenia Bulletin*, 9:504-527, 1983.

Engel, G.L. The need for a new medical model: A challenge for biomedicine. *Science*, 196.129-136, 1977.

Engel, G.L. The clinical application of the biopsychosocial model. *American Journal of Psychiatry*, 137:535-544, 1980.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Razni, J.; Moss, H.B.; and Gilderman, A.M. Family management in the prevention of exacerbations of schizophrenia: A controlled study. *New England Journal of Medicine*, 306:1437-1440, 1982.

Falloon, I.R.H., and Liberman, R.P. Interactions between drug and psychosocial therapy in schizophrenia. *Schizophrenia Bulletin*, 9:543-554, 1983.

Friedhoff, A.J. A strategy for developing novel drugs for the treatment of schizophrenia. *Schizophrenia Bulletin*, 9:555-562, 1983.

Freud, S. *Neuropsychoses of Defense.* London: Hogarth Press, 1953.

Freud, S. *On Narcissism.* London: Hogarth Press, 1957.

Fromm-Reichmann, F. Notes on the development of treatment of schizophrenia by psychoanalytic psychotherapy. *Psychiatry*, 11:263-273, 1948.

Gibbons, R.D.; Lewine, R.R.J.; Davis, J.M.; Schooler, N.R.; and Cole, J.O. An empirical test of a Kraepelinian vs. a Bleulerian view of negative symptoms. *Schizophrenia Bulletin*, 11:390-396, 1985.

Glantz, S.A. *Primer of Biostatistics.* New York: McGraw-Hill, 1981.

Goldberg, S.C. Negative and deficit symptoms in schizophrenia do respond to neuroleptics. *Schizophrenia Bulletin*, 11:453–456, 1985.

Grinker, R.R., Sr. The relevance of general systems theory to psychiatry. In: Arieti, S., ed. *American Handbook of Psychiatry*. New York: Basic Books, 1975. pp. 251–274.

Gunderson, J.G. Individual psychotherapy. In: Bellak, L., ed. *Disorders of the Schizophrenic Syndrome*. New York: Basic Books, 1979. pp. 364–398.

Gunderson, J.G.; Frank, A.F.; Katz, H.M.; Vannicelli, M.L.; Frosch, J.P.; and Knapp, P.H. Effects of psychotherapy in schizophrenia: II. Comparative outcome of two forms of treatment. *Schizophrenia Bulletin*, 10:564–598, 1984.

Hamburg, D.A. Health and behavior. *Science*, 217:399, 1982.

Heinrichs, D.W., and Carpenter, W.T., Jr. The efficacy of individual psychotherapy: A perspective and review emphasizing controlled outcome studies. In: Arieti, S., and Brodie, H.K., eds. *The American Handbook of Psychiatry*. Vol. VII. New York: Basic Books, Inc., 1981. pp. 586–613.

Heinrichs, D.W., and Carpenter, W.T., Jr. The psychotherapy of schizophrenic disorders. In: Grinspoon, L., ed. *Psychiatry 1982: The American Psychiatric Annual Review*. Washington, DC: American Psychiatric Press, 1982. pp. 154–166.

Heinrichs, D.W., and Carpenter, W.T., Jr. The coordination of family therapy with other treatment modalities for schizophrenia. In: McFarlane, W.R., and Beels, C.C., eds. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 267–287.

Heinrichs, D.W.; Hanlon, T.E.; and Carpenter, W.T., Jr. The Quality of Life Scale: An instrument for rating the schizophrenic deficit syndrome. *Schizophrenia Bulletin*, 10:388–398, 1984.

Hirsch, B.J. Coping and adaptation in high-risk populations: Toward an integrative model. *Schizophrenia Bulletin*, 7:164–172, 1981.

Jaspers, K. *General Psychopathology*. Translated by J. Hoenig and M.W. Hamilton. Manchester, England: Manchester University Press, 1963.

Kane, J.M. Low dose medication strategies in the maintenance treatment of schizophrenia. *Schizophrenia Bulletin*, 9:528–532, 1983.

Kessler, S. The genetics of schizophrenia: A review. *Schizophrenia Bulletin*, 6:404–416, 1980.

Kraepelin, E. *Dementia Praecox and Paraphrenia*. Translated by R.M. Barclay. Huntington, NY: R.E. Krieger Publishing, 1971.

Leff, J.P.; Kuipers, L.; and Berkowitz, R. Intervention in families of schizophrenics and its effect on relapse rate. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 173–187.

Lewine, R.R.J. Negative symptoms in schizophrenia: Editor's introduction. *Schizophrenia Bulletin*, 11:361–363, 1985.

Marmor, J. Systems thinking in psychiatry: Some theoretical and clinical implications. *American Journal of Psychiatry*, 140:833–838, 1983.

May, P.R.A. *Treatment of Schizophrenia: A Comparative Study of Five Treatment Methods*. New York: Science House, 1968.

Mayer, M.; Alpert, M.; Stastny, P.; Perlick, D.; and Empfield, M. Multiple contributions to clinical presentation of flat effect in schizophrenia. *Schizophrenia Bulletin*, 11:420–426, 1985.

McFarlane, W.R., and Beels, C.C. A decision-tree model for integrating family therapies for schizophrenia. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia*. New York: Guilford Press, 1983. pp. 325–335.

McGlashan, T.H.; Levy, S.T.; and Carpenter, W.T., Jr. Integration and sealing-over: Clinically distinct recovery styles from schizophrenia. *Archives of General Psychiatry*, 32:1269–1272, 1975.

McGlashan, T.H., and Miller, G.H. The goals of psychoanalysis and psychoanalytic psychotherapy. *Archives of General Psychiatry*, 39:377–388, 1982.

McGlashan, T.H. Intensive individual psychotherapy of schizophrenia: A review of techniques. *Archives of General Psychiatry*, 40:909–920, 1983.

Mednick, S.A., and McNeil, F.F. Current methodology in research on the etiology of schizophrenia: Serious difficulties which suggest the use of the high risk group method. *Psychological Bulletin*, 70:681–693, 1968.

Monroe, R.R. *Episodic Behavioral Disorders*. Cambridge: Harvard University Press, 1970.

Monroe, R.R. *DSM-III* style diagnoses of episodic disorders. *Journal of Nervous & Mental Disease*, 170:664–669, 1982.

Mosher, L.R., and Keith, S.J. Psychosocial treatment: Individual, group, family, and community support approaches. *Schizophrenia Bulletin*, 6:10–41, 1980.

Mosher, L.R., and Meltzer, H.Y. Drugs and psychosocial treatment. *Schizophrenia Bulletin*, 10:8–9, 1984.

Neale, J.M., and Oltmanns, T.F. *Schizophrenia*. New York: John Wiley & Sons, Inc., 1980.

Nuechterlein, K.H., and Dawson, M.E. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin*, 10:300-312, 1984.

Paul, G.L.; Tobias, L.T.; and Holly, B.L. Maintenance psychotropic drugs in the presence of active treatment programs. A triple blind withdrawal study with long-term mental patients. *Archives of General Psychiatry*, 27:106-115, 1972.

Pogue-Geile, M.F., and Harrow, M. Negative symptoms in schizophrenia: Their longitudinal course and prognostic importance. *Schizophrenia Bulletin*, 11:427-439, 1985.

Pulver, A.E., and Carpenter, W.T., Jr. Lifetime psychotic symptoms assessed with the diagnostic interview schedule. *Schizophrenia Bulletin*, 9:377-382, 1983.

Pulver, A.E.; Sawyer, J.; and Childs, B. The association between the risk for schizophrenia and season of birth. *American Journal of Epidemiology*, 114:735-749, 1981.

Pulver, A.E.; Stewart, W.; Carpenter, W.T., Jr.; and Childs, B. Risk factors in schizophrenia: Season of birth in Maryland, USA. *British Journal of Psychiatry*, 143:389-396, 1983.

Reiss, D., and Wyatt, R.J. Family and biologic variables in the same etiologic studies of schizophrenia: A proposal. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 14):64-81, 1975.

Rosenthal, D.; Goldberg, I.; Jacobsen, B.; Wender, P.H.; Kety, S.S.; Schulsinger, F.; and Eldred, C.A. Migration, heredity, and schizophrenia. *Psychiatry*, 37:521-539, 1974.

Sacks, M.H.; Carpenter, W.T., Jr.; and Richmond, M.B. Psychotherapy in hospitalized research patients. *Archives of General Psychiatry*, 32:581-585, 1975.

Schooler, N.R. Antipsychotic drugs and psychological treatment in schizophrenia. In: Lipton, M.A.; Dimascio, A.; and Killam, K.R., eds. *Psychopharmacology: A Generation of Progress*. New York: Raven Press, 1978. pp. 1155-1168.

Schooler, N.R., and Carpenter, W.T., Jr. New drug treatment strategies in schizophrenia. *Schizophrenia Bulletin*, 9:500-503, 1983.

Schwartz, M., and Wiggins, O. Medical science and humanism: A phenomenological view. *Perspectives of Biology and Medicine*, in press.

Sommers, A.A. "Negative symptoms": Conceptual and methodological problems. *Schizophrenia Bulletin*, 11:364-379, 1985.

Spring, B., and Zubin, J. Vulnerability to schizophrenic episodes and their prevention in adults. In: Albee, G.W., and Joffe, J.M., eds. *Primary Prevention in Psychopathology: The Issues*. Vol. 1. Hanover, NH: University Press of New England, 1977. pp. 254-284.

Stanton, A.H.; Gunderson, J.G.; Knapp, P.H.; Frank, A.F.; Vannicelli, M.L.; Schnitzer, R.; and Rosenthal, R. Effects of psychotherapy in schizophrenia: I. Design and implementation of a controlled study. *Schizophrenia Bulletin*, 10:520-551, 1984.

Stein, M.A.; Test, M.A.; and Marx, A.J. Alternative to the hospital: A controlled study. *American Journal of Psychiatry*, 132:517-522, 1975.

Strauss, J.S. Negative symptoms: Future developments of the concept. *Schizophrenia Bulletin*, 11:456-460, 1985.

Strauss, J.S., and Carpenter, W.T., Jr. Prediction of outcome in schizophrenia: III. Five-year outcome and its predictors: A Report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry*, 34:159-163, 1977.

Strauss, J.S., and Carpenter, W.T., Jr. *Schizophrenia*. New York: Plenum Press, 1981.

Strauss, J.S.; Carpenter, W.T., Jr.; and Bartko, J.J. Toward an understanding of the symptom picture considered characteristic of schizophrenia: Its description, precursors and outcome. *Schizophrenia Bulletin*, 1 (Experimental Issue No. 11):61-69, 1974.

Strauss, J.S., and Hafez, H. Clinical questions and "real" research. *American Journal of Psychiatry*, 138:1592-1597, 1981.

Tamminga, C.A., and Carpenter, W.T., Jr. The *DSM-III* diagnosis of schizophrenic-like illness and the clinical pharmacology of psychosis. *Journal of Nervous & Mental Disease*, 170:744-751, 1982.

Tsuang, M.T. Genetic counselling for psychiatric patients and their families. *American Journal of Psychiatry*, 135:1465-1475, 1978.

Von Bertalanffy, L. *Problems of Life*. New York: John Wiley & Sons, Inc., 1952.

Von Bertalanffy, L. *Robots, Men and Minds*. New York: Braziller, 1967.

Wadeson, H., and Carpenter, W.T., Jr. Subjective experience of schizophrenia. *Schizophrenia Bulletin*, 2:302-316, 1976.

World Health Organization. *Report of the International Pilot Study of Schizophrenia*. Vol. 1. Geneva, Switzerland: WHO Press, 1974.

Wynne, L.C., and Singer, M. Thought disorder and family relations. *Archives of General Psychiatry,* 9:199-206, 1963.

Wynne, L.C.; Singer, M.T.; Bartko, J.J.; and Toohey, M.L. Schizophrenics and their families: Recent research on parental communication. In: Tanner, J.M., ed. *Psychiatric Research: The Widening Perspective.* New York: International Universities Press, 1975. pp. 254-286.

Zubin, J. How to break the logjam in schizophrenia. *Journal of Nervous & Mental Disease,* 169:477-492, 1981.

Zubin, J. Negative symptoms: Are they indigenous to schizophrenia? *Schizophrenia Bulletin,* 11:461-470, 1985.

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An Invitation to Readers

Providing a forum for a lively exchange of ideas ranks high among the *Schizophrenia Bulletin's* objectives. In the section **At Issue**, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. **At Issue** is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the *Bulletin* or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—*The Editors.*

Send your remarks to:

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Psychiatric Rehabilitation of Schizophrenia: Editor's Introduction

by Robert Paul Liberman

Abstract

Well-planned and conceptualized rehabilitation of psychiatric patients who demonstrate persisting impairments, disabilities, and handicaps begins with assessment of symptoms and psychosocial functioning. Rehabilitation interventions—aimed at building skills and providing supportive and prosthetic environments—are linked to initial and ongoing assessment. The refractoriness of large numbers of psychiatric patients to conventional and customary forms of therapy will fuel the development of psychiatric rehabilitation that is efficacious and empirically based.

With the growing appreciation of the chronicity of many major mental disorders and their refractoriness to psychotherapeutic and pharmacotherapeutic treatments, the field of psychiatric rehabilitation has come of age. For patients with persisting and disabling psychopathology, rehabilitation offers training in social and vocational skills, as well as prosthetic environments that are congruent with maximal feasible functioning and adaptation. As research using epidemiological, longitudinal, and reliable diagnostic and assessment instruments has flourished, it has become increasingly clear that rehabilitation is indicated for significant numbers of psychiatric patients.

Treatment outcome and naturalistic studies have documented that many individuals with affective, anxiety, and somatoform disorders have now joined ranks of persons with schizophrenia in suffering from persisting psychopathology and lingering social and occupational disabilities. For example, a substantial minority of depressed patients have been found to suffer from "double depression" where acute episodes of depression are superimposed upon unrelenting dysthymia. Approximately 25 percent of patients with major affective disorders do not respond to the intensive inpatient and outpatient treatments currently available; a like percentage of agoraphobic and obsessive-compulsive patients do not respond adequately to drugs or short-term behavior therapy.

But it is schizophrenia that poses the greatest challenge to the rehabilitation practitioner. The chronicity of this disorder is well known; however, the recidivism and failure of existing methods for promoting community tenure are gaining more attention and concern. In one county using a case register, persons with schizophrenia had two psychiatric hospitalizations per year, far greater than any other mental disorder save alcoholism (Kramer 1977). Increasing since 1979 have been the number of patients with long, continuous stays in mental hospitals (Craig et al. 1984). Even with reasonable state-of-the-art treatment, approximately 40 percent of patients with schizophrenia will relapse within 1 year and 75 percent within 5 years of discharge from inpatient care (Talbott 1981; Hogarty 1984).

A new subgroup refractory to customary treatments—young adult chronic patients—has emerged from the baby boom after World War II and reduction in number of long-term hospital beds. With the advent of civil libertarian codes regulating commitments to psychiatric hospitals, hundreds of thousands of young chronic patients under age 35 are served intermittently and inade-

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quately by community-based agencies. Often entangled with the law and substance abuse, these individuals desperately need longer-term strategies of rehabilitation that address their widespread deficits in social and living skills, and their atrophied social networks (Pepper 1985).

The revolving-door phenomenon of hospital admission-brief treatment-discharge-relapse-readmission points to the insufficiency of current methods of symptomatic treatment and aftercare for the chronically mentally ill. Moreover, the enduring incapacity, burden, and symptoms of chronic illness demoralize patients, relatives, and professionals—generating therapeutic nihilism and fruitless quests for causes and cures that are decades away. While we invest in research that may bring the "magic bullet" closer to reality, there are practical and proven methods of rehabilitation that can give optimism and hope to professionals and consumers alike.

Psychiatric rehabilitation, originally inspired by the enormous successes of physical rehabilitation, has now acquired its own conceptual base from the "vulnerability-stress-coping-competence" model of mental disorder. Advances in conceptualization are described in the articles by Anthony and Liberman and Spaulding et al. in the first two articles of this special issue. Any new applied science requires a data base, and reliable and valid methods of symptom and functional assessment—reviewed by Lukoff et al. and Wallace—provide just that for the field of rehabilitation.

The failures of deinstitutionalization and early efforts at community care of the chronically mentally ill have underlined the fact that changing the locus of care without innovations in the modus yields little of significance in long-term outcomes. But innovative techniques for rehabilitating the severely and chronically mentally ill are now available and have been empirically validated as shown in the brace of articles in this special issue by Liberman et al., Glynn and Mueser, and Strachan. Other methods of rehabilitation, not given major emphasis in this special issue, have been supported with empirical evidence of efficacy and offer the practitioner a spectrum of service models (Beard, Malamud, and Rossman 1978; Bachrach 1980; Bond et al. 1984; Stein and Test 1985). The final articles in this issue indicate the need and strategies for disseminating rehabilitation principles and techniques broadly to professionals and consumers. It would be a tragedy if the promising methods of rehabilitation now available in a few locales and centers are delayed in their dissemination and utilization for the next generation of teachers and students, professionals and patients. This special issue is a small step toward diffusing psychiatric rehabilitation throughout the mental health professions, agencies, and institutions.

References

Bachrach, L.L. Overview: Model programs for chronic mental patients. *American Journal of Psychiatry*, 137:1023–1031, 1980.

Beard, J.H.; Malamud, T.J.; and Rossman, E. Psychiatric rehabilitation and long-term rehospitalization rates: The findings of two research studies. *Schizophrenia Bulletin*, 4:622–635, 1978.

Bond, G.R.; Dincin, J.; Setze, P.J.; and Witheridge, T.F. The effectiveness of psychiatric rehabilitation: A summary of research at Thresholds. *Psychosocial Rehabilitation Journal*, 7:6–22, 1984.

Craig, T.J.; Goodman, A.B.; Siegel, C.; and Wanderling, J. The dynamics of hospitalization in a defined population during deinstitutionalization. *American Journal of Psychiatry*, 141:782–785, 1984.

Hogarty, G.E. Depot neuroleptics: The relevance of psychosocial factors. *Journal of Clinical Psychiatry*, 2:36–42, 1984.

Kramer, M. *Psychiatric Services and the Changing Institutional Scene, 1950–1985.* (DHEW Publication No. (ADM) 77-433) Washington, DC: Superintendent of Documents, Government Printing Office, 1977.

Pepper, B. The young adult chronic patient: Population overview. *Journal of Clinical Psychopharmacology*, 5:3S–7S, 1985.

Stein, L.I., and Test, M.A., eds. *The Training in Community Living Model: A Decade of Experience. New Directions for Mental Health Services.* No. 26. San Francisco: Jossey-Bass, 1985.

Talbott, J.A., ed. *Chronic Mental Patients: Treatment, Programs, Systems.* New York: Human Sciences Press, 1981.

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The Practice of Psychiatric Rehabilitation: Historical, Conceptual, and Research Base

by William A. Anthony and Robert Paul Liberman

Abstract

With the recognition that most psychiatric disorders are associated with severe and persisting disability and the development of effective procedures for improving the long-term outcome of patients, the term "psychiatric rehabilitation" is becoming routinely used in the mental health field. Psychiatric rehabilitation has begun to take its place as a viable, credible intervention approach, even infiltrating professionals' jargon and administrators' program descriptions. The field of psychiatric rehabilitation has progressed to the stage where its history can be traced; its conceptual base and treatment strategies described; its practice observed, monitored, and replicated; and its future growth anchored in a research foundation.

> How many times it thundered before Franklin took the hint! How many apples fell on Newton's head before he took the hint! Nature is always hinting at us. It hints over and over again. And suddenly we take the hint.—*Robert Frost*

The essential elements of a psychiatric rehabilitation approach have been hinted at for well over a century. Different elements of a psychiatric rehabilitation approach have periodically moved in and out of favor, highlighted almost serendipitously as the mental health field progressed through various developmental phases. Currently there is a consensus developing as to what constitutes the field of psychiatric rehabilitation. The overall goal of psychiatric rehabilitation is to assure that the person with a psychiatric disability can perform those physical, emotional, social, and intellectual skills needed to live, learn, and work in the community, with the least amount of support necessary from agents of the helping professions (Anthony 1979). The major methods by which this goal is accomplished involve either teaching persons the specific skills needed to function effectively or developing the community and environmental resources needed to support or strengthen their present levels of functioning (Anthony, Cohen, and Cohen 1983; Livneh 1984; Liberman and Evans 1985).

The work of current researchers and practitioners will determine whether psychiatric rehabilitation becomes an evolving field of study and practice, or merely a historical footnote. At present it seems that many mental health professionals recognize the need for rehabilitation interventions to complement existing treatment approaches (Anthony 1977; Liberman and Foy 1983). However, this recognition of need does not mean that psychiatric rehabilitation is a well-understood field of study. Because all types of mental health disciplines practice psychiatric rehabilitation, and because relevant research and conceptual articles appear in a wide range of professional journals, psychiatric rehabilitation is a field that until recently has been difficult to define.

Historical Context

The origins of the psychiatric rehabilitation field are rooted in several historical developments: (1) the moral therapy era; (2) the inclusion of the psychiatrically disabled into

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public-supported vocational rehabilitation programs; (3) the development of community mental health ideology; (4) the psychosocial rehabilitation center movement; and (5) the development of skills training techniques as an effective mental health intervention.

Moral Therapy Era. The 19th century reformists for more humane care of the mentally ill aimed "to treat the patients as far as their condition would possibly admit, as if they were still in the enjoyment of the healthy exercise of their mental faculties . . . and to make their condition as comfortable as possible" (Bockoven 1963, p. 26). Moral treatment stressed a comprehensive assessment of the psychiatrically disabled, examining the person's work, play, and social activities. For example, a chaplain at a British asylum recognized the importance of patients' reentry into social life "by obtaining for them a change of scene and air and assisting them to obtain suitable employment" (Hawkins 1871, p. 107). Consistent with present-day rehabilitation practice, moral treatment recognized that structured activity can have therapeutic value. Today the goal of psychiatric rehabilitation practice is to have the person *do* something differently.

Vocational Rehabilitation Programs. While initial governmental programs for employment of the disabled, sparked by the end of World War I, focused on the physically handicapped, they did demonstrate that rehabilitation principles could be effectively implemented and won public support for rehabilitation as a societal responsibility. The 1943 amendments to the United States Vocational Rehabilitation Act extended financial support and vocational rehabilitation services to the psychiatrically disabled, with similar legislation appearing at the same time in England. These governmental actions provided legitimacy to the idea of training and rehabilitating people with psychiatric disabilities and grounded the practice of psychiatric rehabilitation in the vocational arena. The discovery of the capacity of the mentally retarded for gainful employment (O'Connor and Tizard 1956) led to studies on the effects of psychiatric disability on work and to the introduction of paid industrial subcontract work into large mental hospitals (Carstairs, O'Connor, and Rawnsley 1956).

The last three decades have seen the scope of rehabilitation move beyond a singular concern with vocational functioning to additional arenas of social and community functioning, but vocational activity has remained a preeminent ingredient in current rehabilitation practices (Beard, Propst, and Malamud 1982; Grob 1983). For example, Lamb (1982) has written that "work therapy geared to the capability of the individual patient should be a cornerstone of community treatment of the long-term patient" (p. 176).

Community Mental Health. The legislation that established community-based treatment for the mentally ill and retarded in the late 1950s and early 1960s endorsed the parity between physical and psychiatric disorders and their treatment. Mental illness and retardation no longer merited removal from society to large institutions; thus, the British Mental Health Act of 1959 supported "forms of training and social services which can be given without bringing patients into hospital as inpatients, or which make it possible to discharge them from hospital sooner" (Royal Commission 1957, p. 76). A new basic assumption prevailed—namely, people with major mental illness should be helped to maintain themselves in the community in as normal a manner as possible. Unfortunately, community mental health centers (CMHCs) failed to provide the comprehensive services needed by the severely psychiatrically disabled (Braun et al. 1981), who were not a high priority population for the CMHCs, perhaps because their interdisciplinary staff members were ill-equipped with techniques for effective work with chronic psychotics (Liberman, King, and DeRisi 1976).

The deinstitutionalization movement, which accompanied the opening of the CMHCs, fostered an appreciation of the value of work training in the preparation of patients for resettlement in the community. Psychiatrists were forced to match their view of the patient's abilities and disabilities against the realities of adaptation in the "real world." Studies were carried out in industrial therapy programs that showed the benefits and hazards of work for the mentally ill, changed the attitudes of citizens and professionals about the employability of mentally ill persons, and led to job placement programs (Bennett 1983).

Consistent with modern concepts of rehabilitation are those additional elements of community mental health ideology—accessibility to and comprehensiveness of services, and continuity of care. The emphasis on treating patients in proximity to their natural families and work settings has now been extended by the initiative of the National Institute of Mental Health for community support programs, which has encouraged State and local investments in a spectrum of services for the chronically mentally ill. In turn, this has meant an infusion of key operating principles for psychiatric rehabilitation, including case management, coordination, and advocacy with a variety of agencies capable of meeting the full range of needs of persons with severe psychiatric disabilities; involvement of patients and relatives in self-help; and assertive outreach.

Instead of expending great effort on reversing the disabling effects of the social breakdown syndrome from institutionalism, community-based rehabilitation has focused on the somewhat different problems of young adult persons with severe psychiatric disability, many of whom have spent only brief episodes in mental hospitals. Such individuals demonstrate severe handicaps in social adaptation and often resist engagement in professional services; moreover, their deficits, while less extensive than those of the older institutionalized patient, are marked by instability, intercurrent crises, and sensitivity to stress. Thus, even though CMHC personnel seemed to be intrigued by primary intervention for the not yet ill, and enamored of intensive verbal therapy for the not yet chronic, some of the innovations of the CMHC initiative became very much a part of the psychiatric rehabilitation field.

Psychosocial Rehabilitation Centers. The realization that severely and chronically mentally ill persons would rarely experience a full return of psychosocial functioning in the community led to a movement emphasizing accommodation to the needs of these persons in sustaining some semblance of normalization. With mental health professionals' neglect of the chronic mentally ill, nonprofessionals and patients themselves initiated psychosocial self-help clubs located in cities where the mentally ill congregated in large numbers. The early clubs, such as Fountain House and Horizon House, were founded by groups of ex-patients for the purpose of mutual aid and support. These early social clubs gave birth to comprehensive, multiservice psychosocial rehabilitation centers such as Thresholds in Chicago; the Social Rehabilitation Center in Fairfax, Virginia; Center Club in Boston; Fellowship House in Miami; Hill House in Cleveland; and Portals House in Los Angeles. The psychosocial centers assist patients to deal with their "real life" problems by providing opportunities for acceptable role performance and successful mutually interdependent relationships with others, by buffering stressors, and by making available a range of housing and employment options. From the very beginning, these centers have emphasized (1) strategies to help people *cope* with the environment rather than *succumb* to it, (2) health induction rather than symptom reduction, and (3) belief in the potential productivity of the most severely psychiatrically disabled client (Beard, Propst, and Malamud 1982).

Psychosocial centers have not valued the development of therapeutic insight (Dincin 1981). Their orientation has been on reality factors rather than intrapsychic factors, and on improving the person's ability to do something in a specific environment, even in the presence of residual disability (Grob 1983).

Psychosocial centers have played a significant role in the development of the psychiatric rehabilitation field. Their influence in the foreseeable future should be even greater since Fountain House has been conducting a training program to assist CMHCs and other types of mental health settings to establish rehabilitation services based on the psychosocial center model. In a 5-year period, individuals from agencies located in 38 states, the District of Columbia, Sweden, and Canada have been trained. During that time the number of rehabilitation programs has increased from 18 to 148. As the number of psychosocial rehabilitation centers continues to grow, this serves to ensure the presence of settings in which future research can be conducted and new rehabilitation techniques tested.

Skills Training. The most recent development in shaping psychiatric rehabilitation has been the introduction of skills training methods derived from social learning principles, human resource development training, and vocational rehabilitation. Effective coping with life stressors requires skills to promote problem solving, engage others in successful affiliative and instrumental relationships, mobilize supportive networks, and engage in work. Rehabilitation techniques that use active-directive learning principles—behavioral practice and role playing, social and tangible reinforcement, shaping, coaching, and prompting, and generalization activities—strengthen an individual's problem-solving capacities and confer protection against exacerbations of psychiatric symptoms (Cohen, Ridley, and Cohen 1983; Wallace and Liberman 1985).

Skills training has been found effective in group and individual therapy, family therapy, milieu therapy, and vocational settings (Anthony, Howell, and Danley 1984; Liberman et al., this issue; Strachan, this issue). The point of training interpersonal skills is to improve the individual's ability to master the challenges and problems inherent in daily life. It is assumed that, within genetic and constitutional constraints, each patient tries to do his or her best, so that the problem lies not in resistance or lack of motivation, but in a deficit of some sort. The source of deficit is regarded as less important than using remedial training methods to enhance the individual's ability to cope. Successful coping leads to attainment of social and emotional goals that define competence and adjustment. While skills-training approaches have shown much promise and empirical efficacy (Carkhuff 1972; Paul and Lentz 1977; Carkhuff 1983; Liberman 1984), they have been adopted by practitioners very slowly because of the demanding competencies required of those who seek to do such training (Backer, Liberman, and Kuehnel 1986; Cohen et al., this issue).

Conceptual Model for Rehabilitation

Within the last several decades, a consensus has developed about what constitutes a conceptual model for rehabilitation and its underlying philosophy, as well as what constitutes appropriate rehabilitation practice. (See table 1.) The conceptual model was introduced by leaders in physical medicine and rehabilitation and later extended the mental health disciplines (Anthony 1980, 1982). Both physically and psychiatrically disabled persons exhibit disabilities in social function; handicaps in role performance; needs for a wide range of services, often for a long period of time; and frequent failure to experience total recovery. Rehabilitation of psychiatric disorders, as with physical illnesses, begins when the *pathology* and *impairments* of the acute stage stabilize. In cardiac rehabilitation, then, intervention begins after the myocardial infarction and its associated pain and stress have resolved. Similarly, rehabilitation for a person with schizophrenia begins when the acute and florid psychotic symptoms recede or stabilize. Even if symptoms persist, rehabilitation can proceed within the limits of the individual's capability to respond to

Table 1. Stages in the rehabilitation of chronic mental patients, with examples of the elements in each stage

Stage:	**Pathology**	**Impairment**	**Disability**	**Handicap**
Definition:	Lesions or abnormalities in the central nervous system caused by agents or processes responsible for the etiology and maintenance of the biobehavioral disorder	Any loss or abnormality of *psychological, physiological*, or *anatomical structure* or *function* (resulting from underlying pathology)	Any restriction or lack (resulting from an impairment) of *ability to perform an activity* in the manner or within the range considered normal for a human being	A disadvantage for a given individual (resulting from an impairment or a disability) that limits or prevents *the fulfillment of a role* that is normal (*depending on age, sex, social, cultural factors*) for that individual
Example:	Brain tumors or infections etiologically linked to psychotic symptoms	Positive and negative symptoms of schizophrenia (delusions, anhedonia)	Deficient social skills	Unemployment, homelessness,
Interventions:	Laboratory and radiographic tests	Syndromal diagnosis, pharmacotherapy, hospitalization	Functional assessment, skills training, social support	National and State vocational rehabilitation policies; Community support programs

training and supportive interventions. The focus of the rehabilitation practitioner is on remediating *disabilities* and compensating for *handicaps* (Wood 1980; Frey 1984).

The pathology in schizophrenia is still poorly understood, although brain-imaging techniques are revealing intriguing leads for structural and functional abnormalities in certain brain regions (Weinberger and Kleinman 1986). Symptoms such as delusions, hallucinations, and thought disorder are the impairments of schizophrenia. These impairments can impede an individual's ability to perform certain skills and activities which, in turn, can limit the fulfillment of social and vocational roles.

In view of the intrusion of symptoms of major mental disorders—delusions, hallucinations, depression, and anxiety—on the cognitive and interpersonal capabilities of afflicted individuals, it is not surprising that such impairments lead to significant disability and handicap. For example, unemployment has been found to be as high as 70 percent in the chronic mentally disabled (Goldstrom and Manderscheid 1982). Unemployment is a prime index of handicap since the demands of work in modern society accentuate the deficits, deviance, and stigma of mental patients. Employment rates following hospital discharge range between 10 and 30 percent, and only 10–15 percent of patients sustain their employment 1–5 years after discharge, with more recent studies revealing lower rates (Anthony et al. 1972; Anthony, Howell, and Danley 1984). Difficulties in work tolerance, endurance, following instructions, cooperating with co-workers and supervisors, problem solving, task orientation, sustained concentration, and ability to accept criticism and ask for assistance are all examples of disabilities that are caused by symptomatic and cognitive impairments and that lead to significant handicap.

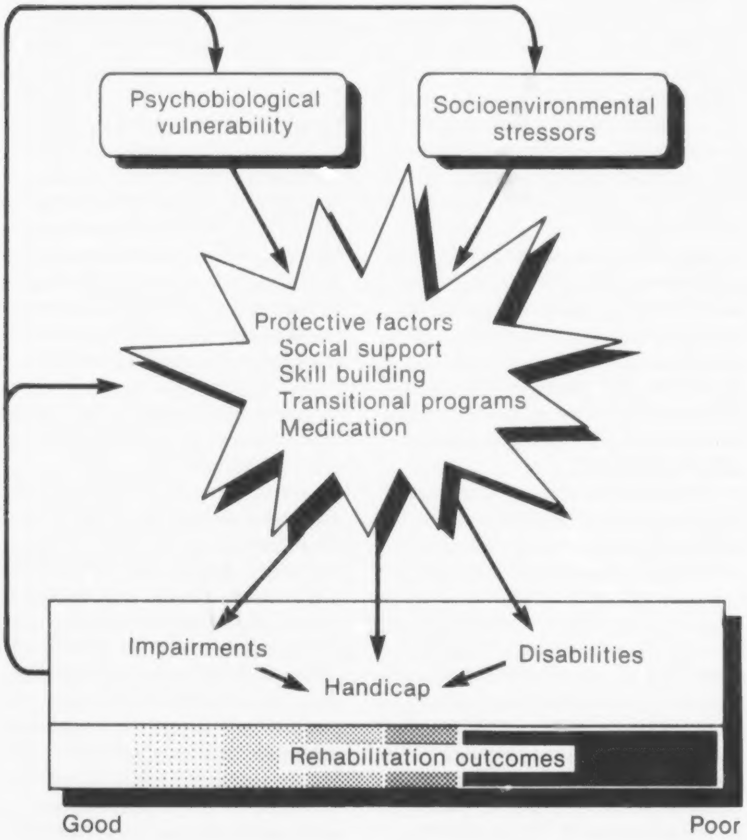
Thus, the impairments, disabilities, and handicaps of persons with psychiatric disorders are related to their psychiatric symptoms, and their vocational and social deficits. Symptomatic and functional problems exist on a continuum. For example, the nature of the illness can influence the severity and chronicity of the symptoms and social dysfunctions. Individuals with schizophrenia generally are more impaired and disabled than those with affective or anxiety disorders (Harrow et al. 1978). Within any one type of disorder, however, symptomatic and social-vocational course and outcomes vary considerably, both for different individuals and for the same individual over time (Liberman 1982*a*). Moreover, there are not always high correlations among the major dimensions of outcome of severe mental disorders; for example, social functioning may be adequate even in the face of poor vocational functioning and persisting symptoms (Anthony and Buell 1974; Buell and Anthony 1976; Anthony 1979). Alternatively, symptoms may persist but still permit adequate job adjustment (Strauss and Carpenter 1981; Anthony and Jansen 1984). The variability in the manifestations of major mental disorders is one of the most salient features of these disorders and can be best understood and studied through the interplay of vulnerability, stress, coping, and competence factors.

The *vulnerability-stress-coping-competence* model of major mental disorders explains the onset, course, and outcome of symptoms and social functioning as a complex interaction among biological, environmental, and behavioral factors (Liberman 1982*b*; Nuechterlein and Dawson 1984), and is congruent with the rehabilitation conceptualization. Psychobiological vulnerability may result in psychotic symptoms when stressful life events or ambient tensions in the family or work setting overwhelm coping skills.

Vulnerability and stressors (figure 1) are moderated in their impact on impairment, disability, and handicap by the presence and action of protective and potentiating factors. Prime among protective factors are coping and competence exercised by individuals, families, natural support systems, and professional treatment. Examples of protective factors include rehabilitation programs that offer skill building, social support, or transitional employment. Coping and competence can be attributes of the individual or of the social environment. From this point of view, an exacerbation or relapse of schizophrenic symptoms that accompanied use of street drugs of abuse (e.g., PCP, amphetamines) would result from the stressful action of these drugs on the individual's underlying biological diathesis for schizophrenia. In like manner, stressful life events (e.g., loss of a trusted therapist or discharge from long-term hospitalization) that overwhelm the protective effects of medication, personal coping, and social support can also lead to symptomatic exacerbation. Even in the absence of a time-limited stressor, vulnerable individuals can succumb to ambient levels of challenge, tension, or conflict in their environment if they lack the protection conferred by medication, coping abilities, and social support.

The *vulnerability-stress-coping-competence* model highlights the role of specific psychosocial interventions in developing personal and familial coping skills, and interpersonal and vocational competence as protective factors in the course of mental disorders. Psychosocial protective factors buffer the impact of potentiators and stressors, and thereby reduce the probability of symptomatic relapse. Socially learned coping helps individuals to obtain their instrumental and social-emotional needs by meeting the challenges and solving the problems of everyday life. Coping and competence protect an individual with a given level of vulnerability to schizophrenia from stressful life events and ambient levels of environmental tension. At any level of psychopathology, coping can reduce the social, occupational, and self-care impairments that are associated with the disorder.

Figure 1. Vulnerability, stress, coping, and competence model of mental disorders



In this conceptual framework protective factors that facilitate coping and competence can modulate the deleterious affects of psychobiological vulnerability and socioenvironmental stressors. Coping and competence confer protection against impairment, disability, and handicap.

The model also encourages investigators to design optimal psychopharmacological interventions to modify the effect of psychobiological vulnerability factors. For example, antipsychotic medication serves as a personal protective factor against biological vulnerability; it thereby decreases relapse rates and improves the course of schizophrenic disorders. Antipsychotic medication also raises the threshold at which environmental potentiators and stressors precipitate psychotic symptoms in an individual with a given level of vulnerability to schizophrenic episodes (Leff et al. 1973; Vaughn and Leff 1976*a*, 1976*b*). However, the modulation of biological vulnerability by antipsychotic medication cannot fully remediate a vulnerable individual's susceptibility to relapse when faced with severe stressors, loss of social support, or diminution in personal problem-solving skills. Even with reliable ingestion of neuroleptics, for example, upwards of 30–40 percent of schizophrenic patients relapse within a year (Liberman 1984).

In this *vulnerability-stress-coping-competence* model, the appearance or exacerbation of characteristic schizophrenic symptoms and associated disabilities may occur in susceptible individuals when:

- Underlying psychobiological vulnerability factors are triggered, which is more likely in the absence of optimal antipsychotic medication.
- Stressful life events intervene that exceed the individual's coping skills and competencies in social and instrumental roles.
- The social support network weakens or diminishes;
- Coping and problem-solving skills atrophy as a result of disuse, reinforcement of the sick role, or loss of motivation.

Psychiatric Impairment, Disability, and Handicap

From the point of view of rehabilitation professionals, the psychobiological abnormalities in the nervous system that produce deficiencies in cognitive, attentional, and autonomic functions, and in regulation of arousal and information processing, represent the active *pathology* or *disease* state. The neurosciences are just beginning to develop instruments and techniques that can sensitively measure these abnormalities. However, they can be inferred through assessment of *impairments* that presumably index the more basic disturbances in brain function.

While impairments in physical rehabilitation include vision or hearing loss, reduced range of motion in an extremity, and loss of strength in a muscle group, *psychiatric impairments* can include thought disorder and speech incoherence, delusions, hallucinations, anxiety, depression, loss of concentration or memory, distractibility, and apathy and anhedonia. Thus, psychiatric symptoms and cognitive-emotional deficits are both correlates of the course and outcome of psychiatric illnesses and of the pathological processes in the nervous system. Presumably, these impairments are somewhat specific for each of the major mental disorders and vary with the severity of the underlying psychobiological vulnerability. The specificity of the impairments enables diagnosticians to categorize psychiatric disorders reliably (e.g., *DSM-III*); however, there is considerable overlap between disorders as evidenced by the presence of delusions and hallucinations in a variety of psychotic disorders (Pfohl and Andreasen 1986).

When functional limitations imposed by psychiatric *impairments* result in decrements in the ability to perform certain activities, the individual is said to have a *disability*. *Disabilities* are defined as inability or limitation to perform tasks expected of an individual within a social environment (Frey 1984). Among individuals with severe psychiatric disorders such as schizophrenia, *disabilities* include poor self-care skills (e.g., cooking, cleaning, grooming, and teeth care), social withdrawal and seclusiveness, abandonment of family responsibilities, and work incapacity. *DSM-III* (American Psychiatric Association 1980) has highlighted the importance of these disabilities by including them as criteria for many diagnosable psychiatric conditions. To be diagnosed as having schizophrenia, for example, it is not sufficient to experience the characteristic symptoms of thought disorder, delusions and hallucinations, but the person also must evince a "deterioration from a previous level of functioning in such areas as work, social relations, and self-care" (American Psychiatric Association, 1980, p. 189).

The *disabilities* shown by persons with psychiatric disorders are influenced by the same protective and risk factors that influence the appearance, exacerbation, and remission of symptoms or *impairments*. Thus, correlations between symptoms and disabilities would be expected; however, not every impairment results in a disability. Furthermore, similar patterns of disability can result from different disorders and impairments. Persons with mental retardation or affective disorders may have the same profile of disabilities as those with schizophrenia. Social and vocational disabilities form a major cluster of behaviors that both reflect and influence the course and outcome of a psychiatric disorder.

Research data substantiate a consistent relationship between a person's abilities and vocational outcome. For example, in every study in which work adjustment skills were assessed, they were found to be significantly related to future work performance (Anthony and Jansen 1984). Similar to these data on work adjustment skills, ratings of interpersonal or social skills have been found to predict vocational performance even though social functioning was measured differently in the various studies (Anthony, Cohen, and Cohen 1984).

A final element in the rehabilitation model is *handicap*, which occurs when *disabilities* place the individual at a disadvantage relative to others in society. This can occur through stigma and discrimination, as when employers are reluctant to hire persons with mental illnesses. Handicap also occurs because society does not provide settings where mentally ill persons can find accommodation and compensation for their *impairments* and *disabilities*. Wheelchairs and ramps have enabled paraplegics to overcome their impairments and disabilities to find remunerative work and fulfilling recreation; hence, their handicaps are compensated. Because mentally ill persons require special *social environments* to compensate for their problems, overcoming handicap is much more difficult. Long-term institutional care in State and county mental hospitals was formerly society's method of dealing with the *impairments* and *disabilities* of psychiatric patients; unfortunately, institutionalization also created its own set of secondary disabilities and handicaps. That society has not yet succeeded in compensating for the impairments and disabilities of psychiatric patients is reflected in the extraordinarily high rates of unemployment and homelessness among this population (Anthony, Cohen, and Vitalo 1978; Farr and Koegel 1986).

Psychiatric Rehabilitation

The conceptual framework elucidated above, joined with the rehabilitation view of disability, provides a coherent set of strategies for rehabilitation interventions with the psychiatrically disabled. The clinical practice of psychiatric rehabilitation, just like its counterpart in physical rehabilitation, comprises two intervention strategies: (1) patient skill development and (2) environmental resource development. In developing these intervention techniques, psychiatric rehabilitation practice is guided by the basic philosophy of rehabilitation; that is, disabled persons need *skills* and *environmental supports* to fulfill the role demands of various living, learning, and working environments. The assumption of clinical rehabilitation is that if psychiatrically disabled persons' skills and/or the supports in their immediate environment are augmented, they will be more able to perform those activities necessary to function in specific roles of their choice. In other words, interventions designed to lessen or compensate for the disability are assumed to lead to a decrease in the handicap.

Interventions begin with a comprehensive medical-psychiatric diagnosis and a functional and resource assessment. This enables mental health and rehabilitation professionals to describe individuals by diagnostic disorder, level of behavioral functioning, and amount of environmental supports, key to identifying their impairments and disabilities. Identification of impairments and disabilities permits the mental health and rehabilitation practitioner to prioritize problems, formulate specific goals, and organize and implement treatment and rehabilitation plans. Medical-psychiatric diagnosis and a functional and resource assessment are necessary to match patients to drug and psychosocial treatments and rehabilitation programs that will be effective (Taylor, Liberman, and Agras 1982). Furthermore, knowing the patient's diagnosis aids the clinician in providing the patient and family members with a reasonable prognosis and in determining the degree to which environmental support vs. remedial skills training can be provided to overcome handicaps.

Reduction of Impairments. Rehabilitation interventions with psychiatric patients require reduction or elimination of the symptomatic and cognitive impairments that interfere with social and vocational performance. Interventions for reducing impairments are fortunately available from psychopharmacology. The past 20 years has seen major accomplishments in both treatment and prevention of morbidity from psychopathology through the use of antipsychotic, antidepressant, and anxiolytic drugs. Note that these psychotropic drugs are not panaceas. Even when taken regularly, they are frequently only able to reduce rather than abolish symptoms, and delay rather than prevent relapse. Psychotropic drugs are also associated with unpleasant side effects which at times can interfere with skills-training activities. However, they are usually helpful in reducing impairments to the point where psychosocial strategies can be used effectively to remediate disabilities and handicap.

While a patient's symptoms and syndromal diagnosis clearly impair social and vocational performance, as is highlighted by the mixed symptomatic and functional criteria in *DSM-III*, the remitting and exacerbating nature of most major mental disorders obscures the relationship between psychopathology at time A and behavioral functioning at time B. The changing character of the symptomatic impairments of psychiatric disorders with the passage of time accounts for the insubstantial correlations often found between psychopathology measured during an acute episode and future work performance (for a review, see Anthony and Jansen 1984).

The disappointing failure of research studies to find correlations between impairments and disabilities might be a result of their having been conducted in an era preceding the availability of objective and reliable instruments for eliciting and rating psychopathology. Research using the standardization in diagnosis brought about by *DSM-III* should yield studies that suggest a stronger relationship between syndromes and long-term outcome (Tsuang, Wooson, and Fleming 1979; Pfohl and Andreasen 1986).

Remediation of Disabilities Through Skills Training. Once a patient has benefited optimally from psychotropic drugs and the therapeutic effects of brief hospitalization, rehabilitation strategies use skills training to remediate disabilities in social, family, and vocational functioning. Skills training as a principal strategy in psychiatric rehabilitation starts from the assumption that many patients will suffer persisting disabilities despite the best efforts at pharmacotherapy and hospitalization. A relatively new field, training of social and vocational skills has relevance for a wide variety of psychiatric patients and for the professionals who serve them.

The psychiatric disorders with the greatest chronicity—schizophrenia, major depression, and organic syndromes—are those most in need of a skills-training focus. While appropriate drug treatment significantly reduces symptoms in most schizophrenics and depressives, many are refractory to drugs and others experience continuing social and vocational handicaps even with symptomatic improvement. The negative or deficit symptoms of schizophrenia, for example, pose a largely unanswered challenge to the pharmacopeia. Social withdrawal, apathy, energy, slovenliness, and anhedonia do not respond as well to neuroleptic drugs as do hallucinations, delusions, and thought disorder. Neither do drugs teach life and coping skills, except indirectly through removal or reduction of symptoms. Most schizophrenic persons need to learn or relearn social and personal skills for surviving in the community.

Skills training can begin immediately after the stabilization of an acute episode or exacerbation of a psychiatric disorder which usually results in the loss of social and role functioning. The goals of rehabilitation professionals are to sustain symptomatic improvement over the long haul; establish or reestablish interpersonal and independent living skills; and help the individual reach a satisfactory quality of life.

Because the goals of rehabilitation center on adjustment to everyday life, it is vital for the schizophrenic individual to participate maximally in the choice of objectives, and in the learning process. Comprehensive rehabilitation involves assessment, training, and modification of living environments in those areas relevant to personal and community life—self-care, including medication and symptom management; family relations; peer and friendship relations; avocational and employment pursuits; money management and consumerism; residential living; recreational activities; transportation; food preparation; and choice and use of public agencies. Specific goal setting, within these generic areas, should actively involve the patient, his or her family, and significant others.

Skills training has proved to be an effective way of increasing the coping and competence of chronic mental patients (Anthony 1979; Wallace et al. 1980; Liberman, Falloon, and Wallace 1984). There are several sources of empirical data that recommend skills training to improve patients' competence and coping ability. Many studies have highlighted the importance of premorbid and postmorbid social competence as a predictor of outcome in major psychiatric disorders (Liberman 1982*a*; Presly, Grubb, and Semple 1982; Hirschfeld et al. 1983). This suggests that social-skills training might improve the long-term prognosis by upgrading the postmorbid social competence of chronic patients. Second, the magnitude of deficits in social and living skills has been well documented in chronic psychiatric patients. For example, in one study major functional deficits in social and personal areas were found in over 50 percent of a sample of chronic psychiatric patients (Sylph, Ross, and Kedward 1978). Two studies of schizophrenic patients found that both discharge and remission rates were significantly higher among patients who had higher levels of living and social skills (Linn, Klett, and Caffey 1980; Farkas, Rogers, and Thurer 1986). In another large-scale study comparing depressed patients and normals, social introversion and interpersonal dependency were the most significant premorbid personal characteristics associated with the patient sample (Hirschfeld et al. 1983).

Moreover, the recent scientific literature has seen publication of well-controlled and efficacious training programs to enhance social, vocational, and living skills (Monti et al. 1979; Nelson and Cone 1979; Monti, Corriveau, and Curran 1982; Falloon, Boyd, and McGill 1984; Jacobs et al. 1984; Kelly and Lampariski 1985). The learning of social and living skills is often affected—for better or worse—by the concurrent administration of appropriate types and doses of psychotropic drugs; therefore, it is important for rehabilitation practitioners using skills-training methods to work in close liaison with medical-psychiatric practitioners who prescribe psychotropic drugs. Judicious administration of psychotropic drugs reduces cognitive and symptomatic impairments, enabling patients to attend to learning stimuli and to engage actively in skills-training procedures (Liberman, Falloon and Wallace 1984).

In 1974, Anthony and Margules published a review which concluded that "persons with a psychiatric disability can in fact learn useful skills" (p. 102). Specifically, the studies reviewed at that time, and again 10 years later (Anthony, Cohen, and Cohen 1984), showed that psychiatrically disabled persons could learn a variety of self-care, social-interpersonal, and intellectual skills.

For example, training programs have had an impact on the psychiatrically disabled person's skills in a variety of areas, including personal hygiene, cooking, use of public transportation, use of recreational facilities, physical fitness, interpersonal skills, self-control skills, job-interviewing skills, family relationship skills, money management, and job-seeking and work-adjustment skills (Anthony 1979; Falloon, Doane, and Pederson 1985; Wallace and Liberman 1985).

In many of these studies psychiatrically disabled persons who were long-term inpatients with a lengthy history of symptomatic behavior were successfully trained. For example, longitudinal, follow-through study of over 200 chronic psychotic patients who were involved in an intensive rehabilitation program as part of their discharge from Vermont State Hospital in the late 1950s found that approximately two-thirds were functioning in a near-normal fashion in social and vocational roles 20 years later in the community (Harding and Brooks 1984). It has now become an accepted fact that psychiatrically disabled persons can learn skills, and that chronicity and symptomatology may qualify but do not prevent skill learning. The results of these studies are thus seen as supportive of a psychiatric rehabilitation approach, with its emphasis on assessing and increasing psychiatrically disabled persons' skills.

Social and vocational skills training uses procedures based on principles of human learning to train specific interpersonal skills and to promote the generalization and maintenance of these skills. The training procedures have been empirically tested and "packaged" for ready access by practitioners.

While many psychosocial programs bill themselves as offering social-skills training, it is important to distinguish between nonspecific group activities that engage patients in "socialization," and methods that deliberately and systematically use behavioral learning techniques in a structured approach to skills building (Farkas, Cohen, and Nemec 1986). While socialization activities can lead to acquisition of skills through incidental learning during spontaneous social interactions, they do not harness social learning and reinforcement techniques that may be required to promote the acquisition, generalization, and durability of skills needed in interpersonal situations (Liberman et al. 1975).

The learning disabilities experienced by many persons with prolonged psychiatric disorders require the use of highly directive behavioral techniques for training social skills. For example, most psychiatrically disabled individuals have attentional and information-processing deficits. They show hyperarousal or underarousal in psychophysiological testing, and they experience overstimulation from emotional stressors or even from therapy sessions that are not carefully structured and modulated. These individuals often fail to be motivated by the customary forms of social and tangible rewards available in traditional therapy. In addition, they may lack conversational ability, a basic building block for social competence. Persons with schizophrenia, in particular, are deficient in social initiative and perception, and they have difficulty generating alternatives for coping with everyday problems such as missing a bus, making an appointment, or getting help with bothersome drug side effects. They tend to make less eye contact, have more verbal dysfluencies, and use less vocal intonation, all of which may impair social learning (Wallace et al. 1980).

Skills training procedures must be tailored to the needs of the individual patient, as patients present different constellations of social abilities and deficiencies. Moreover, a comprehensive assessment of personal and community resources that can be tapped for promoting the learning of skills should be a prelude to goal setting and skills training (Cohen, Farkas, and Cohen 1986).

Several training models are presently available to the clinician. Longest in use is a treatment "package" for training skills which includes specific, goal-oriented instructions to the patient, the therapist modeling appropriate use of the skills, the patient role-playing interpersonal situations, and the therapist reinforcing and providing corrective feedback to the patient (Liberman et al. 1975; Cohen, Danley, and Nemec 1986). Recently, training within an information-processing framework has been shown to be effective for patients capable of learning problem-solving strategies (Foy, Wallace, and Liberman 1983; Medication Management Module 1986). Disabled individuals are taught to improve their perception of information in immediate interpersonal situations, process that information to choose a response, and send a response back to the other person. However, both of these approaches are ineffective for patients with severe attentional deficiencies. A model using attention-focusing procedures that simplify the learning of complex skills has been effective in training conversational skills in some seriously regressed, chronic psychiatric patients (Liberman et al. 1985).

Remediating Disabilities Through Supportive Interventions. When restoration of social and vocational functioning through skills training is limited by continuing deficits and refractory symptoms, rehabilitation strategies aim at helping the individual compensate for the disability by (1) locating living, learning, and working environments that can accommodate to the residual deficits and symptoms; and (2) adjusting the individual's and family's expectations to a level of functioning that is realistically attainable. Thus, environmental modification and supportive-prosthetic social and vocational environments are complementary approaches to skills training in the reconstitution of social roles for patients with severe and chronic psychiatric disorders.

Environmental interventions attempt to provide the patient with supportive persons, supportive settings, or both. A "support person" might reduce a person's disability and handicap through a number of different roles (e.g., advocate, companion, counselor, and advisor). Attempts at making the setting more supportive focus on the programs or resources within the environment rather than on support persons, per se (e.g., sheltered work and living settings, and special discharge programs). The distinction between supportive persons and supportive settings simply highlights the different ways in which environmental modifications occur. In practice, these modifications often occur simultaneously.

The main identifying feature of both types of supportive interventions, as distinguished from skill-development interventions, is that they do not attempt to change the patient's behavior systematically and directly. Rather, the attempt is simply to support and accommodate the patient's present level of functioning. The early studies by Katkin (1971, 1973) and a more recent study by Cannady (1982) have clearly demonstrated the positive impact on patient outcome of a support person. Cannady (1982) used citizens from the discharged patients' rural neighborhood to function as "supportive case workers." Over a 12-month period, inpatient days were decreased by as much as 92 percent.

Witheridge, Dincin, and Appleby (1982) have reported on the use of a support team for psychiatrically disabled individuals at high risk for readmission. Working out of the patients' homes and neighborhoods, this team had as its goal to develop an individualized support system for each patient. Of the original 50 participants, 41 remained in the program. One-year followup data indicated that average days hospitalized per year decreased from 87.1 to 36.6. Even better results were obtained in a similar program which has been a model for assertive outreach and advocacy for chronic mentally ill persons (Stein and Test 1978).

A study by Stickney, Hall, and Gardner (1980) investigated the effects of introducing both a support person *and* a more supportive environment, separately and in combination, at the time of the person's hospital discharge. They studied the impact of four predischarge formats, which differed in level of personal and environmental support. The goal of the discharge plans was to increase use of the CMHC and to decrease recidivism for 400 patients discharged from a State mental hospital. The results of the study demonstrated the impact of increasing personal and environmental support on patient compliance with referral and 1-year hospital recidivism rates. With minimal support, the respective referral compliance and recidivism percentages were 22 percent and 68 percent; with increased personal support, 36 percent and 39 percent; with increased environmental support, 68 percent and 31 percent; and with both increased personal and environmental support, 75 percent and 28 percent. Thus, whenever an added element of support was introduced, the referral compliance percentage increased and the recidivism rate decreased.

Ballantyne (1983) and Wasylenski et al. (1985) have reported results from an environmental intervention study based directly on the principles of psychiatric rehabilitation (Anthony 1979). Community rehabilitation workers were trained to provide interpersonal support and case management to patients being discharged from the hospital. Compared to a matched group of patients receiving traditional discharge planning and aftercare, patients in the experimental group were discharged an average of 21 days sooner and participated in approximately twice as many aftercare services. Patient perception of the program indicated high levels of involvement and satisfaction. At 6-month followup, over 75 percent of the patients were still involved in the program; 87 percent reported "feeling understood"; and 88 percent reported "feeling like a partner" in the rehabilitation planning process. In addition, there were significant differences in instrumental role functioning as student, worker, or homemaker.

Weinman and Kleiner (1978) compared the effectiveness of community-based "enablers" with two hospital-based conditions: socio-environmental therapy and traditional hospital treatment. The enablers' major roles were to assist their patients in coping with community life and escort them to various community resources. Personal support proved superior to each of the hospital-based treatment approaches in recidivism, patient self-esteem, and patient instrumental and role performance.

Remediation of Handicap. In addition to clinical rehabilitation interventions of skill and support development, psychiatrically disabled persons can be helped to overcome their handicaps through societal rehabilitation interventions (Anthony 1972). Societal rehabilitation is designed to change the system in which psychiatrically disabled persons must function. Unlike clinical rehabilitation, its focus is neither on the skills of specific psychiatrically disabled individuals nor on their unique environments. Rather, the focus is on system changes that can help many psychiatrically disabled persons overcome their handicaps. Examples of this type of system intervention are the Targeted Job Tax Credit legislation, changes in the length of the trial work period in the Social Security Disability program, and the development of a European-type quota system for the employment of disabled workers. The importance of these system-type interventions cannot be overemphasized. Obstacles in overcoming a handicap may be more a function of a nonaccommodating and discriminating social and economic system than of the person's impairment and disability. Community support programs are another example of a system-wide response to the problems of persons who are severely psychiatrically disabled (Turner and TenHoor 1978).

Clinical and societal rehabilitation interventions are not mutually exclusive. As a matter of fact, the 1973 amendments to the Vocational Rehabilitation Act recognize the value of societal rehabilitation efforts. The amendments established the principle of affirmative action by contractors who do business with the Federal Government and also attempted to establish the Government as a model employer with respect to architectural access (Stubbins 1982).

Integrating Rehabilitation Strategies

In view of the pervasive impairments, disabilities, and handicaps of most persons with chronic mental disorders, a combined approach to psychiatric rehabilitation, employing skills training and environmental modification strategies, is most often required. As the following case vignette illustrates, the rehabilitation practitioner or team needs to integrate skills training, social support, and governmental incentives and regulations to bring about an optimal outcome.

> A 30-year-old male with a 15-year history of being in and out of private and State psychiatric hospitals decided he would like to get a job in the community after being in a psychosocial work adjustment program for 1 year. He did not have a good work history; for example, 6 months was the longest he had ever been able to hold a job. Using the targeted job tax credit legislation as an incentive to the employer, the psychiatric rehabilitation team was able to find the disabled person a job working in a video repair shop, a job consistent with the client's interests and talents. To keep his job at the repair shop, the patient needed to learn the skills of taking orders from authority figures (showing understanding of what others say and expressing his own thoughts and feelings to others). The team also made the environment more supportive to the patient by educating the employer to the patient's needs, and obtained employer agreement on reducing the initial work load/time until the disabled person became comfortable with the new environment.

In general, all available interventions are used in combination—psychotropic medication; partial and full hospitalization; case management; skills training; social self-help clubs; and environmental support initiatives—to achieve the maximum degree of adaptation that is feasible. The emphasis on each of the types of intervention varies with the nature of the disorder, the premorbid level of competence of the patient, and the phase of illness.

Historically, mental health treatment has tried to develop interventions aimed at the patient's impairments. Somatic and psychological treatment efforts have attempted to alleviate the signs and symptoms of psychopathology. Leitner and Drasgow (1972), in analyzing the differences between treatment and rehabilitation, point out that treatment attempts are directed more toward minimizing sickness, whereas rehabilitation aims more toward maximizing health. Eliminating or suppressing impairments does not lead automatically to more functional behaviors. Likewise, a decrease in disability does not always lead to reductions in impairment. Note that a chronic or severe impairment (e.g., diabetes, schizophrenia, and stroke) does *not* always result in chronic disability or handicap. What the impairment does is increase the risk of chronic disability and handicap.

Conclusion

With an emerging consensus that major psychiatric disorders are stress-linked biomedical disorders, rehabilitation approaches have grown out of eclectic and empirical traditions emphasizing the development of patients' skills or supportive environments for coping with the enduring disabilities and handicaps of illnesses such as schizophrenia and affective disorders. Psychiatric rehabilitation uses assessment and intervention techniques based on such orientations as social learning and behavior therapy (Paul and Lentz 1977; Liberman and Foy 1983; Liberman and Evans 1985); client-centered therapy and human resource development (Rogers 1957; Carkhuff 1972, 1983; Carkhuff and Berenson 1976); and lifespan developmental psychology (Strauss and Carpenter 1981; Pepper 1985).

The psychiatrically disabled person must be involved as much as possible in setting rehabilitation goals—a process that necessitates the development of a trusting, mutually respectful, and empathic relationship with service providers. The preferred mode of intervention combines judicious and rational psychopharmacology with an educational approach that trains patients directly in the knowledge and skills they need to function in society. "Teaching as treatment" (Carkhuff and Berenson 1976) is supplemented by interventions at the level of the person's immediate environment, as well as at the community support system and societal level. An operational philosophy defining the scope of psychiatric rehabilitation has recently been issued by the National Institute of Mental Health (Stockdill 1985).

> Psychosocial rehabilitation refers to a spectrum of programs for persons with long-term, severe psychiatric disabilities. The goal is to improve the quality of life for these individuals by assisting them to assume responsibility over their lives and to function as actively and independently in society as possible. The programs are designed to both strengthen the individual's skills and to develop the environmental supports necessary to sustain the individual in the community.
>
> Major services, which may be offered on a continuum, should be coordinated with those offered by other agencies providing psychiatric, as well as the broad array of human services. The specific services include opportunities to meet social/recreational needs, vocational training and job placement rehabilitative residential services, training in the skills of daily and community living, and case management services. In addition, some psychosocial rehabilitation programs include client assessment and goal-planning activities, educational programs and family support activities.
>
> The services, which the individual may need to use on a short-term basis or indefinitely to achieve or maintain community adjustment, are provided in the context of a supportive, normalizing environment in the community. They are offered in a manner that emphasizes the "personhood" rather than the "patienthood" of the individual, that maximizes the individual's responsibility, control and feelings of self-worth, and that encourages ownership in the rehabilitation process. [p. 2]

The rehabilitation process encompasses three overlapping stages that recur for as long as the patient requires professional services (Anthony, Cohen, and Cohen 1983). *Assessment* at the symptomatic, functional, and resource levels initiates the patient and professional in a collaborative *planning phase*. Through diagnostic and assessment interviews, inventories, informants, historical data, role plays, and direct behavioral observation, the assessment yields information about the psychiatrically disabled person's current deficits, psychopathology, skills, and supports, as well as the skill level demanded by the living, learning, or working environments in which the patient wishes to function. The assessment information enables the rehabilitation practitioner to work with the patient and family member in the *planning phase* to develop a rehabilitation plan that specifies how the person or the person's environment must change to achieve the goals of rehabilitation. With respect to changes in the person, the plan develops the skill steps the person needs to raise his or her level of functioning to the level required by the environment. With respect to changes in the person's environment, a sequential plan describes what and how the necessary coordination, advocacy, and modifications are to be made. The rehabilitation plan also identifies the persons (e.g., practitioner, patient, agency, and family member) responsible for implementing the various parts of the plan.

In the *intervention phase*, the rehabilitation plan is implemented to increase the person's skills and to make the environment more supportive of the person's functioning. These interventions can lead to the achievement of the rehabilitation goals, first identified during the assessment phase. Repeated and regular monitoring of the change process informs all concerned about goal attainment and enables clinical decisions to be made about continuation or change of interventions and goals.

Psychiatric rehabilitation is the recovery of social and instrumental role functioning to the fullest extent possible through learning procedures and environmental supports. When restoration of functioning is limited by continuing deficits and symptoms, rehabilitation efforts aim at helping the individual (1) acquire living, learning, and working environments that are compensatory; and (2) adjust to the level of functioning that is realistically attainable. Because of the limitations of currently available training methods, patients with more chronically disabling disorders, such as schizophrenia or organic brain disorders, may be unable to reestablish specific impaired or lost skills. In such cases, alternate compensatory skills and environments, such as learning to function in sheltered employment and residential settings, would be the focus of rehabilitative efforts.

With chronic psychiatric disorders, some amount of continuing symptoms and residual impairment makes necessary the acceptance of disability and the identification of new, attainable goals. Learning how to cope with symptoms, manage medication, and utilize professional resources when necessary become important targets for rehabilitation. When deficits preclude gainful employment, the patient and family need assistance in generating alternative types of meaningful activity, social contacts, and daily structure. While persisting symptoms may limit the level of functional engagement in life, the reciprocal also holds—namely, the more that rehabilitation improves the patient's social and role performance, the more likely that symptoms will be held in check.

Rehabilitation begins immediately after the stabilization of an acute episode or exacerbation of psychiatric disorder which usually results in the loss of social and role functioning. The goals of rehabilitation professionals are to sustain symptomatic improvement over the long haul; establish or reestablish interpersonal and independent living skills; and help the individual reach a satisfactory quality of life.

Comprehensive rehabilitation involves assessment, training, and modification of environments in areas relevant to personal and community life. Professionals contributing to psychiatric rehabilitation include psychologists, vocational rehabilitation counselors, occupational therapists, recreational therapists, nurses, social workers, psychiatrists, and mental health paraprofessionals. The professionals are careful not to overlook the natural caregivers in the patient's living environment—for example, peers, relatives, and operators of board-and-care homes. (Vaughn et al. 1982).

There are many patients who for the foreseeable future will remain relatively refractory to skills training and environmental support programs aimed at independent living and full employment. Such individuals can still benefit, however, from rehabilitation. It is possible to improve social adjustment, role performance, and autonomy and quality of life even within the limited opportunities provided by a mental hospital or residential care facility in the community.

Research conducted by a large number of investigators in the United States and Europe supports the following conclusions:

- Severely psychiatrically disabled persons can learn skills.
- The psychiatrically disabled person's skills are positively related to measures of rehabilitation outcome.
- Skill development interventions improve the psychiatrically disabled person's rehabilitation outcome.
- Environmental resource development improves the psychiatrically disabled person's rehabilitation outcome.

In our attempts to conceptualize psychiatric rehabilitation, the "key and lock" analogy might be apt. While it is desirable to maximize an individual's social and instrumental role functioning through training and reeducative procedures, persistent deficits are likely to plague the patient, the family, and the rehabilitation team. Endeavors to upgrade the patient's repertoire of skills hone the "key," but it is often necessary to modify the "lock"—the patient's environment. Recent interventions aimed at modification of the family environment have led to dramatic reductions in relapse, exacerbation, and rehospitalization of patients with schizophrenia (Strachan, this issue). Other examples of environmental prostheses are sheltered workshops, transitional employment, halfway houses, and psychosocial clubs.

Guiding the rehabilitation professional is:

- Optimism that desirable change is possible if principles of human learning can be harnessed to the needs of the patient.
- Belief that motivation for change can come from special arrangements of the patient's rehabilitation and natural environments, as well as from within the patient.
- Confidence that by building upon the patient's assets and interests, including supportive treatment and family environments, even small improvements can lead to significant functional changes and uplift the patient's quality of life.

The principles of rehabilitation have the potential to tie researchers and professionals engaged in psychiatric rehabilitation into a cohesive, empirically based field. Rehabilitation practice has taken root in a variety of settings, including mental health centers and clinics, mental hospitals, general hospitals, psychosocial centers, and community support programs. More than ever before, psychiatric rehabilitation is perceived as a legitimate and credible field of practice, education, and research; as complementary to the existing fields of prevention and treatment; and as a necessary component of mental health system planning and policy-making.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Anthony, W.A. Societal rehabilitation: Changing society's attitudes toward the physically and mentally disabled. *Rehabilitation Psychology,* 19:117-126, 1972.

Anthony, W.A. Psychological rehabilitation: A concept in need of a method. *American Psychologist,* 32:658-662, 1977.

Anthony, W.A. *Principles of Psychiatric Rehabilitation.* Baltimore: University Park Press, 1979.

Anthony, W.A. A rehabilitation model for rehabilitating the psychiatrically disabled. *Rehabilitation Counseling Bulletin,* 24:6-21, 1980.

Anthony, W.A. Explaining "psychiatric rehabilitation" by an analogy to "physical rehabilitation." *Psychosocial Rehabilitation Journal,* 5:61-65, 1982.

Anthony, W.A., and Buell, G.J. Predicting psychiatric rehabilitation outcome using demographic characteristics: A replication. *Journal of Counseling Psychology,* 21:421-422, 1974.

Anthony, W.A.; Buell, G.J.; Sharratt, S.; and Althoff, M.E. Efficacy of psychiatric rehabilitation. *Psychological Bulletin,* 78:447-456, 1972.

Anthony, W.A.; Cohen, M.; and Cohen, B. The philosophy, treatment process and principles of the psychiatric rehabilitation approach. *New Directions in Mental Health,* 17:67-79, 1983.

Anthony, W.A.; Cohen, M.R.; and Cohen, B. Psychiatric rehabilitation. In: Talbott, J., ed. *The Chronic Mental Patient: Five Years Later.* New York: Grune & Stratton, 1984. pp. 213-252.

Anthony, W.A.; Cohen, M.R.; and Vitalo, R. The measurement of rehabilitation outcome. *Schizophrenia Bulletin,* 4:365-383, 1978.

Anthony, W.A.; Howell, J.; and Danley, K. The vocational rehabilitation of the psychiatrically disabled. In: Mirabi, M., ed. *The Chronically Mentally Ill: Research and Services.* New York: SP Medical & Scientific Books, 1984. pp. 215-237.

Anthony, W.A., and Jansen, M. Predicting the vocational capacity of the chronically mentally ill: Research and policy implications. *American Psychologist,* 39:537-544, 1984.

Anthony, W.A., and Margules, A. Toward improving the efficacy of psychiatric rehabilitation: A skills training approach. *Rehabilitation Psychology,* 21:101-105, 1974.

Backer, T.E.; Liberman, R.P.; and Kuehnel, T.G. Dissemination and adoption of innovative psychosocial interventions. *Journal of Consulting & Clinical Psychology,* 54:111-118, 1986.

Ballantyne, R. Community rehabilitation services: A new approach to aftercare. *Network,* 3:4-6, 1983.

Beard, J.H.; Propst, R.N.; and Malamud, T.J. The Fountain House model of psychiatric rehabilitation. *Psychosocial Rehabilitation Journal,* 5:47-59, 1982.

Bennett, D.H. The historical development of rehabilitation services. In: Watts, F.N., and Bennett, D.H., eds. *Theory and Practice of Psychiatric Rehabilitation.* New York: John Wiley & Sons, Inc., 1983. pp. 15-42.

Bockoven, J.S. *Moral Treatment in American Psychiatry.* New York: Springer, 1963.

Braun, P.; Kochansky, G.; Shapiro, R.; Greenberg, S.; Gudeman, J.E.; Johnson, S.; and Shore, M.F. Overview: Deinstitutionalization of psychiatric patients. *American Journal of Psychiatry,* 131:736-749, 1981.

Buell, G.J., and Anthony, W.A. The relationship between patient demographic characteristics and psychiatric rehabilitation outcome. *Community Mental Health Journal,* 11:208-214, 1976.

Cannady, D. Chronics and cleaning ladies. *Psychosocial Rehabilitation Journal,* 5:13-16, 1982.

Carkhuff, R.R. New directions in training for helping professionals: Towards a technology for human and community resource development. *The Counseling Psychologist,* 3:12-20, 1972.

Carkhuff, R.R. *Interpersonal Skills and Human Productivity.* Amherst, MA: Human Resource Development Press, 1983.

Carkhuff, R.R., and Berenson, B.C. *Teaching as Treatment.* Amherst, MA: Human Resource Development Press, 1976.

Carstairs, G.M.; O'Connor, N.; and Rawnsley, K. Organization of a hospital workshop for chronic psychotic patients. *British Journal of Preventive and Social Medicine,* 10:136-140, 1956.

Cohen, M.R.; Danley, K.; and Nemec, P. *Psychiatric rehabilitation trainer packages: Direct skills* *teaching.* Center for Psychiatric Rehabilitation, Boston University, Boston, MA, 1986.

Cohen, B.F.; Ridley, D.; and Cohen, M.R. Teaching skills to severely psychiatrically disabled persons. In: Marlowe, H.A., ed. *Developing Competence.* Tampa, FL: University of South Florida Press, 1983. pp. 96–115.

Dincin, J. A community agency model. In: Talbot, J.A., ed. *The Chronic Mentally Ill.* New York: Human Sciences Press, 1981. pp. 212–226.

Eisenberg, M.G., and Cole, H.W. A behavioral approach to job seeking for psychiatrically impaired persons. *Journal of Rehabilitation,* April/May/June, 46–49, 1986.

Falloon, I.; Boyd, J.; and McGill, C. *Family Care of Schizophrenia.* New York: Guilford Press, 1984.

Falloon, I.R.H.; Doane, J.A.; and Pederson, J. Family versus individual management in prevention of morbidity of schizophrenia: III. Family functioning. *Archives of General Psychiatry,* 42:887–896, 1985.

Farkas, M.; Cohen, M.R.; and Nemec, P. *Psychiatric Rehabilitation Programs: Putting Concepts Into Practice?* Center for Psychiatric Rehabilitation, Boston University, Boston, MA, 1986.

Farkas, M.; Rogers, S.; and Thurer, S. *Rehabilitation Outcome for the Recently Deinstitutionalized Client: The Ones We Left Behind.* Center for Psychiatric Rehabilitation, Boston University, Boston, MA, 1986.

Farr, R.K., and Koegel, P. *A Study of Homelessness and Mental Illness in the Skid Row Area of Los Angeles.* Project Report available from Los Angeles Dept. of Mental Health, 2415 W. 6th Street, Los Angeles, CA 90057, March 1986.

Foy, D.W.; Wallace, C.J.; and Liberman, R.P. Advances in social skills training for chronic mental patients. In: Craig, K.D., and McMahon, R.J., eds. *Advances in Clinical Behavior Therapy.* New York: Brunner/Mazel, 1983. pp. 153–172.

Frey, W.D. Functional assessment in the 80's: A conceptual enigma, a technical challenge. In: Halpern, A., and Fuhrer, M., eds. *Functional Assessment in Rehabilitation.* New York: Brooke Publishing Co., 1984. pp. 11–43.

Goldstrom, I., and Manderscheid, R. The chronically mentally ill: A descriptive analysis from the uniform client data instrument. *Community Support Services Journal,* 2:4–9, 1982.

Grob, S. Psychosocial rehabilitation centers: Old wine in a new bottle. In: Barofsky, I., and Budson, R., eds. *The Chronic Psychiatric Patient in the Community.* Jamaica, NY: SP Medical and Scientific Books, 1983. pp. 265–280.

Harding, C.M., and Brooks, G.W. Life assessment of a cohort of chronic schizophrenics discharged 20 years ago. In: Mednick, I.S.; Harway, S.M.; and Finello, K., eds. *The Handbook of Longitudinal Research.* New York: Praeger Press, 1984. pp. 375–393.

Harrow, M.; Grinker, R.R.; Silverstein, M.L.; and Holzman, P. Is modern-day schizophrenic outcome still negative? *American Journal of Psychiatry,* 135:1156–1162, 1978.

Hawkins, H. A plea for convalescent homes in connection with asylums for the insane poor. *Journal of Mental Science,* 17:107–116, 1871.

Hirschfeld, R.M.A.; Klerman, G.L.; Clayton, P.J.; Keller, M.; McDonald-Scott, P.; and Larkin, B. Assessing personality: Effects of the depressive state on trait measurement. *American Journal of Psychiatry,* 140:695–699, 1983.

Jacobs, H.E.; Kardashian, S.; Kreinbring, R.K.; Ponder, R.; and Simpson, A.R. A skills-oriented model for facilitating employment among psychiatrically disabled persons. *Rehabilitation Counseling Bulletin,* 28:87–96, 1984.

Katkin, S.; Ginsburg, M.; Rifkin, J.J.; and Scott, J.T. Effectiveness of female volunteers in the treatment of outpatients. *Journal of Counseling Psychology,* 18:97-100, 1971.

Katkin, S.; Zimmerman, U.; Rosenthal, T.; and Ginsburg, M. Using volunteer therapists to reduce hospital readmissions. *Hospital & Community Psychiatry,* 26:151-153, 1975.

Kelly, J.A., and Lampariski, D.M. Outpatient treatment of schizophrenics: Social skills and problem-solving training. In: Hessen, M., and Bellack, A.S., eds. *Handbook of Clinical Behavior Therapy With Adults.* New York: Plenum Press, 1985. pp. 485–508.

Lamb, H.R. *Treating the Long-term Mentally Ill.* San Francisco: Jossey-Bass, 1982.

Leff, J.P.; Hirsch, S.R.; Gaind, R.; Rhode, P.D.; and Stevens, B.C. Life events and maintenance therapy in schizophrenic relapse. *British Journal of Psychiatry,* 123:659–668, 1973.

Leitner, L., and Drasgow, J. Battling recidivism. *Journal of Rehabilitation,* July-August, 29–31, 1972.

Liberman, R.P. Social factors in schizophrenia. In: Grinspoon, L., ed. *The American Psychiatric Association Annual Review.* Washington, DC: American Psychiatric Press, 1982*a*. pp. 97–111.

Liberman, R.P. What is schizophrenia? *Schizophrenia Bulletin,* 8:435–437, 1982*b*.

Liberman, R.P. Psychosocial therapies for schizophrenia. In: Kaplan, H.I., and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry.* 4th ed. Baltimore: Williams and Wilkins, 1984. pp. 724–734.

Liberman, R.P., and Evans, C.C. Behavioral rehabilitation for chronic mental patients. *Journal of Clinical Psychopharmacology,* 5:8S–14S, 1985.

Liberman, R.P.; Falloon, I.R.H.; and Wallace, C.J. Drug-psychosocial interventions in the treatment of schizophrenia. In: Mirabi, M., ed. *The Chronically Mentally Ill: Research and Services.* New York: SP Medical & Scientific Books, 1984. pp. 175–212.

Liberman, R.P., and Foy, D.W. Psychiatric rehabilitation for chronic mental patients. *Psychiatric Annals,* 13:539–545, 1983.

Liberman, R.P.; King, L.W.; and DeRisi, W.J. Behavior analysis and therapy in community mental health. In: Leitenberg, H., ed. *Handbook of Behavior Analysis and Modification.* Englewood Cliffs, NJ: Prentice Hall, 1976. pp. 566–603.

Liberman, R.P.; King, L.W.; DeRisi, W.J.; and McCann, M. *Personal Effectiveness: Guiding People to Assert Themselves and Improve Their Social Skills.* Champaign, IL: Research Press, 1975.

Liberman, R.P.; Massell, H.K.; Mosk, M.; and Wong, S.E. Social skills training for chronic mental patients. *Hospital & Community Psychiatry,* 36:396–403, 1985.

Linn, M.W.; Klett, C.J.; and Caffey, E.M. Foster home characteristics and psychiatric patient outcome. *Archives of General Psychiatry,* 41:157–161, 1980.

Livneh, H. Psychiatric rehabilitation: A dialogue with Bill Anthony. *Journal of Counseling and Development,* 63:86–90, 1984.

Medication Management Module. *A Skills Training Program to Teach Patients Reliable Use of Neuroleptic Drugs.* Available from the UCLA Research Center, Box A, Camarillo, CA 93011, 1986.

Monti, P.M.; Corriveau, D.P.; and Curran, J.P. Social skills training for psychiatric patients: Treatment and outcome. In: Curran, J.P., and Monti, P.M., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment.* New York: Guilford Press, 1982. pp. 185–223.

Monti, P.M.; Fink, E.; Norman, W.; Curran, J.P.; Hayes, S.; and Caldwell, A. The effect of social skills training groups and social skills bibliotherapy with psychiatric patients. *Journal of Consulting and Clinical Psychology,* 47:189–191, 1979.

Nelson, G.L., and Cone, J.D. Multiple baseline analysis of token economy for psychiatric inpatients. *Journal of Applied Behavior Analysis,* 12:255–271, 1979.

Nuechterlein, K., and Dawson, M. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin,* 10:300–312, 1984.

O'Connor, R.N., and Tizard, J. *The Social Problems of Mental Deficiency.* London: Pergamon Press, 1956.

Paul, G.L., and Lentz, R. *Psychosocial Treatment of Chronic Mental Patients.* Cambridge, MA: Harvard University Press, 1977.

Pepper, B. The young adult chronic patient: Population overview. *Journal of Clinical Psychopharmacology,* 5:5S–8S, 1985.

Pfohl, B., and Andreasen, N.C. Schizophrenia: Diagnosis and classification. In: Frances, A.J., and Hales, R.F., eds. *Psychiatry Update: The American Psychiatric Association Annual Review.* Vol. 5. Washington, DC: American Psychiatric Press, 1986. pp. 7–24.

Presly, A.J.; Grubb, A.B.; and Semple, D. Predictors of successful rehabilitation in long stay patients. *Acta Psychiatrica Scandinavica,* 66:83–88, 1982.

Rogers, C.R. The necessary and sufficient conditions of therapeutic personality change. *Journal of Consulting and Clinical Psychology,* 21:95–103, 1957.

Royal Commission on the Law *Relating to Mental Illness and Mental Deficiency 1954–1957.* London: Her Majesty's Stationery Office, 1957.

Stein, L.I., and Test, M.A. *Alternatives to Mental Hospital Treatment.* New York: Plenum Press, 1978.

Stickney, S.K.; Hall, R.L.; and Gardner, E.R. The effect of referral procedures on aftercare compliance. *Hospital & Community Psychiatry,* 31:567–569, 1980.

Stockdill, J. *Definition of Psychosocial Rehabilitation.* Technical Assistance Transmittal No. 1—NIMH Office of State & Community Liaison to State Mental Health Directors. Rockville, MD: National Institute of Mental Health, 1985.

Strauss, J.S., and Carpenter, W.T., Jr. *Schizophrenia.* New York: Plenum Press, 1981.

Stubbins, J. *The Clinical Attitude in Rehabilitation: A Cross-Cultural View.* World Rehabilitation Fund Monograph #16. New York: World Rehabilitation Fund, 1982.

Sylph, J.A.; Ross, H.E.; and Kedward, H.B. Social disability in chronic psychiatric patients. *American Journal of Psychiatry*, 134:1391–1394, 1977.

Taylor, C.B.; Liberman, R.P.; and Agras, W.S. Treatment evaluation and behavior therapy. In: Lewis, J., and Usdin, G., eds. *Treatment Planning in Psychiatry*. Washington, DC: American Psychiatric Press, 1982. pp. 151–224.

Tsuang, M.T.; Woolson, R.F.; and Fleming, J.A. Long term outcome of major psychoses. *Archives of General Psychiatry*, 36:1295–1301, 1979.

Turner, J., and TenHoor, W. The NIMH Community Support Program: Pilot approach to a needed social reform. *Schizophrenia Bulletin*, 4:319–348, 1978.

Vaughn, C.E., and Leff, J.P. The influences of family and social factors on the course of psychiatric illness. *British Journal of Psychiatry*, 129:125–137, 1976*a*.

Vaughn, C.E., and Leff, J.P. The measurement of expressed emotion in the families of psychiatric patients. *British Journal of Social and Clinical Psychology*, 15:157–165, 1976*b*.

Vaughn, C.E.; Snyder, K.S.; Freeman, W.E.; Jones, S.; Falloon, I.R.H.; and Liberman, R.P. Family factors in schizophrenic relapse: A replication. *Schizophrenia Bulletin*, 8:425–426, 1982.

Wallace, C.J., and Liberman, R.P. Social skills training for patients with schizophrenia: A controlled clinical trial. *Psychiatry Research*, 15:239–247, 1985.

Wallace, C.J.; Nelson, C.J.; Liberman, R.P.; Aitchinson, R.H.; Lukoff, D.; Elder, J.P.; and Ferris, C. A review and critique of social skills training with schizophrenic patients. *Schizophrenia Bulletin*, 6:42–63, 1980.

Wasylenski, D.A.; Goering, P.N.; Lancee, W.J.; Ballantyne, R.; and Farkas, M. Impact of a case manager program on psychiatric aftercare. *Journal of Nervous Mental Disease*, 173:303–308, 1985.

Weinberger, D.R., and Kleinman, J.E. Observations on the brain in schizophrenia. In: Frances, A.J., and Hales, R.E., eds. *Psychiatry Update: The American Psychiatric Association Annual Review*. Washington, DC: American Psychiatric Press, 1986. pp. 42–67.

Weinman, B., and Kleiner, R.J. The impact of community living and community member intervention on the adjustment of the chronic psychiatric patient. In: Stein, L.I., and Test, M.A., eds. *Alternatives to Mental Hospital Treatment*. New York: Plenum Press, Inc., 1978. pp. 139–159.

Witheridge, T.F.; Dincin, J.; and Appleby, L. Working with the most frequent recidivists: A total team approach to assertive resource management. *Psychosocial Rehabilitation Journal*, 5:9–11, 1982.

Wood, P.H.N. Appreciating the consequences of disease—The classification of impairments, disability, and handicaps. *The WHO Chronicle*, 34:376–380, 1980.

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Applications of Experimental Psychopathology in Psychiatric Rehabilitation

by William D. Spaulding, Lowell Storms, Victoria Goodrich, and Mary Sullivan

Abstract

Persons with schizophrenia show deficits in basic psychological functions such as attention, perception, and cognition. Remediation of these deficits by direct training may facilitate the effectiveness of neuroleptic medications, social skills training, and family therapy. In the vulnerability-stress model of schizophrenia, persons with schizophrenia may have lower thresholds for disorganization that contribute to vulnerability. Stress increases arousal, which brings many competing responses to the same strength, leading to intrusion of inappropriate responses. Interventions that reduce arousal and lower the strengths of competing responses should reduce psychological deficits. Arousal-reducing, attentional, and cognitive interventions are appropriate for the prodromal, acute, and chronic stages of schizophrenic disorders. Laboratory-based assessment and ongoing measurement of basic psychological deficits in schizophrenia are keys to the development and validation of multimodal psychiatric rehabilitation.

The methods and findings of experimental psychopathology can be useful in psychiatric rehabilitation. In experimental psychopathology behavioral disorders and mental illness are understood through laboratory measurement of basic psychological processes such as psychophysiological activation, perception, attention, reasoning, memory, and learning. These processes are often deficient in severely disabled psychiatric patients. Methods of experimental psychopathology can identify patients' psychological deficits and precisely monitor their severity. Laboratory measurement can improve upon traditional methods of clinical assessment and outcome evaluation, and so may lead to more effective use of existing treatment and rehabilitation technologies. An understanding of psychological deficits may also point to innovative treatment procedures for their remediation.

Application of the methods and principles of experimental psychopathology promises to fill some of the current gaps in psychiatric rehabilitation technology. Pharmacological treatment often reduces the most dangerous and disruptive behavioral symptoms, and restores some basic psychological functions; but many psychological deficits are not remediated by drugs. Also, a significant proportion of severely disordered patients do not respond at all to drugs. Procedures such as social skills training contribute substantially to rehabilitation success, but they do not directly address psychological deficits. Broader application of experimental psychopathology might increase the overall effectiveness of psychiatric rehabilitation.

Our discussion focuses on techniques developed for assessment and treatment of schizophrenia. Schizophrenia is the diagnosis of the plurality of psychiatric rehabilitation patients, and it has been a nosological keystone in experimental psychopathology research. Note, however, that no specific psychological deficit or group of deficits is *unique* to schizophrenia. Other psychotic and severely disordered patients have the deficits found in

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patients with schizophrenia. Moreover, the heterogeneity of schizophrenia is highlighted by the finding that even within the category of schizophrenia, patients have different constellations of deficits. There is no reason to expect that any particular treatment should be effective for all members of any diagnostic group. The current state of knowledge demands that individual patients be presumed to have unique constellations of psychological deficits. The applicability of any intervention to any patient is thus an empirical question.

There are three prerequisites for effectively using psychological assessment and intervention for rehabilitation: (1) a conceptual model that organizes and describes in general terms the possible relationships between biological, psychological, and social deficits; (2) a repertoire of assessment methods to provide objective and quantitative measurement of the deficits; and (3) an armamentarium of intervention techniques that specifically addresses psychological deficits. A guiding conceptual model can be synthesized from the convergence of several contemporary views of the etiology of schizophrenia. For assessment, a wide range of relevant measurement methods can be adapted from the experimental psychopathology laboratory. Recent developments in cognitive-behavior therapy provide many potentially useful intervention techniques.

Theoretical Foundations

A conceptual framework is valuable in the design of rehabilitation strategies if it can stimulate working hypotheses, organize what is known about the etiology and course of schizophrenia, and suggest possible relationships between specific psychological deficits and remedial interventions.

A model meeting these criteria was originated two decades ago (Broen and Storms 1966; Storms and Broen 1969). It integrated much of what was then known about schizophrenia, and as new understandings emerged, they were successfully incorporated with minor modifications of the original model. The contemporary version, the *hierarchy-collapse model*, explicitly incorporates two important psychopathological principles of schizophrenia not emphasized in the original version: the pervasiveness of schizophrenia, and the role of diathesis-stress in its etiology.

The idea of the pervasiveness of schizophrenia represents a convergence of several approaches to psychopathology. In ego psychology, schizophrenia has been conceptualized as severe deficits across a range of ego functions (Bellak, Hurvich, and Gediman 1973). In "medical" models, it can be seen as a final common pathway at social-behavioral levels of functioning whose tributaries extend to biological levels (e.g., Strauss and Carpenter 1981). In behavioral psychopathology, schizophrenia represents deficits across a variety of response systems (Adams, Doster, and Calhoun 1977), or across levels of functioning (Spaulding 1986). Models based on general systems theory (Scheflin 1981) and models of human occupation (e.g., Kielhoffner, Burke, and Igi 1980) describe disorganization of adaptive subsystems. All these use some version of the concept of "levels of functioning"; that is, the idea that molar social behavior represents the integration of specific processes that operate at more molecular cognitive, attentional, perceptual, and psychophysiological levels. From this perspective, schizophrenia can be characterized by deficits at all these levels. Although there is agreement that deficits are pervasively distributed in schizophrenia, the causal relationships between deficits are incompletely understood.

The diathesis-stress view of schizophrenia hypothesizes two interacting etiological factors. A diathesis, or vulnerability, is a deficit that exists before the onset of illness and predisposes an individual for the illness. Stress is the body's reaction to an environmental event or series of events that place a demand on the person. Stress interacts with the diathesis, either at a particular time or over the course of development, to precipitate illness. Like pervasiveness, the idea of diathesis-stress represents the convergence of a diversity of theory and data (Meehl 1962; Zubin and Spring 1977; Cromwell and Spaulding 1978; Liberman, Nuechterlein, and Wallace 1982; Nuechterlein and Dawson 1984*a*). A corollary of the stress-diathesis view is that schizophrenia occurs in at least two phases. In one, a vulnerable state exists, represented by a particular pattern of enduring psychobiological deficits. In the second phase, these predisposing deficits are unlocked by stress to produce the illness state, represented by characteristic symptoms, associated disabilities, and even more severe basic deficits.

Response-Hierarchy-Collapse Model. A useful theory of schizophrenia should explain how vulnerability is translated by stress into behavioral disabilities and symptoms. Most psychological theories of schizophrenia attempt to characterize the basic nature of schizophrenic deficits, but they do not specify or predict how such deficits might be produced or alleviated—perhaps explaining why research on these theories has led to so little in the way of therapeutic interventions.

In their comprehensive reviews of psychological research in schizophrenia, Buss and Lang (1965) and Lang and Buss (1965) concluded that interference theories are most consistent with the accumulated evidence. However, most such theories do not specify how interference with cognitive functions is increased and decreased. In this light, it is interesting that Lang and Buss (1965) also stated that interference theories that include arousal are promising. In a review of more recent research, Nuechterlein and Dawson (1984*b*) conclude that a major feature of vulnerability to schizophrenia is reduced information-processing capacity, especially when higher demand and interference are present.

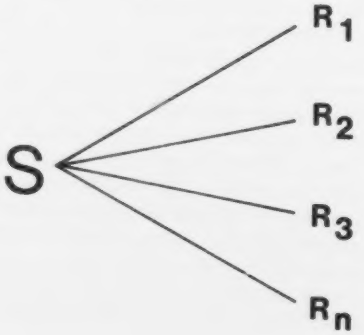
The response-hierarchy-collapse model of Broen and Storms (1966) and Storms and Broen (1969) suggests how arousal, which is increased by demand-induced stress, can produce response interference. It also suggests ways of reducing response interference for therapeutic effect. As an example of the kind of thinking disturbance most characteristic of schizophrenia, Arieti (1959) reports that a person with schizophrenia who meant to refer to the steering wheel of a car said "handlebars" instead. Let us see how the model can explain such associative intrusions.

Response hierarchy. The hierarchy-collapse model is organized around the concept of a response hierarchy and hypothesizes ways in which arousal and response strength ceiling interact to affect response hierarchies.

In a given situation, there are within a person a number of simultaneous response tendencies that collectively can be termed a "response hierarchy," similar to Hull's (1952) "habit family hierarchy." Competing response tendencies occur at different strengths, and these strength relationships constitute the organization of the hierarchy. In figure 1, S represents the stimulus situation which includes both external and internal stimuli, and R_1-R_n represent response tendencies at different strengths. Imagine that S represents the situation in which Arieti's patient meant to refer to a car's steering mechanism. R_1 represents the dominant linguistic response "steering wheel" and R_2 the response "handlebars." Further concepts are needed to account for the occurrence of R_2.

Arousal. This is the concept of generalized arousal, which includes the influence of the reticular activating system (Magoun 1952), especially its activation of response tendencies (Hebb 1955; Malmo 1959). Because cortical arousal is accompanied by autonomic arousal, psychophysiological measures of autonomic activation can index arousal (Dawson and Nuechterlein 1984). Increased arousal is assumed to lead to increased strength of all coexisting response tendencies. Arousal can be varied in a number of ways, such as by varying muscle tension or psychological stress. Arousal can be highly situational; a patient of one of the authors (L.S.) who had been hospitalized for schizophrenia found going to a nightclub with his girlfriend stressful. This increased his arousal and was associated with other problems in his psychological functioning.

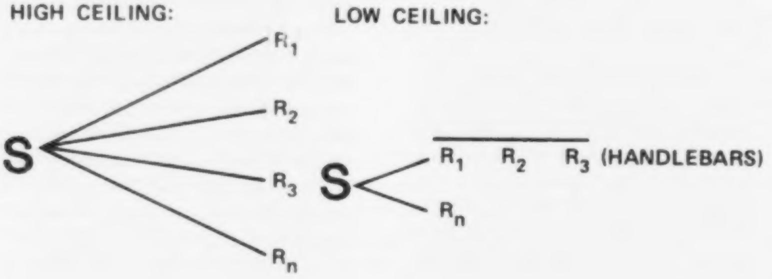
Figure 1. Response hierarchy associated with a particular stimulus situation



Response-strength ceiling. Broen and Storms (1966) have proposed an upper limit to response strengths that may be reached at less than maximal arousal. A necessary additional hypothesis is that there are individual differences in response strength ceiling, and schizophrenics have an enduring tendency to have lower ceiling levels than nonschizophrenics. We hypothesize that the patient in the nightclub had a lower R ceiling and was thus more vulnerable to disorganization than people not suffering from schizophrenia.

Figure 2 illustrates what happens to a hierarchy with high ceiling or low ceiling after an increase in arousal. At the lower ceiling, increased arousal raises the dominant response in the hierarchy to ceiling, and further increases tend to bring other responses to ceiling, equalizing their strengths. This partial collapse of the hierarchy leads to interference from responses lower in the hierarchy, which become as strong as the dominant response. In figure 2, the response "handlebars" becomes as strong as "steering wheel" and as likely to occur. The hierarchy with the low ceiling has become partially disorganized or collapsed. Thus, the lower the ceiling, the greater the vulnerability to disorganization—or the greater the vulnerability to schizophrenia.

Figure 2. Partial collapse of a response hierarchy



Note that while the responses in the hierarchy have become partially randomized, inappropriate responses come from the response tendencies in the individual's hierarchy. The responses are meaningfully related to the stimulus situation because of past learning. Wide individual differences in learning histories may affect responses lower in the hierarchies more than those at the top. The content of the errors and intrusions would be expected to vary greatly from one person with schizophrenia to another. Thus, the behavior of schizophrenics should be very different from one individual to the next, while the underlying process is the same.

The patient in the nightclub had a recurrence of voices saying things like, "you're no good" and "get out of here." The voices probably arose from memories or thoughts initially low in a hierarchy but made as vivid as perceptions by partial randomization of the hierarchy. Changing him to a more effective neuroleptic eliminated the voices, possibly by reducing arousal so that the hierarchy could reorganize.

The partial randomization or collapse of hierarchies seems to be described by a number of authorities on schizophrenia. Bleuler (1950) described schizophrenics as responding as if "ideas of a certain category are thrown into one pot, mixed, and subsequently picked out at random" (p. 16). The "certain category" is analogous to a response hierarchy. Freeman (1960) described schizophrenics as "unable to organize and subordinate the events occurring simultaneously in their organisms" (p. 934). This includes perceptions as well as thoughts. Shakow (1962) spoke of irrelevant associations "floating around on top of the barrel" (p. 9).

The collapse of many response hierarchies in an acute psychosis is an extremely aversive experience. The person is highly motivated to prevent its recurrence by avoiding high levels of arousal and high levels of response interference. Different individuals have different learning histories and different skill repertoires, so they are likely to develop different strategies for avoiding collapsed hierarchies. Some of these strategies may be effective but may also interfere with other behavioral processes. Also, reorganization of the hierarchy itself after its collapse may be different from the original hierarchy. The new organization may be dysfunctional with respect to the effectiveness of behaviors left at the top of the hierarchy.

Storms and Broen (1969) show how collapsed response hierarchies produced by arousal can account for many phenomena and pervasive deficits of schizophrenia, including associative disturbances, conceptual disturbances, hallucinations, delusions, disturbances of attention, autistic behavior, and regression. The key to this range of applicability is the presence of response hierarchies at all levels of psychological functioning. At the psychophysiological level, hierarchies regulate autonomic and central nervous system activation. At the perceptual and attentional levels, hierarchies are involved in selection of input channels, deployment of attention, and selection and activation of stimulus-processing modalities. At the cognitive level, hierarchies are involved in concept formation, processing of memory information, and selection and activation of complex behavioral responses. Acute hierarchy collapse and/or maladaptive reorganization can produce deficits in any or all of these.

The applicability of the hierarchy collapse model across levels of functioning anticipates the recent emphasis on the pervasiveness of schizophrenic deficits. It also complements diathesis-stress models of schizophrenia.

Diathesis-stress and collapsed hierarchies. The hierarchy-collapse theory complements vulnerability-stress models by specifying how vulnerability is converted by stress into the phenomena of schizophrenic psychosis. A detailed vulnerability-stress model is presented and diagrammed by Liberman, Nuechterlein, and Wallace (1982) and Nuechterlein and Dawson (1984*a*). In this model, vulnerability includes reduced information-processing capacity, autonomic hyperreactivity, and social coping deficits. Social stressors interact with vulnerability to produce transient intermediate states of information-processing capacity overload, hyperarousal, and deficient processing of social stimuli. These transient intermediate states result in the observable prodromata and florid symptoms of schizophrenia.

In the response-hierarchy-collapse model, lowered response-strength ceiling leads to lowered information-processing capacity. Sensitivity to hyperarousal may be present. Social coping deficits would be reflected in response hierarchies with strong competing responses in social situations and could add to basic vulnerability. Social stressors would increase arousal and would thus bring some dominant responses and competing responses to ceiling, resulting in a partial collapse of response hierarchies. This would certainly be a form of processing-capacity overload that is especially likely with hyperarousal. Deficient processing of stimuli, especially complex social stimuli, would result. The transient intermediate state of collapsed hierarchies would result in symptomatic behaviors.

The hierarchy-collapse model adds specificity to the general vulnerability-stress model, leading to more detailed predictions. For example, high-demand (stress arousal), high-difficulty (i.e., much response competition) situations would be predicted to lead to partial collapse of some hierarchies and reduced performance, which can be taken as a reflection of vulnerability to schizophrenia (Nuechterlein and Dawson 1984*a*). The hierarchy-collapse model predicts not only that reductions in arousal will be of benefit, which is implied by the general vulnerability model, but also that increased clarity, structure, and redundancy of stimulus input and increased informative feedback about response consequences will be helpful due to reduced response competition.

Treatment Applications

The hierarchy-collapse model suggests three possible strategies for reducing psychological deficits, corresponding to the three parameters of the model. One is to raise the response-strength ceiling; i.e., to elevate the threshhold for disorganization so that the degree of psychophysiological arousal that produced hierarchy collapse no longer does so. A second strategy is to reduce arousal from the level that produces acute disorganization, allowing the hierarchy to reorganize itself. A third is to provide environmental conditions and learning experiences that favor organized hierarchies.

Raising Response Strength Ceiling. The response-strength ceiling presumably has a neurophysiological basis. Neuroleptic medications may help raise response strength ceiling. Influences such as alcohol, drugs, or improper diet may compromise the integrity of the nervous system and thus lower the ceiling. Correction of these problems could help raise the ceiling. Methods of orchestrating drug-psychosocial treatment interactions to improve the response strength ceiling and protect against the noxious combination of stress and vulnerability in schizophrenia have been reviewed by Falloon and Liberman (1983).

Regulation of Arousal. Administration of antipsychotic drugs clearly has an arousal-reducing effect. However, the therapeutic psychological effects of antipsychotic drugs are poorly understood and rarely measured. In clinical practice, evaluation of drug effects depends on observations of overt behavior and subjective report, although psychological effects are often inferred from these observations. The well-known latency between administration of drugs and their antipsychotic effects may be understood in the hierarchy-collapse model as the time required for the molar response hierarchies, which mediate social behavior and report of subjective experience, to reorganize. Reorganization of more molecular response hierarchies may require less time; thus, molecular psychological functioning may improve before clinical improvement is evident.

In one of the more comprehensive studies of drug effects on psychological deficits, Spohn et al. (1977) found that molecular attentional processes associated with simple vigilance and rapid processing of visual features improve with antipsychotic drug treatment long before clinical improvement is evident. There was a general monotonic relationship between improvement in the attentional processes and reduction of psychophysiological arousal. There was also a nonsignificant trend toward improvement in more molar psychological functions (abstraction and reasoning), suggesting that eventually they too would have shown an effect, possibly secondary to normalization at the more molecular levels.

The Spohn et al. (1977) findings are complemented by a number of more focal studies of drug effects on psychological deficits (e.g., Held et al. 1970; Kornetsky 1972; Oltmanns, O'Hayon, and Neale 1978; Zahn, Carpenter, and McGlashan 1981; Braff and Sacuzzo 1982; Strauss et al. 1985). Taken together, they suggest that: (1) some molecular psychological deficits show drug effects before clinical improvement is evident; and (2) some psychological deficits are not affected by drugs at all. Systematic laboratory assessment should therefore help predict longer-term clinical improvement and help articulate the precise therapeutic effects contributed by the drug.

Drugs are not the only means of directly reducing arousal. The success of a highly structured inpatient program in treating acutely psychotic patients without drugs (Carpenter, McGlashan, and Strauss 1977) suggests that, at least in some cases, therapeutic psychosocial interactions can achieve the same result. It has long been known (Azima and Kramer 1956; Reitman and Cleveland 1964) that reducing arousal by limiting sensory input can have therapeutic effects on acutely psychotic patients.

In the prodromal and postacute phases, patients can be taught to manage their own arousal. Zeisset (1968) demonstrated that relaxation training, a technique designed to reduce arousal through systematic induction of muscular relaxation, could produce symptomatic improvement in patients with schizophrenia. Since then, relaxation training has become a popular modality in service programs for severely disordered psychiatric patients.

A case supervised by one of the authors (W.S.) illustrates the impact of a simple relaxation intervention:

> The patient was a 26-year-old man with a long history of schizophrenia, psychiatric hospitalizations and failures in rehabilitative efforts. Even when optimally medicated, he was visibly tense and anxious. His arousal became more pronounced in minimally stressful social interactions. His social behavior was punctuated in such situations by frequent "tantrums," which sometimes included threats, physical assaults, and incoherence. He saw himself as a "hopeless case," out of control of himself and his environment, a belief shared by mental health providers and others.
>
> The patient was taught standard relaxation training techniques. He responded unusually well, and reported practicing the exercises even more frequently than was prescribed. He came to see the exercises as one acceptable thing he could do to "feel good." He also readily accepted a suggestion that his success with the exercises showed he was potentially more in control than he had believed. His self-esteem and motivation to participate in rehabilitation activities improved accordingly. He began to perceive correctly that he was more effective in interpersonal interactions when not extremely aroused. His improved social performance had a significant impact on clinicians, who became more willing to work with him and tolerate his other behavioral deficits.

Desensitization techniques incorporate relaxation training and use conditioning principles to reduce fear, anxiety, or anger. The result is a reduction of psychophysiological arousal in the presence of stimuli that previously elicited those emotions. In patients with phobias and other anxiety disorders, desensitization effects are sometimes relatively specific; a fear and/or avoidance response in a specific stimulus situation is changed. In schizophrenic patients, desensitization effects may be more generalized. Slade (1972) used systematic desensitization to a series of family scenes (e.g., talking to mother, arguing with father) with a hallucinating patient, and found improvements in self-rated mood measures, as well as a reduction in hallucinations. Similar results have been achieved with a variety of related techniques, including *in vivo* desensitization (Alumbaugh 1971), implosive therapy (Hogan 1966), and induced anxiety (Friedberg 1969; Cockshott 1971).

There is one report of negative results using systematic desensitization with persons with schizophrenia (Serber and Nelson 1971). Desensitization techniques and other approaches that require attention and cooperation may be difficult for many persons suffering from schizophrenia. Attention and/or motivation may be deficient, so the approach may have to be adapted to the patient. For example, briefer sessions, continuous monitoring and encouragement of imagery, and rewards such as points used in a token economy may increase success.

The hierarchy-collapse model suggests that several things may be happening during relaxation training, desensitization, and other related techniques. First, the patient may be learning or reorganizing basic arousal-regulation skills. In the prodromal phase of schizophrenia this would moderate arousal and make more information-processing capacity available for coping with stress. Second, the arousal-producing potential of specific situations would be reduced, permitting better coping with those situations, reducing the danger of hierarchy collapse, and eliminating the need for avoidance.

Biofeedback may prove to be a useful adjunct to desensitization and arousal-reduction techniques in schizophrenics. Although biofeedback does not add significantly to the effects of relaxation training in less severely disordered patients (Budzynski 1977), it appears to be more important with lower functioning patients (Small, Giganti, and Steinberg 1978; Ford et al. 1982). A recent report (Schneider and Pope 1982) raises the intriguing possibility that biofeedback can produce changes in patients' brain activity analogous to those produced by antipsychotic drugs. Further research may confirm that specific psychophysiological abnormalities associated with the etiology of schizophrenia can be mitigated with psychological interventions.

Collections of specific techniques, including desensitization and biofeedback, have been brought together in packages generically termed "stress management" (Smith 1980; Woolfolk and Lehrer 1984). The goal of stress management is to teach the patient to reduce the effects of stress and also to help the patient create a less stressful environment. The concept of stress management complements a diathesis-stress view of schizophrenia. Psychophysiological processes are known to be involved in the relationship between family stress and recidivism in schizophrenic patients (Tarrier et al. 1979). Interventions such as social skills training and family management have components that clearly address stress management skills.

There is one report of a systematic attempt to use a stress-management package in the treatment of patients with schizophrenia (Lukoff et al. 1986). In this study, daily yoga and exercise sessions, daily meditation sessions, twice weekly stress education sessions, weekly positive imaging sessions, sessions to encourage acceptance of psychotic experiences, and sessions on building self-esteem were combined in a "holistic health" program. After 9 weeks of treatment, this group displayed at least as great and possibly greater improvement on a variety of measures of psychopathology as a comparable group trained in social skills, while the social skills group showed greater improvement in social functioning. All subjects were also treated with neuroleptic medications and a token economy.

Hierarchy-Organizing Treatment Approaches. When acute disorganization is present, reducing competing responses allows some reintegration of hierarchies (thus, reduction of confusion). With appropriate as well as inappropriate responses at ceiling for many hierarchies, it is more important to reduce the competing, inappropriate responses by minimizing distractions, providing a highly structured environment, giving explicit, repetitive instructions, and offering clear feedback about the consequences of behavior than it is to reward appropriate responding (Storms and Broen 1969; Liberman, Nuechterlein, and Wallace 1982).

With the greater stability of the postpsychotic (or prepsychotic) phase, individuals with schizophrenia can be taught strategies and skills for organizing and protecting the organization of their own hierarchies. They can learn basic attentional skills to reduce competing responses—how to ignore distractions and focus on task-relevant internal and external stimuli. They can learn to structure their physical environment and their time. With attentional deficits decreased, processing at the cognitive level is facilitated. Persons with schizophrenia can learn problem-solving skills. They can learn to discriminate fantasy from reality, and internal from external cues. These skills can help them compensate constructively for their vulnerability to deficits in situations characterized by complexity and high demand.

Liberman, Nuechterlein, and Wallace (1982), at the UCLA Clinical Research Center for Schizophrenia and Psychiatric Rehabilitation, have developed practical training procedures that emphasize the clear input and feedback endorsed by the hierarchy-collapse model:

- Keep the training setting uncluttered and devoid of distracting stimuli.
- Post graphic charts for clear and simple visual cueing of cognitive strategies.
- Use mild censure contingent on inappropriate responding as well as praise for appropriate responding.
- Conduct task analysis and break down tasks into simple steps.
- Decrease novelty by many repetitions before moving to new material or scenes.

Attentional Interventions. In the terminology of the hierarchy-collapse model, attentional deficits that do not respond to psychophysiological normalization should be understood as the result of maladaptive reorganization of a response hierarchy. Treatment should therefore aim to reestablish a more adaptive organization, through environmental structure, enriched informational input, and feedback on performance. This can be accomplished through specially structured practice on tasks that put specific demands on attentional functioning.

Meichenbaum and Cameron (1973) developed a method for strengthening attentional functioning that emphasized use of self-instructions during task performance. Patients were taught to "talk to themselves," first vocally and later covertly, while performing attention-demanding tasks. Self-talk included instructions to keep attention focused on the tasks and resist distraction, and self-reinforcing statements. Meichenbaum and Cameron (1973) demonstrated treatment effects in such diverse areas as psychiatric status, psychomotor performance, projective test performance, and interview behavior. Subsequent studies of self-instructional training with severely disordered patients yielded mixed results. In a review of these studies, Lowe and Higson (1981) conclude the bulk of the evidence supports its efficacy, provided the self-instructional set is highly individualized, constructed after a careful idiographic assessment of each patient's attentional and cognitive difficulties.

Of course, since self-instructional sets address a diversity of attentional and cognitive functions, the specific nature or role of attentional changes in self-instructional training cannot be determined. Cueing increases use of normal processes, activating hierarchies with salient dominant responses and reduced response competition. Increased use of those processes may also reorganize hierarchies in the attentional response system, normalizing deficits. Whatever the case, self-instructional training does improve patients' attentional performance, and this facilitates performance at behavioral and social levels (Lowe and Higson 1981).

Attentional deficits were more directly treated in a patient with schizophrenia in a case reported by Adams et al. (1981). Several laboratory "distractibility" tasks from experimental psychopathology research were adapted for clinical use. The tasks were treated as exercises rather than tests, although they also yielded longitudinal measures of the patient's performance. Among other things, the patient practiced attending to recorded conversations while ignoring visual, auditory, or conversational distractions. Memory recall under distraction conditions was also practiced. The patient's performance on the laboratory measures improved with practice. At termination and 6-month followup, the patient also showed reduced thought disorder, increased social effectiveness, and improved mood.

In a case supervised by one of the authors (W.S.), the effectiveness of the above procedure was replicated:

> The patient was a 25-year-old mother of one who was undergoing a stressful separation from her husband. She had been found by friends huddled in a closet with her child. She complained of hearing the "voice of the devil," and believed the devil was after the child. Before referring herself for psychiatric services, she underwent an "exorcism" at the suggestion of friends who belonged to a fundamentalist religious group. She reported later that the "exorcism" had relieved her of the belief that the devil was about to take her child, but the voices continued unabated and she still experienced extreme anxiety.
>
> When first seen for psychiatric services, the patient was put on a low-dose regimen of haloperidol. She reported the drug improved her sleep cycle, blunted her anxiety, and diminished the voices somewhat. She also reported the content of the voices had changed from deprecation and threats to "just voices." After she had reached a point of optimal drug response, the voices were still a significant problem. She reported she could sometimes engage in stressful activities, but that usually the voices became too intrusive and distracting to continue. An attentional training procedure was adapted from the "dichotic listening" paradigm (e.g., Wishner and Wahl 1974) of experimental psychopathology. The patient practiced listening to tape-recorded readings while a distraction tape also played. After only two sessions of the training procedure, the patient was able to perform the task without error. She reported she was able to "disattend" from her hallucinated voices the same way she could disattend from the distraction tape. She was instructed to use this disattention tactic whenever the voices became intrusive. The patient reported success in *in vivo* situations, and showed continuing improvement in her ability to deal with stress without being disabled by hallucinations. The haloperidol was gradually discontinued without a return of psychotic symptoms.

Another case illustrates the significance of attentional deficits that remain after acute psychosis and their potential role in preventing relapse and recidivism:

> A patient of one of the authors (L.S.), who had recovered from a schizophrenic episode, reported making serious mistakes in her housework, which brought disapproval from her scholarly physician husband. Analysis of the problem suggested her attention was frequently disrupted by distraction produced by her own distressing thoughts, resulting in faulty analyses of situational demands (a desire for her husband's attention may also have played a part). Her distraction problem was corroborated by poor performance on subtests of the Wechsler Adult Intelligence Test known to be affected by distractibility: Block Design, Picture Arrangement, and Digit Span. With coaching by the therapist, the patient practiced standard psychological tests which demand continuous attention and resistance to distraction: the Wechsler Intelligence Scale for Children Block Design and the Minnesota Paper Form Board. She improved significantly on all the tests, and she reported that her mistakes at home were markedly reduced. Conflict between the patient and her husband was also reduced.

One aspect of attentional functioning that has particular relevance to rehabilitation is continuous work performance, or the ability to maintain continuous attention and activity in a complex task. Individuals who cannot maintain continuous work performance for more than a few minutes are poor candidates for vocational training of any kind.

Continuous work performance is a relatively molar function, requiring vigilance, resistance to distraction, and repetitive use of instructions held in short-term memory. Severely disordered patients have deficits in all these specific functions, and they are notoriously poor at continuous work performance. Little is known, however, about how continuous work performance might be improved through therapy.

A therapeutic approach to continuous work performance was investigated in an exploratory study at the Lincoln (Nebraska) Regional Center. The treatment is part of a systematic approach to occupational therapy, designed to prepare chronic institutionalized patients for vocational rehabilitation in a sheltered workshop. Establishment of adequate continuous work performance is the first of several steps through the therapy sequence.

Nine subjects were selected for the study from a pool of 136 psychiatric inpatients. The nine selected were not distinct from the others with respect to quality or severity of symptomatology, or chronicity, but they were the worst of the pool on a simple laboratory measure of work-related attention, the Continuous Work Performance Test (CWPT). The CWPT consists of several paper-manipulation tasks: folding, cutting, stapling, unstapling, or sorting. Only one task is used in a single work session; the tasks are rotated across sessions to avoid monotony. The CWPT score is simply the number of minutes the trainee performs the task without becoming distracted or otherwise stopping work. All nine of the patients selected for special training had CWPT scores of less than 5 minutes.

Training sessions were held three times per week, 30 minutes per session. A CWPT goal was set at the beginning of each session, with a therapist/trainer prompting maintenance of attention, and providing encouragement, performance feedback, and praise for effort. The ultimate goal of the training was to achieve a CWPT score of 30 minutes for five continuous sessions. At that point the trainee graduated to a more advanced training regimen involving increasingly complex tasks.

Of the original nine patients, seven reached the criterion for graduation and continued to progress in the subsequent stages of vocational training. One reached the criterion after 4 weeks of training but suffered a severe psychotic exacerbation shortly thereafter and was transferred to a more intensive care ward. The ninth patient showed steady clinical deterioration over an 8-month period and finally had to be transferred. Despite her deteriorating condition, she did manage to increase her CWPT score from 5 to 10 minutes in 8 months.

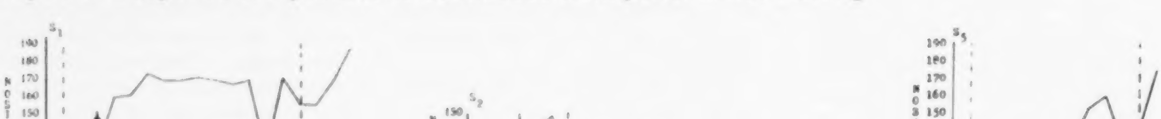
Figure 3 shows the progress of the seven successful trainees. Their CWPT scores are shown along with Nurses Observation Scale for Inpatient Evaluation (NOSIE-30) scores so that their workshop performance can be compared with their global psychiatric status. Inspection of figure 3 reveals that the seven patients all showed different longitudinal responses to the continuous work performance training. The least amount of training required was 4 weeks, or 12 sessions (subject 4). The most was 84 weeks, or approximately 252 sessions (subject 3). Five of the subjects showed a net improvement in NOSIE-30 ratings during the training period. In two of those, there was a relatively parallel, monotonic increase in both NOSIE-30 and CWPT (subjects 5 and 6). For these two, the hypothesis that CWPT improvement was secondary to general improvement in psychiatric status rather than a specific training effect cannot be ruled out. For subject 6, however, the continuous work performance training was the only new intervention, introduced after his NOSIE-30 baseline had been stable for at least 10 months. It is more likely that for subject 6 the training produced a generalized change in clinical status. The other three subjects who had a net NOSIE-30 increase (subjects 1, 2, and 3) showed relatively steady CWPT improvement while their NOSIE-30 scores showed nonmonotonic fluctuations. For these three patients, CWPT progress does not appear to be correlated with improvement in symptoms or other areas of social and personal functioning. Such independence of therapeutic effects is now a well-established finding in research on schizophrenia (Strauss and Carpenter 1981).

This exploratory study demonstrates four important points: (1) laboratory measures can detect relevant psychological deficits in patients whose clinical status does not distinguish them from patients without the deficits; (2) the attentional deficits associated with continuous work performance can be improved through psychosocial intervention; (3) patients show highly individualized responses to treatment; and (4) improvement of the psychological deficit is associated with improvement of clinical status in some patients, but not others. All of these point to the value of laboratory measurement of psychological functioning in selecting and sequencing skill-training interventions, and in evaluating progress and outcome.

Another area of rehabilitation where attentional deficits are particularly relevant is social skills training (SST). As we mentioned earlier, investigators in this area have expressed concern that some of schizophrenia's most significant characteristics are not addressed in skills training (Liberman, Nuechterlein, and Wallace 1982). Attentional deficits are assumed to be limiting factors in social skills acquisition in a group setting. Shuart (1985) tested the effects of an attentional training procedure on performance in a five-patient SST group. The subjects' daily performance during SST sessions was assessed through observational scoring of videotapes and quizzes administered at the end of each session. The rating scales were designed to assess observable behavior associated with attention (alert posture, eye contact). The quizzes assessed subjects' assimilation and retention of material presented and rehearsed in the SST session. After an initial baseline period, subjects received two sessions per week of either an attention-enhancing self-instructional training regimen similar to that developed by Meichenbaum and Cameron (1973) or a "placebo" procedure (semistructured conversation about the group sessions).

Attentional functioning showed a significant treatment effect. On the post-SST-session quizzes, four of the five subjects showed an improvement over placebo baseline when the attentional treatment was introduced. There was a significant rank-order correlation ($r = .90$; $p < .05$) between absolute level of quiz performance at the end of the study and number of actual attentional treatment sessions (the latter was different for each subject). Observa-

Figure 3. Response of 7 patients to continuous work performance training



The NOSIE-30 (Nurses' Observational Scale for Inpatient Evaluation) (Honigfeld, Gillis, and Klett 1966) is a 30-item behavioral checklist filled out by ward staff after at least 3 days of patient observation. The figure shows monthly averages of the NOSIE-30 Total Assets Scale; the data were collected weekly by ward staff. Arrows indicate change in contingency management program for subject 1, and drug titration for subject 3.

tional ratings of attentional behavior made from videotapes of the SST sessions also showed significant improvement, although the effect was not strong enough to differentiate treatment from placebo effects.

Cognitive Interventions. Cognitive interventions that facilitate the organization of response hierarchies fall into two subcategories, process-oriented and content-oriented. Content-oriented interventions attempt to change the content of key thought processes. Often they focus on attributions about the self, such as ability to be successful or current level of functioning. Well-known techniques for modifying cognitive content have been developed to treat depression (e.g., Beck 1976). Severely disordered patients often have depression-related attributional patterns, but it is not known whether they would also respond to such treatments.

The Adams et al. (1981) treatment described earlier included a cognitive content intervention that addressed a more typically schizophrenic attribution. The patient was prone to attribute unsuccessful social interactions to persecutory intentions of other people. As the patient's social skills improved, these failures were reinterpreted as a result of formerly deficient skills. Successful interactions began to occur as a result of the improved skills, and this further reduced attributions of persecutory intent.

Patients' attributions about their own psychophysiological and perceptual experiences can be an important factor in determining how disabling those experiences are. For example, a patient with auditory hallucinations may believe that they are the result of a brain disorder, or may believe that they are the voice of Satan. The attributional difference could have a significant impact on how much the hallucinations interfere in rehabilitation activities. Johnson, Ross, and Mastria (1977) report a case in which misattributions of proprioceptive sensations produced the "delusional" belief that the patient was having sex with a supernatural "warm form." Observation revealed that the patient was generating the sensations himself. In a primarily didactic intervention, the patient was counseled to accept masturbation as normal and acceptable, and thus to understand his mysterious orgasms. This reformulation eliminated his delusional belief. Various strategies for modifying patients' problematic attributions are described by Valins and Nisbett (1971).

Self-instructional sets such as those developed by Meichenbaum and Cameron (1973) can also be categorized as modification of cognitive content. In addition to cues for maintaining attention, the sets include self-reinforcing comments and reassurances against frustration when failure occurs. These are presumably elements in most people's self-instructional sets, albeit unconscious or taken for granted. Their effectiveness in improving patients' performance can be understood in the hierarchy-collapse model as the result of enriched input. The self-instructions cue a variety of necessary responses in the psychophysiological, attentional, and cognitive levels of functioning. In other words, they change the stimulus situation to produce hierarchies with strong (and appropriate) dominant responses and weak competing responses. Without cues, these responses are vulnerable to interference from competing responses. In the case of self-instructions, the patient provides the input enrichment.

A relatively molar cognitive process is that described by the ego-psychological term "reality testing" (Bellak, Hurvich, and Gediman 1973). Reality testing is a complex collection of cognitive activities that includes sampling perceptual data, comparing it with information in memory, and devising plans to resolve discrepancies. Delusions and hallucinations can often be understood as the result of faulty reality testing, especially when perceptual data are distorted by psychophysiological or attentional functions. Shemburg and Leventhal (1972) described several cases wherein reality testing was augmented by procedures which clearly constitute input enrichment. In one example a patient reported seeing blood flowing under the door into the therapy room. The therapist said loudly and firmly, "There is no blood there," and went to the door and put his hand on the floor, and then had the patient do the same thing. The patient was relieved and the therapist continued by discussing with her other ways of discriminating the real from the unreal. Specific therapeutic effects using objective laboratory measures of reality testing have not been studied, however, because precise measures of the component processes have not been developed.

Recently Johnson (Johnson and Raye 1981; Johnson, in press) has developed a more highly operationalized cognitive model of reality testing, which she calls "reality monitoring." Reality monitoring is a collection of cognitive skills by which a person selects information from memory, discriminates the sources of memory information, and uses the information to interpret ongoing events. Unlike the original concept of reality testing, the specific processes of reality monitoring can be separately measured. The reality-monitoring model may provide the laboratory measures prerequisite to assessment and treatment of patients who misinterpret or misattribute perceptual and memory information.

Another molar cognitive process is "problem solving," formalized by D'Zurilla and Goldfried (1974) as a five-stage process of recognition, analysis, solution generation, solution selection, and outcome evaluation. Therapeutic procedures to improve the problem solving of several patient populations, including chronic psychiatric patients, were developed by Spivak, Platt, and Shure (1976) and Wallace (1982). A variety of less severely disturbed patients also benefit from problem-solving therapy, which suggests that many people never completely learn this cognitive skill in the first place. In persons suffering from schizophrenia, the therapeutic effect may therefore be partially due to learning a new cognitive skill. In addition, the highly formalized procedures of problem-solving therapy provide structure and enriched input that could reorganize a collapsed cognitive response hierarchy.

The fact that the five specific stages of problem solving can be separately assessed has allowed detailed research in normal populations. For example, in most people the rate-limiting stage is in solution generation; the more alternative solutions a person can generate, the more effective is that person's problem solving (see D'Zurilla and Goldfried 1974). There is probably considerable potential in quantitatively assessing performance in each stage of the process, and tailoring treatment accordingly. Procedures for clinically assessing problem solving in severely disordered patients are beginning to be explored (Hansen, St. Lawrence, and Christoff 1985), and recent evidence (Wallace and Liberman 1985) suggests a problem-solving approach enhances the effectiveness of social skills training.

A more molecular cognitive process known to be deficient in many psychiatric patients is the formation of concepts. Sometimes patients use conceptual categories that are overly broad and overly narrow. Sometimes the conceptual problem lies in concept *modulation*, the process of discarding a conceptual scheme when it is no longer useful and constructing a new one. This type of deficit appears to be associated with hostility, belligerence, and persecutory beliefs (Spaulding 1978). It may be due to a particular style of processing information, wherein new perceptual data are rejected in favor of memory data (Magaro 1984). There are no reports of controlled group studies wherein concept modulation is directly modified, but a case study illustrates the potential of this type of intervention:

> The patient was a 23-year-old man with chronic schizophrenia. After living for about 2 years in a supervised residential setting, he had made no rehabilitative progress, and his life had been punctuated by frequent altercations with service personnel and other patients. He had been denied admission to a residential psychosocial program because of his belligerent behavior and hostile demeanor.
>
> The patient had been on a stable antipsychotic drug regimen for 12 months. His history indicated that drugs were necessary to control more florid psychotic symptoms. He was compliant with his drug regime and was judged to be optimally medicated. As part of a systematic assessment procedure, the patient was administered a battery of psychomotor, attentional, and cognitive tests. The patient was within normal limits in simple reaction time, backward masking, span of apprehension, distraction effects on the reaction time task, redundancy-associated effects on the reaction time task, vigilance, and size estimation. He showed an isolated deficit on a version of the Wisconsin Card Sorting Test (see Lezak 1983), a task that requires formulation and reformulation of a conceptual scheme through trial and error. His difficulty on the sorting task was a tendency to adopt a particular scheme and perseveratively continue to use it long after the task demands had changed.
>
> The laboratory data suggested that the patient had a specific conceptual deficit that was preventing him from using his reasonably good social repertoire in stressful situations. The nature of the deficit was hypothesized to be a tendency to schematize a situation rapidly, and then perseverate with that schematization despite changes in the situation. The quality of the patient's attributional behavior in social situations suggested that he used a stereotypic conceptualization of all interactions; i.e., that the other person was trying in some way to take advantage of him. He was able to understand interactions in a better perspective when not in the situation, but this did not help when he was actively engaged. An exercise was designed to increase his ability to reconceptualize a social situation rapidly, and reject his stereotype. For 10 therapy sessions, the patient was asked to generate alternative schematizations, first to inkblots and then to Thematic Apperception Test (TAT) cards. That is, he was instructed to generate as many different percepts (to the inkblots) or stories (to the TAT cards) as he could to a single stimulus. He found this extremely difficult, especially with the TAT cards. Gradually, however, he gained an ability to generate three to four stories for each card without bizarre or perseverative elements in subsequent stories. One particular card caused him extraordinary difficulty. He reported that it reminded him of

> an incident in his past that was still upsetting. Even after responding well to two or three cards, he could only generate one morbid scenario for the problem card. This experience served to demonstrate to the patient that his problem could be exacerbated by stressful situations.
>
> The conceptual exercise was accompanied by counseling about the importance of conceptual flexibility, especially in social situations. After the end of the 10 exercise sessions, he was retested with the initial assessment battery. His previous conceptual deficit was reduced to within normal limits. He continued to receive regular counseling about social interactions, as he had before the exercises. In the following weeks, residential staff reported a positive change in his demeanor and attitudes. This was corroborated by an improvement in weekly NOSIE ratings, particularly on the Irritability subscale. At last followup, he had been accepted to the residential program that had previously rejected him.

Systematic Multimodal Assessment and Treatment. The foregoing discussion and case examples show the potential usefulness of interventions aimed at specific deficits in psychological levels of functioning. Cases selected for illustration of single interventions are always misleading, however. Recall that pervasiveness is a hallmark of schizophrenia and other severe psychiatric disorders. A patient with an isolated deficit in a single level of functioning represents the exception not the rule. In the overwhelming majority of cases, the clinician is confronted with an array of deficits with unknown mediational relationships.

Nevertheless, multiple interventions can be delivered in a systematic way. The key to delivering interventions systematically is to treat the most molecular deficits first, whenever possible. When mediational relationships are present, they can be expected to be molecular deficits causing molar deficits. Thus, normalization of the molecular deficit should eliminate the molar deficit as well. If an intervention is aimed at a molar deficit that is mediated by a molecular deficit (e.g., social skills training attempted with patients having severe attentional deficits), the effect of the intervention can be suboptimal.

A final case study illustrates systematic use of multiple biological, psychological, and social interventions in a patient whose deficits are typically pervasive:

> The patient was a 38-year-old woman. She had been transferred to an extended care ward because of failure to recover sufficiently after 2 months on an acute ward, where treatment had consisted of antipsychotic medication and milieu therapy. She showed a mixed picture of depressive, compulsive, and schizophrenic-like symptomatology, including withdrawal, auditory hallucinations, psychomotor retardation, impoverishment of thought, and bizarre posturing. She complained of fears that she would be infected by germs on the surfaces of common objects, and this was associated with bizarre dress and habits, including wearing plastic sandwich bags on her hands. She did not recognize her germ fears as irrational. Despite her fears, her grooming and hygiene were remarkably poor.
>
> The patient had a mixed social history. Although she had sporadically supported herself for some periods, was married and had children, her lifelong pattern was one of dependence on a few family members. Over a 2-year period, her symptom picture had developed from a fairly typical chronic compulsive disorder, limited to compulsive handwashing, to psychosis. At times during that period, her fear of germs was reported to be elaborated into bizarre somatic delusions.
>
> There was a controversy about how the patient should be treated, but the clinical staff agreed on a working hypothesis that her most molecular deficit was a drug-responsive physiological process of the type associated with endogenous depression. She was treated initially with an antidepressant drug (doxepin) and low-demand social activities. Over the next 6 weeks, the clinical data indicated a strong but selective response to the antidepressant regime. Her weekly NOSIE and repeated Inpatient Multidimensional Psychiatric Scales (IMPS) (Lorr and Klett 1966) showed an improvement in psychomotor retardation, social withdrawal, and impoverishment of thought. She no longer reported hallucinations. However, the same measures indicated that her posturing and other bizarre behavior remained unchanged.
>
> At 8 weeks after initiating medication, the drug response appeared to be stabilized. An attribution-oriented intervention was initiated, consisting of daily discussions with staff about personal hygiene, and realistic and unrealistic concerns about health hazards. This was reinforced with a contingency system wherein normal and appropriate hygenic behavior was rewarded with ward privileges. Over the next 4 weeks, the patient's appropriate self-care behaviors increased in frequency. In conversation she began to acknowledge that her fear of germs and her precautionary measures were irrational. These changes were accompanied by improvements in her weekly NOSIEs, to the degree that no overtly psychotic or compulsive behavior was observed on the living unit. Her IMPS profile corroborated a reduction in disordered thinking. The NOSIEs also showed improvements in the Neatness subscale, a measure of grooming and hygiene.
>
> Despite these changes the patient was subsequently caught stealing baggies from the ward supply room and using them in private. Although she continued to acknowledge the irrationality of her fears, a behavioral avoidance

test revealed she still could not touch some objects, especially doorknobs, without considerable distress. A traditional systematic desensitization treatment was initiated, and after 4 weeks she had regained her ability to touch objects without discomfort. Her compulsive hand-washing did not reappear.

Finally, with the patient's psychophysiological and attributional problems resolved, her living skill repertoire was assessed. The Independent Living Skill Inventory (a 60-item checklist developed in our laboratory) identified significant deficits in ordinary living skills and interpersonal skills. Over the next 4 months, she received a regimen of skills training in those areas. She gradually achieved skills levels adequate for relatively independent living, and was discharged to a community aftercare facility. At an 18-month followup, she had gained competitive employment, moved to her own apartment, and had suffered no relapses.

Future Directions and Conclusions

Evidence is accumulating that deficits in patients' psychological functioning can be reduced by specialized treatment procedures directed at remediation of the basic dysfunctions. There is less evidence that improvements in these psychological levels of functioning have a direct impact on achieving the goals of rehabilitation. A pioneering project has been carried out by Brenner et al. (1983) in which a variety of attentional and cognitive treatment techniques were integrated into a comprehensive rehabilitation program for chronic schizophrenics. Patients are "stepped" through the program, with advancement to a more molar level of treatment contingent on adequate performance at more molecular levels. The molecular treatments include special training in processing information from social interactions and encoding it in memory. More molar levels include social skills training. The project of Brenner et al. (1983) was a "field study" and the data are preliminary, but the outcome suggests that the molecular-level interventions significantly facilitate progress toward global rehabilitation goals.

Ultimately, highly controlled studies with homogeneous patient populations are required to validate the effectiveness of specific psychological interventions. One future direction for psychological interventions in schizophrenia must therefore be toward large-scale trials that test the effectiveness of specific treatments in achieving both focal and comprehensive complete psychiatric rehabilitation. There is a major obstacle to that, however. The psychological deficits found in schizophrenic patients are heterogeneous. A large-scale trial requires a population homogeneous with respect to the deficits being treated. It will be a long time before a significant number of trials have been run on subgroups of patients whose constellations of deficits are similar enough to assume group homogeneity.

Meanwhile, there is still much to be learned from smaller scale studies. Multiple replications of single case, controlled experimental designs can be helpful in the further development and validation of specific treatment techniques. Of the many measurement paradigms in experimental psychopathology, only a few have been adapted for practical use. Through smaller-scale exploratory studies, we learn what treatment approaches and measurement techniques are practicable in clinical settings, and what kinds of interventions deserve more detailed study. Similarly, the importance of short-term treatment outcome should not be underestimated. The benefits of an attentional intervention, for example, may lie in facilitating the first small steps a patient makes in a longer-term trajectory of improvements in social skills training or vocational rehabilitation.

A second future direction in this area should therefore be toward expanding the armamentarium of psychological intervention and measurement techniques through smaller-scale and shorter-term studies. The intriguing questions that could be addressed include: Are autonomic measures of arousal (heart rate, sweat gland activity, electromyogram) adequate for monitoring response to psychophysiological interventions, or will this require more expensive and complex central nervous system measures (e.g., averaged evoked cortical potentials)? Is naturalistic monitoring of psychophysiological responsivity (Tarrier et al. 1979) superior to laboratory measurement? What laboratory measures best identify the "modality-discrimination" deficit apparently associated with auditory hallucinations? Is the modality-discrimination training effect really a change in attentional functioning, or a reattribution of the deficit? To what degree do the effects of distraction-resistance training generalize outside the treatment setting or to other perceptual modalities? Can individualized self-instructional sets be developed based on laboratory assessment of attentional deficits? To what degree are short-term memory deficits in naturalistic settings separable from attentional deficits? Should they be treated separately? Can the ephemeral cognitive skills of "reality testing" and "problem solving" be compartmentalized into meaningful and treatable components?

Even after the psychological armamentarium is expanded and tested in large-scale trials, use in rehabilitation will not be a simple process. Patients' individual constellations of deficits are probably unique enough to preclude a "cookbook" approach to treatment. Idiographic assessment and individualized treatment regimens will probably always be necessary, even to take full advantage of skill training "packages" designed to remediate cognitive deficits through repetition, overlearning, focused demonstrations, video-assisted learning, and teaching problem-solving strategies (Wallace and Boone 1984). Interventions should therefore follow a hypothetico-deductive course, wherein theory guides initial data collection, treatment is delivered on the basis of inferences from the initial data, and outcome data validate the process. In other words, intervention strategies should aspire to well-controlled *n*-of-1 experimental design (Barlow and Hersen 1984).

The hypothetico-deductive approach to treatment brings other problems with it. Extensive continuous data collection capabilities are required. The institutional setting must be amenable to treatment based on measured outcome rather than preconceived policies about what is or is not appropriate treatment. The work of Gordon Paul and his colleagues (Paul and Lentz 1977) has demonstrated that this is possible through extensive clinical staff training, computer-assisted assessment and data management, and careful attention to the bureaucratic and administrative context of mental health services.

The clinical data system devised by Paul and his colleagues (see Paul and Lentz 1977) was supported by a mainframe computer. The data were limited to a social-behavioral level of functioning, focusing on consumption of reinforcers and other aspects of token economy treatment. Since that pioneering project, microcomputers have appeared, bringing powerful data-processing capabilities within the means of any mental health agency. Microcomputers can also provide measures of psychophysiological, attentional, and cognitive functioning at a cost much lower than required for a conventional psychopathology laboratory (Spaulding et al. 1981; Spaulding, Crinean, and Martin 1983). In view of these developments, a third future direction will inevitably be in the areas of multimodal data management technology and data-based clinical administration.

With systematic application of laboratory methods, integrated approaches that address psychological deficits can be evaluated for their impact on social competence, quality of life, and subjective comfort and satisfaction. Two guidelines can assist the rehabilitation practitioner in efficient use of multimodal interventions. First, being capable of *flexible levels of intervention* will reduce the number of patients deemed "refractory" to treatment; increasing the amount of time, spectrum of interventions, accessibility of services, and personalized contact when necessary will improve outcomes. Second, a *minimum-maximum strategy* will improve cost-effectiveness by first offering the least amount of services deemed clinically indicated and then adding additional services incrementally as new goals are formulated.

The choice of techniques to investigate and the variables to measure, as well as the coordination of assessment and treatment interventions in individual cases, is more focused and efficient when guided by a coherent theory. Thus, assessment and intervention should be guided by a general model of schizophrenia sensitive to empirically established psychopathological principles. This is illustrated by the relationships between theory and the approaches to treatment and rehabilitation presented in this article. When these requirements are met, an accumulation of knowledge is begun which can eventuate in meaningful group outcome studies. Given the apparent potential for laboratory assessment and treatment of psychological deficits, it seems appropriate to move forward.

References

Adams, H.; Brantley, P.; Malatesta, V.; and Turkat, I. Modification of cognitive processes: A case study of schizophrenia. *Journal of Consulting and Clinical Psychology*, 49:460–464, 1981.

Adams, H.; Doster, J.; and Calhoun, K. A psychological based system of response classification. In: Ciminero, A.; Adams, H.; and Calhoun, K., eds. *Handbook of Behavioral Assessment*. New York: John Wiley & Sons, Inc., 1977. pp. 47–78.

Alumbaugh, R. Use of behavior modification techniques towards reduction of hallucinatory behavior: A case study. *Psychological Reports*, 21:415–417, 1971.

Arieti, S. Schizophrenia, other aspects: Psychotherapy. In: Arieti, S., ed. *American Handbook of Psychiatry*. New York: Basic Books, 1959. pp. 485–507.

Azima, H., and Kramer, F. Effects of partial isolation in mentally disturbed individuals. *Diseases of the Nervous System*, 17:117–122, 1956.

Barlow, D., and Hersen, M. *Single Case Experimental Designs: Strategies for Studying Behavior Change*. 2nd ed. New York: Pergamon Press, 1984.

Beck, A. *Cognitive Therapy and the Emotional Disorders.* New York: International Universities Press, 1976.

Bellak, L.; Hurvich, M.; and Gediman, H. *Ego Functions in Schizophrenics, Neurotics and Normals.* New York: John Wiley & Sons, Inc., 1973.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias.* New York: International Universities Press, 1950.

Braff, D., and Saccuzzo, D. Effect of antipsychotic medication on speed of information processing in schizophrenic patients. *American Journal of Psychiatry,* 139:1127–1130, 1982.

Brenner, H.; Stramke, W.; Hodel, B.; and Rui, C. "A Treatment Program for Impaired Cognitive Functions Aimed at Preventing Chronic Disability Among Schizophrenic Patients: Results of a Two-Year Field Study." Presented at World Psychiatric Association Symposium, Baltimore, MD, 1983.

Broen, W., and Storms, L. Lawful disorganization: The process underlying a schizophrenic syndrome. *Psychological Review,* 73:265–279, 1966.

Budzynski, T. Clinical implications of EMG training. In: Schwartz, G., and Beatty, J., eds. *Biofeedback: Theory and Research.* New York: Academic Press, 1977. pp. 433–448.

Buss, A., and Lang, P. Psychological deficit in schizophrenia: I. Affect, reinforcement and concept attainment. *Journal of Abnormal Psychology,* 70:2–24, 1965.

Carpenter, W.T., Jr.; McGlashan, T.; and Strauss, J. The treatment of acute schizophrenia without drugs. *American Journal of Psychiatry,* 134:14–20, 1977.

Cockshott, J. The effects of induced anxiety with chronic hospitalized schizophrenics. *Dissertation Abstracts International,* 32:3631, 1971.

Cromwell, R., and Spaulding, W. How schizophrenics handle information. In: Fann, W.; Karacan, I.; Pokorny, A.; and Williams, R., eds. *Phenomenology and Treatment of Schizophrenia.* New York: Spectrum Publications, 1978. pp. 127–162.

Dawson, M., and Nuechterlein, K. Psychophysiological dysfunctions in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin,* 10:204–232, 1984.

D'Zurilla, T., and Goldfried, M. Problem-solving and behavior modification. *Journal of Abnormal Psychology,* 78:107–126, 1974.

Falloon, I., and Liberman, R.P. Interactions between drug and psychosocial therapy in schizophrenia. *Schizophrenia Bulletin,* 9:543–554, 1983.

Ford, M.; Stroebel, C.; Strong, P.; and Szarek, B. Quieting response training: Treatment of psychophysiological disorders in psychiatric inpatients. *Biofeedback and Self-regulation,* 7:331–339, 1982.

Freeman, T. On the psychopathology of schizophrenia. *Journal of Mental Science,* 106:925–937, 1960.

Friedberg, F. The effects of induced anxiety under verbal and nonverbal conditions on withdrawal in chronic hospitalized schizophrenics. *Dissertation Abstracts International,* 30(1-B):379–380, 1969.

Hansen, D.; St. Lawrence, J.; and Christoff, K. Effects of interpersonal problem-solving training with chronic aftercare patients on problem-solving component skills and effectiveness of solutions. *Journal of Consulting and Clinical Psychology,* 53:167–174, 1985.

Hebb, D.O. Drives and the C.N.S. (conceptual nervous system). *Psychological Review,* 62:243–254, 1955.

Held, J.; Cromwell, R.; Frank, E.; and Fann, W. Effect of phenothiazines on reaction time in schizophrenics. *Journal of Psychiatric Research,* 7:1–5, 1970.

Hogan, R. Implosive therapy in the short term treatment of psychotics. *Psychotherapy: Theory, Research and Practice,* 3:25–32, 1960.

Honigfeld, G.; Gillis, R.; and Klett, J. NOSIE-30: A treatment-sensitive ward behavior scale. *Psychological Reports,* 19:180–182, 1966.

Hull, C.L. *A Behavior System.* New Haven: Yale University Press, 1952.

Johnson, M. Discriminating the origin of information. In: Oltmanns, T., and Maher, B., eds. *Delusional Beliefs: Interdisciplinary Perspectives.* New York: John Wiley & Sons, Inc., in press.

Johnson, M., and Raye, C. Reality monitoring. *Psychological Review,* 88(1):67–85, 1981.

Johnson, W.; Ross, J.; and Mastria, M. Delusional behavior: An attributional analysis of development and modification. *Journal of Abnormal Psychology,* 86:421–426, 1977.

Kielhoffner, G.; Burke, J.; and Igi, C. A model of human occupation: Part 4. Assessment and intervention. *American Journal of Occupational Therapy,* 34:777–788, 1980.

Kornetsky, C. Use of a simple test of attention as a measure of drug effects in schizophrenic patients. *Psychopharmacologia,* 24:99, 1972.

Lang, P., and Buss, A. Psychological deficit in schizophrenia: II. Interference and activation. *Journal of*

Abnormal Psychology, 70:77–106, 1965.

Lezak, M. *Neuropsychological Assessment.* 2nd ed. New York: Oxford University Press, 1983.

Liberman, R.P.; Nuechterlein, K.; and Wallace, C. Social skills training and the nature of schizophrenia. In: Curran, J., and Monti, P., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment.* New York: Guilford Press, 1982. pp. 5–56.

Lorr, M., and Klett, J. *Manual: Inpatient Multidimensional Psychiatric Rating Scale.* Rev. ed. Palo Alto, CA: Consulting Psychologists Press, 1966.

Lowe, C., and Higson, P. Self-instructional training and cognitive modification: A behavioral analysis. In: Davey, G., ed. *Applications of Conditioning Theory.* London: Methuen, 1981. pp. 162–188.

Lukoff, D.; Wallace, C.J.; Liberman, R.P.; and Burke, K. A holistic program for chronic schizophrenic patients. *Schizophrenia Bulletin,* 12:274–282, 1986.

Magaro, P. Psychosis and schizophrenia. In: Spaulding, W., and Cole, J., eds. *The Nebraska Symposium on Motivation: Theories of Schizophrenia and Psychosis.* Vol. 31. Lincoln, NE: University of Nebraska Press, 1984. pp. 157–230.

Magoun, H.W. An ascending reticular activating system in the brain stem. *Archives of Neurology and Psychiatry,* 67:145–154, 1952.

Malmo, R.B. Activation: A neuropsychological dimension. *Psychological Review,* 66:367–386, 1959.

Meehl, P. Schizotaxia, schizotypy, and schizophrenia. *American Psychologist,* 17:827–838, 1962.

Meichenbaum, D., and Cameron, R. Training schizophrenics to talk to themselves: A means of developing self-controls. *Behavior Therapy,* 4:515–534, 1973.

Nuechterlein, K., and Dawson, M. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin,* 10:300–312, 1984*a*.

Nuechterlein, K., and Dawson, M. Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin,* 10:160–203, 1984*b*.

Oltmanns, T.; O'Hayon, J.; and Neale, J. The effect of antipsychotic medication and diagnostic criteria on distractibility in schizophrenia. *Journal of Psychiatric Research,* 14:81–91, 1978.

Paul, G., and Lentz, R. *Psychosocial Treatment of Mental Patients: Milieu Versus Social Learning Programs.* Cambridge, MA: Harvard University Press, 1977.

Reitman, E., and Cleveland, S. Changes in body image following sensory deprivation in schizophrenic and control groups. *Journal of Abnormal and Social Psychology,* 68:168–176, 1964.

Scheflin, A. *Levels of Schizophrenia.* New York: Brunner-Mazel, 1981.

Schneider, S., and Pope, A. Neuroleptic-like electroencephalographic changes in schizophrenics through biofeedback. *Biofeedback and Self-Regulation,* 7(4):479–490, 1982.

Serber, M., and Nelson, P. The ineffectiveness of desensitization and assertive training in hospitalized schizophrenics. *Journal of Behavior Therapy and Experimental Psychiatry,* 2:107–109, 1971.

Shakow, D. Segmental set. *Archives of General Psychiatry,* 6:1–17, 1962.

Shemburg, K., and Leventhal, D. Outpatient treatment of schizophrenic students in a university clinic. *Bulletin of the Menninger Clinic,* 36:617–640, 1972.

Shuart, W. "Effects of Self-Instructional Training on Performance in a Social Skills Training Group." Doctoral dissertation, University of Nebraska, Lincoln, 1985.

Slade, P. The effects of systematic desensitization on auditory hallucinations. *Behaviour Research and Therapy,* 10:85–91, 1972.

Small, M.; Giganti, M.; and Steinberg, D. A comparison of thermal biofeedback and relaxation training within a behavior management treatment program. *American Journal of Clinical Biofeedback,* 1:68–70, 1978.

Smith, R. Development of an integrated coping response through cognitive-affective stress management training. In: Sarason, I., and Spielberger, C., eds. *Stress and Anxiety.* Vol. 7. Washington, DC: Hemisphere, 1980.

Spaulding, W. The relationships of some information processing factors to severely disturbed behaviors. *Journal of Nervous and Mental Disease,* 166:417–428, 1978.

Spaulding, W. Assessment of adult-onset pervasive behavior disorders. In: Cummero, A.; Adams, H.; and Calhoun, K., eds. *Handbook of Behavior Assessment.* 2nd ed. New York: John Wiley & Sons, Inc., 1986. pp. 631–669

Spaulding, W.; Crinean, J.; and Martin, T. Microcomputerized clinical and research laboratories in psychiatric inpatient settings. *Behavior Research Methods and Instrumentation,* 15:171–176, 1983.

Spaulding, W.; Hargrove, D.; Crinean, J.; and Martin, T. A microprocessor-based laboratory for psychopathology research in rural settings. *Behavior Research Methods* *and Instrumentation*, 13:616–623, 1981.

Spivak, G.; Platt, S.; and Shure, M. *The Problem Solving Approach to Adjustment*. New York: Jossey-Bass, 1976.

Spohn, H.; Lacoursiere, R.; Thompson, K.; and Coyne, L. Phenothiazine effects on psychological and psychophysiological dysfunction in chronic schizophrenics. *Archives of General Psychiatry*, 34:633–644, 1977.

Storms, L., and Broen, W. A theory of schizophrenic behavioral disorganization. *Archives of General Psychiatry*, 20:129–144, 1969.

Strauss, J.S., and Carpenter, W.T., Jr. *Schizophrenia*. New York: Plenum Press, 1981.

Strauss, M.; Lew, M.; Coyle, J.; and Tune, L. Psychopharmacological and clinical correlates of attention in chronic schizophrenia. *American Journal of Psychiatry*, 142:497–499, 1985.

Tarrier, N.; Vaughn, C.; Lader, M.; and Leff, J. Bodily reactions to people and events in schizophrenics. *Archives of General Psychiatry*, 36:311–315, 1979.

Valins, S., and Nisbett, R. Attributional processes in the development and treatment of emotional disorders. In: Jones, E.; Kanouse, D.; Kelley, H.; Nisbett, R.; Valins, S.; and Weiner, B., eds. *Attribution: Perceiving the Causes of Behavior*. Morristown, NJ: General Learning Press, 1971. pp. 137–150.

Wallace, C. The social skills training project of the Mental Health Clinical Research Center for the Study of Schizophrenia. In: Curran, J., and Monti, P., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment*. New York: Guilford Press, 1982. pp. 57–89.

Wallace, C.J., and Boone, S.E. Cognitive factors in the social skills of schizophrenic patients: Implications for treatment. In: Spaulding, W., and Cole, J., eds. *The Nebraska Symposium on Motivation: Theories of Schizophrenia and Psychosis*. Vol. 31. Lincoln: University of Nebraska Press, 1984. pp. 283–318.

Wallace, C.J., and Liberman, R.P. Social skills training for patients with schizophrenia: A controlled clinical trial. *Psychiatry Research*, 15:239–247, 1985.

Wishner, J., and Wahl, O. Dichotic listening in schizophrenia. *Journal of Consulting and Clinical Psychology*, 42:460–464, 1974.

Woolfolk, R., and Lehrer, P., eds. *Principles and Practice of Stress Management*. New York: Guilford Press, 1984.

Zahn, T.; Carpenter, W.T., Jr.; and McGlashan, T. Autonomic nervous system activity in acute schizophrenia: II. Relationships to short-term prognosis and clinical state. *Archives of General Psychiatry*, 38:260–266, 1981.

Zeisset, R. Desensitization and relaxation in the modification of psychiatric patients' interview behavior. *Journal of Abnormal Psychology*, 73:18–24, 1968.

Zubin, J., and Spring, B. Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, 86:103–126, 1977.

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Symptom Monitoring in the Rehabilitation of Schizophrenic Patients

by David Lukoff, Robert Paul Liberman, and Keith H. Nuechterlein

Abstract

Although precise laboratory methods for measuring psychopathology are not available, interviewer-rated instruments developed to assess symptomatology can be used to monitor schizophrenic patients undergoing rehabilitation. By regularly assessing patients, rehabilitation staff can improve the effectiveness of their interventions. Patients can be screened for high levels of symptomatology which might preclude assignment to rehabilitation programs with high levels of social stimulation. Monitoring the prodromal symptoms of relapse can sometimes prevent florid relapses and sustain a rehabilitative trajectory. Standardized instruments for measuring positive symptoms (e.g., hallucinations, delusions, and conceptual disorganization) and negative symptoms (e.g., affective blunting, amotivation, and asociality) are available. Monitoring target symptoms may be particularly cost effective in the rehabilitation milieu. Use of suggested operational criteria for defining clinical states such as relapse would improve outcome studies on rehabilitation interventions.

The careful and reliable elicitation and rating of psychopathology in the formulation of a *DSM-III* Axis I diagnosis (American Psychiatric Association 1980) is preparatory to rehabilitation efforts with the mentally ill. In the lexicon of the rehabilitation practitioner, the array of characteristic symptoms of psychiatric disorder and the syndromal diagnosis represent the *impairments* of the patient. Given the prime importance of diagnosis in determining phase-specific drug and psychosocial treatments, the role of psychopathology assessment in rehabilitation planning cannot be overstated.

Beyond diagnosis, the repeated or ongoing monitoring of psychopathology is a valuable adjunct to the rehabilitation of individuals with schizophrenic or other major mental disorders. While other medical specialties can monitor the progress of patients through laboratory, radiological, and other quantitative assessments, psychiatric practitioners must rely on regular ratings of psychopathology to determine treatment and rehabilitation decisions. A thorough initial assessment of psychopathology and regular monitoring of symptoms in patients undergoing rehabilitation can improve the quality of treatment decisions in several areas.

1. The selection of an appropriate rehabilitation program is related to type and intensity of symptoms. Goldberg et al. (1977) found that patients assigned to an intensive rehabilitation program varied in their response depending on their initial level of symptomatology. Patients who entered the program with low levels of psychopathology generally benefited from treatment. However, symptomatic patients had high rates of relapse, which suggests that assignment of patients to demanding and socially stimulating programs should be preceded by an assessment of their symptoms. In that way, entry of symptomatic patients could be postponed until their symptoms have receded or stabilized.

The assessment of both positive and negative symptoms, as part of the rehabilitation planning process, can help patients and their caregivers

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avoid programs that might be overstimulating or understimulating. Understimulating social and vocational environments can lead patients to social withdrawal and often foster passivity, anergia, anhedonia, and loss of interest and initiative. On the other hand, placement of the patient in a "total push rehabilitation program" can produce overstimulation with attendant increased risk of exacerbation of florid symptoms of psychosis (Bennett 1978). The patient with schizophrenia, therefore, is often walking a tightrope between exposure to understimulating or overstimulating environments; the means for balance can come from periodic monitoring of symptomatology—including prodromal symptoms. The benefits of symptom monitoring can be amplified by sharing the results of the check-up with the patient and the patient's caregivers, thereby involving them actively in the planning of continued treatment and rehabilitation.

2. The effectiveness of rehabilitation efforts can be evaluated by changes in ratings. For example, by monitoring symptoms regularly, the therapist can determine whether social skills training is reducing the patient's anxiety at work. In addition, exacerbations of symptomatology in response to rehabilitation can be closely monitored and medications, changes in the program, additional therapy, or temporary suspension of the rehabilitation can be implemented to prevent further exacerbation of symptoms.

3. The need for and impact of medication changes can be systematically evaluated. Most medication appointments are brief, with 5 or 10 minutes being the norm. By using a rating scale, a psychiatrist involved in rehabilitation can more accurately, yet efficiently determine whether symptoms are improving or exacerbating. Because they observe patients over a longer time, rehabilitation personnel who are trained to monitor symptoms can provide valuable information to the prescribing psychiatrist about a patient's medication needs.

4. Patients can be taught to monitor their own symptoms and to recognize "warning signs." Patients who are trained to assess symptoms accurately are more capable of participating actively in their treatment decisions. In addition, they can initiate stress-reduction activities when they become sensitive to prodromal symptoms or find that their residual symptoms are exacerbating.

5. Research on rehabilitation would be enhanced by the use of instruments and procedures that accurately assess symptoms and the standardization of criteria for clinical conditions such as relapse. Comparability of psychopathology measures across studies is currently very low and hampers the contributions that research is capable of making to rehabilitation efforts. Accurate assessment of symptoms involves two distinct processes: Symptoms must first be elicited from the patient through interviewing and observation. Then, symptoms need to be categorized and rated for intensity.

Instruments for Rating Psychopathology

Researchers, concerned about the validity and reliability of their instruments, have developed several high quality instruments which can be incorporated into clinical rehabilitation programs with little or no modification. The three different instruments described below illustrate the options available for practitioners who are interested in assessing psychopathology.

Brief Psychiatric Rating Scale (BPRS). The BPRS (Overall and Gorham 1962) originally contained 16 symptom categories and was later expanded to 18 items (Guy 1976). The items are rated on a 1- to 7-point scale of increasing severity. Included in these scales are the psychotic symptoms of greatest importance for assessing the clinical condition of schizophrenic patients, i.e., hallucinations, unusual thought content (including delusions), and conceptual disorganization (including incoherence). The BPRS was designed originally for use by clinical observers of inpatient psychiatric populations in psychopharmacological outcome studies. For a study of schizophrenic outpatients at the UCLA Clinical Research Center for Schizophrenia and Psychiatric Rehabilitation, we found it necessary to devise a brief interview and anchor points applicable to an outpatient population. In addition, a review of the literature on the prodromata of relapse and hospitalization in schizophrenia (Wing 1978; Herz and Melville 1980) led to the addition of three new scales to assess behaviors which might signal a deterioration in an outpatient schizophrenic patient's condition: bizarre behavior, self-neglect, and suicidality. Three scales which assess symptoms of particular importance in the manic phase of bipolar and schizoaffective (manic type) illness were also added (Bigelow and Murphy 1978). The manual, which includes a semistructured interview and anchor points, is presented as Appendix A. Administration of the interview and rating of symptoms takes 10–40 minutes depending upon the interviewer's familiarity with the patient, the number of symptoms present, and the patient's capacity to describe symptoms. The symptoms can be readily graphed so that changes in baseline levels of one or more symptoms can be quickly detected and intervention mounted. Both nonpsychotic, prodromal symptoms and psychotic symptoms can be thus followed.

Schedule for the Assessment of Negative Symptoms (SANS). In Bleuler's (1911/1950) formulation of schizophrenia as a separate disease entity, negative symptoms were held to be a primary feature of schizophrenia. Although the symptoms considered to be in the grouping differ somewhat across authors (Andreasen 1982; Crowe 1985), negative symptoms usually include deficiencies in psychological and behavioral functions such as motivation (amotivation), ability to experience enjoyment (anhedonia), need for social contact (asociality), flow of thought (alogia), and affective expressiveness and experience (blunted affect). While the positive symptoms of schizophrenia (hallucinations, delusions, and incoherence) have been the primary focus of clinicians and theorists, negative symptoms have again attracted attention and are now considered a significant component of the symptom picture of schizophrenia. In approximately one third of schizophrenic patients, the negative symptoms show greater clinical prominence than the positive symptoms (Andreasen and Olsen 1982).

Negative symptoms can be as incapacitating as positive symptoms and, in one study, patients whose clinical picture was dominated by negative symptoms had a poorer outcome than patients with predominantly positive symptoms (Andreasen and Olsen 1982). In rehabilitation, amotivation often presents a greater obstacle to effective treatment than hallucinations. The SANS (Andreasen 1982) was developed to assess 20 of the negative symptoms divided into five areas: affective flattening, alogia, avolition/apathy, anhedonia/asociality, and attention. Symptoms are rated on a 0- to 5-point scale of increasing severity. A brief interview covering the previous month requires 10–30 minutes depending upon the interviewer's familiarity with the patient, the number of symptoms present, and the patient's capacity to describe symptoms.

Target Symptoms. The rating of target symptoms makes use of the finding that patients tend to have exacerbations of the same symptoms across different episodes of relapse (Leff and Wing 1971). Therefore, it is possible and efficient to monitor the specific symptoms that most sensitively reflect a given patient's clinical condition. Battle et al. (1966) first used this approach to monitor progress on target complaints in psychotherapy research. An Idiosyncratic Target Symptom Scale, comprising 100 points for rating one or more symptoms or signs of psychosis, has been used for treatment studies in schizophrenia (May 1980). This, as well as other target symptom scales, can be readily used by clinicians of various stripes, and it correlates well with multidimensional scales of patients' psychopathology (Mintz 1985).

Falloon, Boyd, and McGill (1984) adapted this methodology to monitor the psychopathology of schizophrenic patients who were participating in a 2-year study of family, individual, and drug therapy. For each patient in the study, they selected two symptoms that had recurred in previous exacerbations. Care was taken to specify each symptom exactly as it was manifested in that patient and to avoid behavioral disturbances that might occur in the presence of a nonschizophrenic episode (e.g., social withdrawal during a depressive episode). These target symptoms were monitored monthly and rated on a 1- to 7-point scale of increasing severity similar to the BPRS. The target symptom scale was sensitive to episodes of relapse and better distinguished the treatment conditions than did the BPRS. It is likely that target symptom scales, because they highlight symptoms that are key for each patient, reflect individual differences and are more sensitive to changes in clinical state than multidimensional, more comprehensive instruments.

Methods and Definitions for Symptomatic Outcome and Clinical States

Although symptoms vary on a continuum, for some purposes it is advantageous to demarcate categories of clinical status such as relapse. While studying the effects of a medication washout, Docherty et al. (1978) identified a sequential unfolding of states with uniform symptom configuration. Other studies have identified the characteristic prodromal symptoms that precede relapse (Herz and Melville 1980). Although the concept of relapse is more problematic to define for schizophrenic disorder than for most other diseases, operational definitions of this state have also been developed in the course of research projects. Relapse may be useful to recognize as a clinical state by rehabilitation practitioners because its frequency and intensity may militate against the referral and involvement of patient in a rehabilitation program. Symptoms of high intensity that qualify for the definition of "relapse" are generally believed to interfere with productive involvement and progress in rehabilitation (Liberman and Foy 1983; Anderson, Reiss, and Hogarty 1986). Similarly, recognizing the prodromal stages of relapse may equip rehabilitation personnel with the capability of intervening with patients to prevent deterioration of function and cognitive status (Herz, Szymanski, and Simon 1982). Below are listed some definitions of relapse which could be transplanted to rehabilitation settings for clinical decision-making.

Operational Relapse. The definition of relapse in schizophrenic disorders is characterized by both methodological and conceptual disarray. In a recent survey of 15 treatment outcome studies conducted during the 1970s, Falloon (1984) found that no two studies used the same criteria to define relapse. The designation of relapse was tied to a host of differing variables.

> Admission to a psychiatric hospital unit, increase of medication, worsening of florid symptoms of schizophrenia, worsening of *any* psychiatric symptoms, and threatened clinical exacerbations have all been variables considered under the rubric of relapse. [Falloon 1984, p. 295]

In addition, none of these definitions reported interrater reliability agreement coefficients. The lack of comparable relapse definitions used in various rehabilitation programs contributes to the difficulty in determining and disseminating effective treatments for schizophrenic patients.

Because of its variable symptom presentation and course, schizophrenia presents a unique challenge to the operationalization of the concept of relapse. Classically, the notion of relapse refers to the reemergence of a florid episode of illness in a person previously in a state of stable remission. This concept is appropriate for illnesses such as tuberculosis and peptic ulcer, which are characterized by periods of full remission alternating with periods of symptomatology. However, longitudinal studies have found that perhaps 50 percent of schizophrenic patients do not attain a stable clinical remission (Bleuler 1974; Ciompi 1980). By the traditional concept of relapse, many schizophrenic patients are in a continuous state of partial or full relapse. The issue of persisting symptoms has been one of the major methodological obstacles to the development of reliable and valid definitions of relapse.

Relapse has been used to refer to a longitudinal outcome and also a cross-sectional clinical state. In treatment studies, relapse typically refers to an *outcome*: that is, a measure of the trajectory of change in clinical condition from the beginning to the end of a study. Although the clinical condition of patients defined as relapsed by this method shows signs of severe decompensation, the range of intensity can vary widely depending on the preexisting baseline level of symptomatology. Patients in full remission at the beginning of the evaluation period may relapse at a lower point of severity. When used to designate a *specific clinical state* rather than a relative exacerbation, the definition adheres more closely to the classical concept of relapse. Patients defined as relapsed by this method would be in a narrower range of severity symptoms.

It is important that both outcome and clinical state definitions be keyed to the psychotic symptoms that are characteristic of schizophrenic disorder. Many of the definitions used in the past have confounded relapse with social factors such as behavioral disturbance and hospitalization. Behavioral disturbances are differentially tolerated by families of different ethnic backgrounds, and a wide variety of social factors that are not related to symptoms affect the likelihood that a given patient will be hospitalized (Wing 1968). Therefore, for relapse to serve as an indicator of the schizophrenic disease process, the definition must be based on the core psychotic symptoms that specifically characterize schizophrenia unconfounded by social variables or more peripherally experienced nonpsychotic symptoms.

Moreover, the mere presence of psychotic symptoms does not always represent a condition that warrants the designation of relapse. Even when the symptoms are diagnostically significant (e.g., mood-incongruent third person auditory hallucinations), they may not be at a level of severity associated with the term "relapse" in usual clinical practice. For example, a schizophrenic patient who hears a voice a couple of times a week or occasionally believes songs on the radio give him messages would not usually create much concern among treatment personnel. It is only when the patient's symptoms reach a certain level of frequency and intensity (e.g., auditory hallucinations throughout the day) and the patient's functioning is impaired (e.g., the messages from the radio tell the patient not to eat or go to his job) that the appellation "relapsed" would usually be applied by treatment personnel. Therefore, the definitions presented below incorporate both specific symptom, frequency, and intensity criteria, as well as the degree to which symptoms interfere with social functioning.

The level of severity that is set to define relapse will affect the findings of a study. For example, Kane et al. (1983) reported a relapse rate of 56 percent for schizophrenic patients treated with low-dose medication, whereas Hogarty (1984) found that a similar low dose of medication yielded only a 23 percent incidence of relapse. A parsimonious explanation is that these two sets of investigators used different criterion intensities of symptomatology to define relapses.

Outcome definitions require a careful initial assessment of the patient's level of symptomatology to set the baseline from which relapse is determined. The first study to use a definition of relapse that took into account the level of preexisting symptoms was conducted by Brown, Birley, and Wing (1972). They found that 29 percent of their sample of schizophrenic patients were discharged from the hospital with persisting symptoms as elicited by a structured Present State Examination (PSE) interview (Wing, Cooper, and Sartorius 1974). Brown, Birley, and Wing (1972) distinguished two types of relapse: Type I involved a change from a normal or nonschizophrenic state to a state of schizophrenia as defined by the PSE Catego diagnostic system (Wing, Cooper, and Sartorius 1974). Type II relapse involved a marked exacerbation of psychotic symptoms from the patient's baseline level assessed at discharge from the hospital.

The definitions of relapse used in studies at our UCLA Clinical Research Center have been more quantitatively operationalized than previous definitions because we felt it was essential to achieve high degrees of interrater reliability. Moreover, even when a particular patient's clinical state is being monitored for treatment planning, quantitative ratings are helpful because they enable more sensitive distinctions to be made and permit the practitioner to graph the changes in psychopathology over time. Our colleagues in medicine have monitored illness factors in quantitative fashion for generations (e.g., fever charts, blood counts, and other laboratory values); thus, as psychiatric practice develops a biomedical data base, quantitative monitoring of symptoms should become routine.

The first definition (Nuechterlein et al. 1985) was developed for a study in which schizophrenic patients were given BPRS assessments during their regular clinic visits every 2 weeks. At the end of a 1-year period, the researchers wanted to assign outcomes to the patients based on the course of symptoms over a 1-year period. Figure 1 schematically illustrates the criteria and decision-making process used in this outcome definition of relapse. Actually, nine outcome possibilities were operationally defined and then grouped into relapse/no relapse/unchanged categories.

The second set of criteria for relapse as an outcome measure was developed by Drs. Robert P. Liberman, Ian Falloon, and Simon Jones for a study in which it was not possible to assess the patients during regularly scheduled visits to a clinic. The patients were recruited while still hospitalized, for a study of family factors in relapse (Vaughn et al. 1984). The research called for ascertaining which of them relapsed during the 9 months following discharge. Certain family variables, particularly high "expressed emotion" (Brown, Birley, and Wing 1972; Vaughn and Leff 1976), were predicted to be *associated with* higher likelihood of relapse.

Patients were administered the PSE and the Psychiatric Assessment Scale (PAS) (Krawiecka, Goldberg, and Vaughan 1977) at admission to and discharge from the psychiatric hospital. The PAS is a standardized rating scale that was designed specifically to reflect severity of symptoms among chronic psychiatric patients. It consists of eight items rated on a 0–4 scale of increasing severity.

In the Vaughn et al. (1984) study, the researchers contacted the patient or the patient's family by telephone each month and inquired whether there were any signs of symptomatic exacerbation, deterioration in functioning, or change in medication dosage. The information from the telephone calls was transcribed and retained. A psychiatrist and psychologist trained in the PSE and PAS monitored these reports and made immediate arrangements to conduct a PSE and PAS with any patient for whom the telephone call revealed indications of a possible relapse. All patients who did not have an evaluation triggered by this method were reassessed at the 9-month postdischarge point.

The relapse criteria developed for this study made use of three data sources: PAS, PSE, and anecdotal reports of the patient's clinical and social status. The results from the PAS were reviewed first. Criteria for relapse from a previous study (Wallace 1982) were applied (table 1). A psychoticism score was created from the PSE-elicited symptoms by summing the items that refer to the characteristic symptoms of schizophrenia (table 2). A determination of relapse was made based on the comparison of the discharge psychoticism score versus the followup score. If both the PAS and PSE produced the same outcome, the patient was rated accordingly. However, if they produced differing outcomes or if high levels of symptoms present at discharge persisted to the followup evaluation, the anecdotal information (with all identifying features removed) was reviewed. In some cases this additional information allowed the patient to be assigned to the relapsed or nonrelapsed outcome category. Even with these three sources of symptom data, 22 percent of the patients had such high persisting and unremitting symptoms for the full 9-month followup period that they could not be assigned into the relapsed or nonrelapsed category. Agreement between two psychiatrists across 76 cases was 92 percent.

For an ongoing longitudinal follow-through study of schizophrenic patients, "Developmental Processes in Schizophrenic Disorders" (Principal Investigator: Keith H. Nuechterlein, Ph.D.), an operational definition for relapse as a clinical state has been derived from the BPRS by Drs. Lukoff and Nuechterlein. This study aims to determine predictors of schizophrenic relapse and remission as well as vulnerability-linked versus symptom-linked markers of disorder. The investigators developed a definition that enables patients whose psychotic symptoms have exacerbated to a high level of intensity to be identified immediately. Patients are assessed biweekly, and extensive cognitive and psychophysiological tests are administered to patients who are found to be in a state of relapse.

Operational criteria for two categories of relapse were developed: (1) *psychotic relapse*, which is based on the core BPRS psychotic symptom scales of hallucinations, unusual

Figure 1. Flow chart for determining relapse outcome after 1 year based on Brief Psychiatric Rating Scale (BPRS)

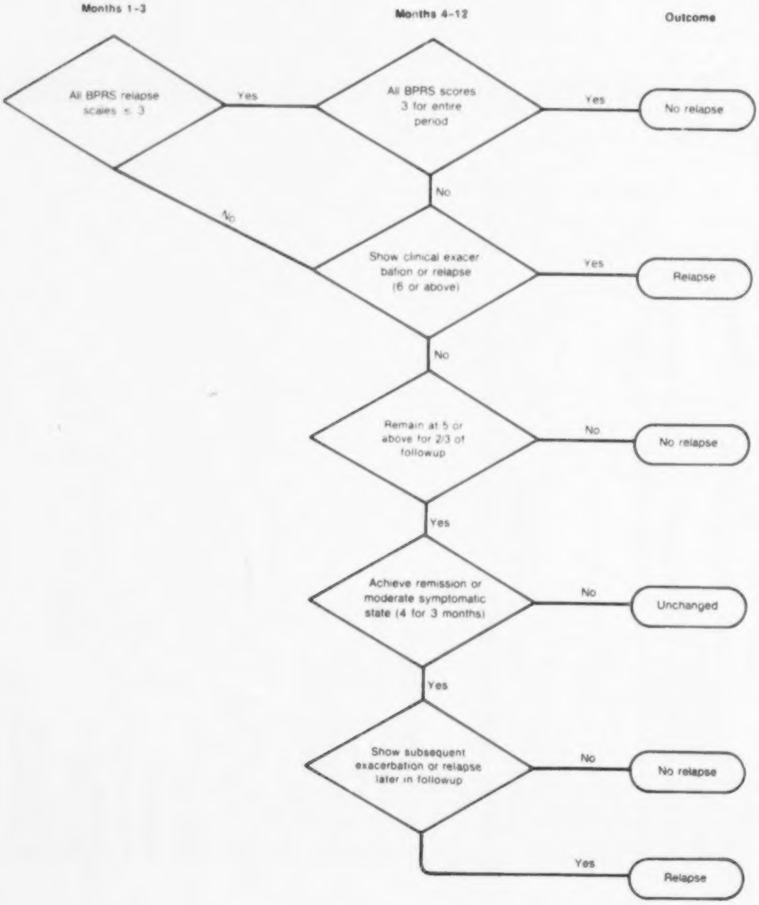

Months 1-3
Months 4-12
Outcome
All BPRS relapse scales ≤ 3
Yes
All BPRS scores 3 for entire period
Yes
No relapse
No
No
Show clinical exacerbation or relapse (6 or above)
Yes
Relapse
No
Remain at 5 or above for 2/3 of followup
No
No relapse
Yes
Achieve remission or moderate symptomatic state (4 for 3 months)
No
Unchanged
Yes
Show subsequent exacerbation or relapse later in followup
No
No relapse
Yes
Relapse

Table 1. Relapse criteria based on the Psychiatric Assessment Scale

Type I. If a change from discharge rating occurs on only 1 of the 3 scales, a 2-point increase is designated as a relapse, providing that a maximum severity score of 4 on that scale occurs. Thus, score increases from 0 to 2 or from 1 to 3 would not be considered a relapse, but an increase from 2 to 4 would be a relapse.

Type II. A total increase of 3 points on 1 or more of the 3 scales is designated a relapse, with the caveat that single point changes from 0 to 1 are not counted (0 = symptoms absent; 1 = symptoms not clearly pathological).

Table 2. Relapse rating form from Vaughn et al. (1984)

Case No. ________		Initial	Discharge	Followup
Psychiatric Assessment Scale Scores				
Delusions				
Hallucinations				
Incoherence				
Decision (circle one)			Relapse	No relapse
Present State Examination (PSE) item scores				
Thought disorder symptoms (Score "1" if rated "1" or "2" on PSE)				
Thought insertion	55			
Thought broadcasting	56			
Thought echo/commentary	57			
Thought block/withdrawal	58			
Delusion of thoughts read: (Score "1" if rated "2" on PSE)	59			
Hallucinations (Score "1" if rated "1" or "2" on PSE)				
Nonverbal auditory	60			
Voice commentary/3rd persons	62			
Voices to subject	63			
Visual	66			
Olfactory	68			
Other	70			
Delusion of odors (Score "1" if rated "2" on PSE)	69			
Delusions (Score "1" only if full delusions are rated "2" on PSE)				
Control	71			
Reference	72			
Misinterpretation	73			
Persecution	74			
Assistance	75			
Grandiose abilities	76			
Grandiose identity	77			
Religious	78			
Paranormal	79			
Physical forces	80			
Alien forces	81			
Primary	82			
Jealousy	84			
Pregnancy	85			
Sexual	86			
Fantastic	87			
Guilt	88			
Appearance	89			

Depersonalization				90			
Hypochondriacal				91			
Catastrophe				92			
	Scoring		**PSE rating**	95			
Preoccupation	0	=	0–2				
	1	=	3				
	2	=	4				
	3	=	5				
Insight	1	=	2	104			
	2	=	3				
Interview behavior:	**Scoring**		**PSE rating**				
	1	=	2				
	2	=	2		**Initial**	**Discharge**	**Followup**
Distractibility				114			
Mannerisms & posturing				116			
Behaves as if hallucinated				118			
Catatonic movements				119			
Suspicion				125			
Perplexity				126			
Incongruity of affect				129			
Muteness				133			
Neologisms				135			
Incoherence				136			

Total PSE psychoticism score. . . . .

Decision (circle one) . . . . Relapse No relapse

Global Ratings of severity of symptoms and acting out symptoms based on telephone and other followup sources

Decision (circle one) . . . . Relapse No relapse

Overall judgment using all data sources. . . . . Relapse No relapse

Comments ____

____

____

thought content, and conceptual disorganization; and (2) *other types of relapse,* which signal gross impairments in the patient's functioning or thinking, but which are not clearly related to schizophrenic psychotic processes, i.e., depression, suicidality, self-neglect, bizarre behavior, hostility. (See table 3.) A rating of 6 (severe) or 7 (extremely severe) on any of the key symptom items signifies a relapse. The intraclass

Table 3. Brief Psychiatric Rating Scale and behavioral anchors used to define types of relapse in the "Developmental Processes in Schizophrenic Disorders Project"

Scale item	Rating	Definition	Reliability coefficient[1]
		Psychotic relapse	
Unusual thought content	6 Severe	Full delusion(s) present with much preoccupation *or* many areas of functioning are disrupted by delusional thinking	.93
	7 Extremely severe	Full delusion(s) present with almost total preoccupation *or* most areas of functioning are disrupted by delusional thinking	
Hallucinations	6 Severe	Several times a day *or* many areas of functioning are disrupted by hallucinations	
	7 Extremely severe	Persistent throughout the day *or* most areas of functioning are disrupted by hallucinations	.97
Conceptual disorganization	6 Severe	Speech is incomprehensible due to severe impairments most of the time	.73
	7 Extremely severe	Speech is incomprehensible throughout interview	
		Other types of relapses	
Depression	6 Severe	Deeply depressed most of the time *or* many areas of functioning are disrupted by depression	.90
	7 Extremely severe	Deeply depressed constantly *or* most areas of functioning are disrupted by depressive thinking	
Suicidality	6 Severe	Wants to kill self. Searches for appropriate means and time *or* suicide attempt that is a potentially serious threat to life with patient knowledge of possible rescue	.97
	7 Extremely severe	Specific suicidal plan and intent (e.g., "as soon as __________, I will kill myself by doing X") *or* suicide attempt characterized by plan that the patient thought was lethal or an attempt in secluded environment	
Self-neglect	6 Severe	Hygiene and eating potentially life-threatening (e.g., eats and/or bathes only when prompted)	.78
	7 Extremely severe	Hygiene and eating life-threatening (e.g., does not eat or engage in hygiene)	
Bizarre behavior	6 Severe	Unusual petty crimes (e.g., directing traffic, public nudity, contacting authorities about imaginary crimes)	.84
	7 Extremely severe	Unusual serious crimes (e.g., setting fires, asocial theft, kidnapping committed in bizarre fashion or for bizarre reasons)	
Hostility	6 Severe	Has assaulted others but with no harm likely (e.g., slapped or pushed others *or* destroyed property, knocked over furniture, broken windows)	.89
	7 Extremely severe	Has attacked others with definite possibility of harming them or with actual harm (e.g., assault with hammer or weapon)	

[1] Median ICC among the 7 Developmental Processes BPRS raters.

correlation coefficient among seven raters across 17 cases was .81.

Detecting Prodromal Symptoms. Several recent studies have pointed to the existence of identifiable intermediate states between remission and relapse in schizophrenic illness. When Herz and Melville (1980) retrospectively interviewed schizophrenic patients and their relatives about the period preceding a relapse, most were able to report a distinct prodromal period. The symptoms mentioned most frequently by patients and their relatives were nonpsychotic:

> symptoms of dysphoria that nonpsychotic individuals experience under stress, such as eating less, having trouble concentrating, having trouble sleeping, depression, and seeing friends less. [Herz and Melville 1980, p. 803]

In a prospective study of schizophrenic outpatients, Marder et al. (1984) also found evidence for a prodromal period by noting changes in ratings on quantitative scales just before relapse. The interpersonal sensitivity, depression, anxiety, paranoid ideation, and psychoticism scales of the Symptom Checklist-90 (Derogatis, Lipman, and Covi 1973) and the thought disorder, depression, and paranoia factors on the BPRS were significantly elevated from previous administrations during the 1- to 3-month period before a significant exacerbation or return of patients' characteristic psychotic symptoms.

From the viewpoint of specificity in the prediction of relapse, it is unfortunate that both of these research teams reported that prodromal symptoms did not always signal an impending relapse. The team led by Marder developed a predictive model but found that there was a distinct trade-off between sensitivity and specificity. When the predictive equation was adjusted for high sensitivity, it identified 92 percent of the patients who subsequently relapsed, but was mediocre in specificity, predicting relapses incorrectly for 51 percent of the patients who did not relapse. Conversely, when specificity was set low to avoid false positives in predicting relapses, false positives could be reduced to 2 percent. However, only 42 percent of actual relapses were identified.

Of course, one would not expect every instance of raised psychopathology to herald a relapse since protective factors in the individual and his social network would occasionally buffer and interdict the stress-linked relapse process. Clinicians who use such criteria to assess impending relapse need to judge the costs versus the benefits of false positives versus false negatives in deciding the level of prodromal symptoms to consider noteworthy. High sensitivity and only modest specificity in predicting actual prodromata would serve an important function in continuing care and rehabilitation programs, permitting clinicians to intervene preventively by increasing the dose of medication or psychosocial therapies. Providing additional treatment even on occasions that would not have led to relapse, from this vantage point, would hardly constitute wasted resources.

Clearly, it would be desirable to be able to identify impending relapses during their formative prodromal stage. Preventive medication and psychosocial treatment strategies could be used in tandem with identification of prodromata. The development of intermittant medication strategies (Herz, Szymanski, and Simon 1982; Carpenter and Heinrichs 1983), a promising innovation in psychopharmacological treatment, requires the ability to recognize the early signs of relapse that signal the reintroduction of medication. By becoming familiar with the prodromal signs of relapse, rehabilitation staff who interact with patients on a regular basis may often be in a position to recognize and forestall patient relapses.

With increased awareness and understanding of the prodromal stage of relapse, schizophrenic patients, who are usually relegated to a passive role regarding their illness, can become more actively involved with their treatment. Mendel (1976) developed a program where schizophrenic patients in the community were trained to recognize their own "warning signs." Each patient carried a list of his or her own idiosyncratic symptoms, ranging from "thinking a lot about past hospitalizations" to "trouble sleeping," along with a phone number to call if these warning signs developed.

In the Herz and Melville (1980) study, the symptom configuration reported by the patients and relatives seemed to show a high degree of intraindividual specificity and stability across incidents of relapse. Yet there was much variability in the types of symptoms present during the prodromal period and also in the time period over which the symptoms developed. Only a small percentage of patients (8 percent) reported that the period between onset of prodromata and frank relapse was less than 1 day. However, 50 percent of patients reported that they noticed symptoms for less than 1 week before florid return of psychosis. Thus, it would seem that a rating of target symptoms would be most appropriate for monitoring prodromal symptoms since the symptoms being monitored would be mostly nonpsychotic and idiosyncratic. A detailed phenomenological interview covering previous relapse periods would be necessary to determine the specific symptoms to be monitored. Checking with the patient's relatives and treating professionals would also contribute to a clearer picture of the patient's prodromal periods. While prodromal symptoms should be monitored on at least a weekly basis, the rating of target symptoms, by patient and responsible clinician, would not be time-consuming.

Training schizophrenic patients in the use of stress-reduction techniques could also play a role in preventing the onset of prodromal symptoms and the exacerbation of prodromal symptoms into relapses. The relationship between stressors such as life events (Brown and Birley 1968) and familial tension (Vaughn et al. 1984) has been established in several studies (for review, see Lukoff et al. 1984). Herz and Melville (1980) found that the most common prodromal symptom that appeared before hospitalization of schizophrenic patients was feeling tense and nervous—reported by 80 percent of the patients. Many of the other prodromal symptoms uncovered in their study are also thought to be related to stress, e.g., trouble sleeping and restlessness.

Lukoff et al. (1986) at the UCLA Clinical Research Center for the Study of Schizophrenia developed a 10-week inpatient program that incorporated aerobic exercise, meditation, and educational sessions on stress. While the schizophrenic patients were in the hospital, they participated actively in the program and showed substantial decreases in psychopathology. Upon discharge, however, they discontinued practicing the stress-reduction techniques. Given the relationship between stress and schizophrenic relapse, the regular use of stress-reduction techniques might act prophylactically. A program incorporating techniques such as exercise, relaxation, and stress-monitoring would need to use behavioral principles and procedures (e.g., reinforcement and modeling) in the training phase and for maintenance of the stress-reduction activities.

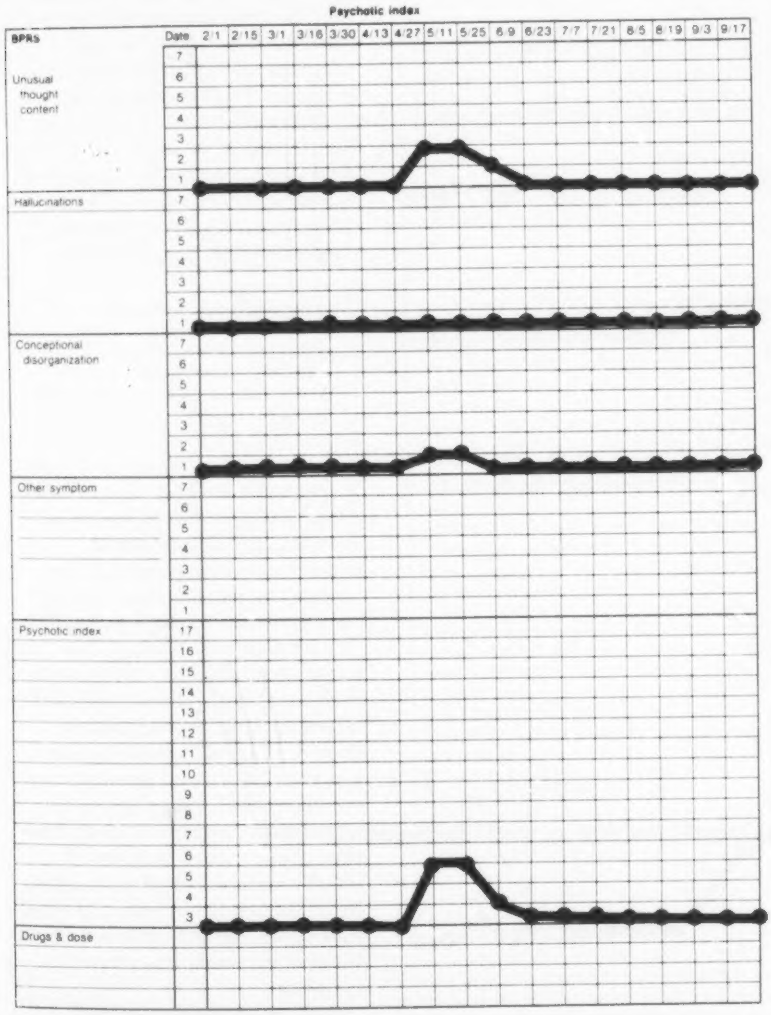
Clinical Vignettes That Illustrate Symptom Monitoring

The following examples (with fictitious names) are compilations drawn from the UCLA Aftercare Clinic, where patients are monitored every 2 weeks with the BPRS. For each patient, a psychotic index consisting of the sum of the ratings on the hallucinations, unusual thought content, and conceptual disorganization scales from the BPRS is graphed. These data provide the case managers with a longitudinal perspective that shows previous levels of psychopathology against which the current levels can be compared. By regular monitoring of psychopathology and computation of the psychotic index, the case managers can evaluate the significance of fluctuations in clinical status and readily mount effective treatment interventions.

Case 1. Bill, a patient at the Aftercare Clinic, requested that the social worker help him move from a small board-and-care facility where he had been living for the past 2 years to a board-and-care facility closer to his parents. The social worker arranged a transfer to a much larger facility in the neighborhood where his parents lived. One month after the move, the social worker had a session with the patient and asked him how he was doing. Bill replied that he enjoyed being able to spend some evenings and weekends with his parents. If she had stopped the interview at that point, everything would have seemed fine. However, when the social worker proceeded to ask questions from the BPRS, she uncovered the patient's belief that others at the board-and-care facility were staring at him and talking about him, an increase from very mild (2) to mild (3) on the unusual thought content item. She also noted the presence of mild conceptual disorganization for the first time. These prodromal symptoms were of concern to the social worker because they represented a definite exacerbation from the level of symptomatology present before the patient's move (see figure 2) even though these symptoms were not at full psychotic levels. Additional questioning revealed that Bill felt overwhelmed at the new larger placement and had not made even any casual friendships. However, he did not want to move further away from his parents. The social worker immediately scheduled an appointment that afternoon with his psychiatrist to determine if an increase in medication was warranted. With the patient's consent, she notified the board-and-care manager and suggested that he pay special attention to the patient. Then she contacted other board-and-care facilities in the area to locate one which housed a smaller number of residents. Medication dose was not increased, but Bill's visits to his social worker therapist were temporarily increased. Three weeks later, a more suitable placement was found and Bill's symptoms soon thereafter returned to their previous level. Through the social worker's careful monitoring of the patient's symptomatology and her efforts to alter the stressful situation, a potential relapse was averted.

Case 2. John told his psychologist that he both wanted to and was very worried about starting to take college courses again. One year before, he had a psychotic relapse which resulted in hospitalization 2 weeks after starting a new semester. John had completed six semesters of college and stated that he wanted to finish college but said that his parents were worried that taking college courses would make him ill again. Dr. Smith arranged for a family session. After allowing time for John's parents to discuss their concerns, she encouraged the family to develop a "game plan" that would allow John to tackle college again. John's family made the suggestion that John should drop down from his usual load of three or four courses to two courses. John's mother also agreed to support John by making him breakfast and bag lunches on his school days. John's father, who initially argued that John should defer one more semester, agreed to take a "wait-and-see" attitude. Dr. Smith said that she would arrange a time when John would call her twice-a-week during the first 2 weeks. In addition, Dr. Smith said that she would be monitoring John's clinical condition very closely using a standardized rating scale that would reveal any trends toward relapse. As figure 3 shows, the first week of classes did produce a mild exacerbation. Dr. Smith consulted the aftercare clinic psychiatrist who managed John's medications and arranged for a temporary increase in dosage. By the fourth week, John had acclimated to the demands of school and reported that he was doing fine in his courses and enjoying being active again. His dose of antipsychotic drug was subsequently reduced when the psychotic index returned to its baseline level.

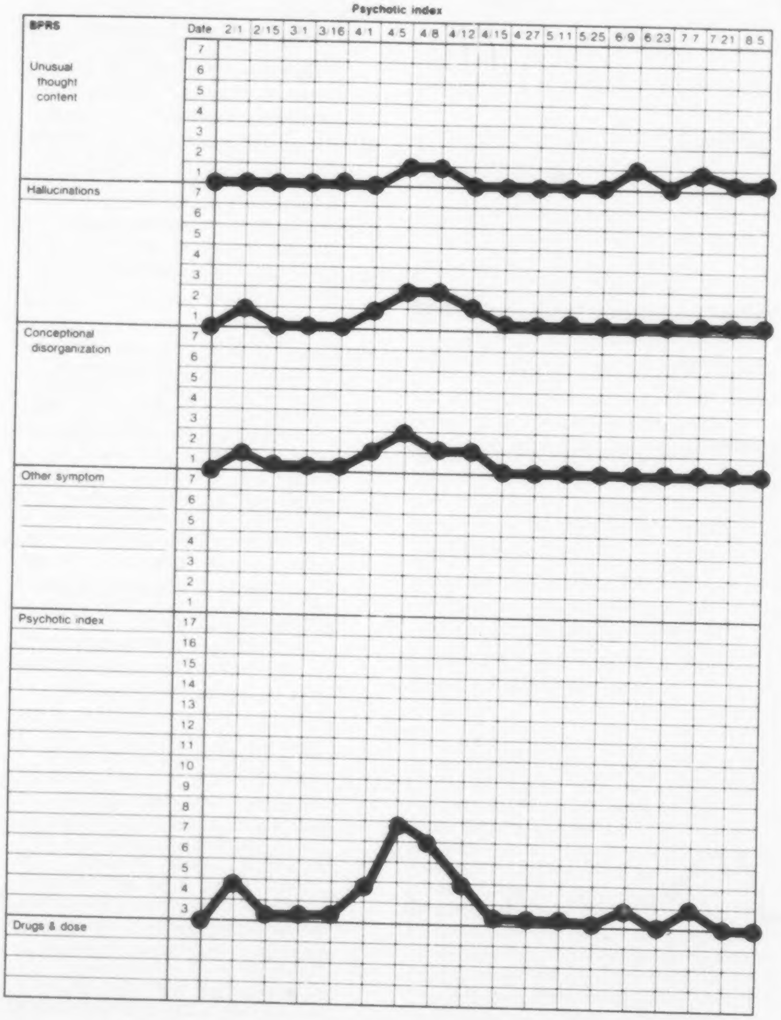
Figure 2. Graph of psychotic index for "Bill"



BPRS = Brief Psychiatric Rating Scale.

Case 3. When Joan was first seen in the hospital, she was administered the BPRS by one of the aftercare clinic psychologists. The unusual thought content item was rated a 7, the highest possible rating, due to Joan's almost total preoccupation with messages from TV, radio, and computers. She also reported hallucinations several times a day, thereby warranting a 6 on that item. She was taking oral Prolixin, 20 mg daily. By hospital discharge, her hallucinations had remitted totally. When she came to the aftercare clinic for the first time 2 days after her discharge from the hospital, Joan told her case manager that she still was getting messages but only from the radio. She did not think about them and they did not interfere with her functioning, which warrants a rating of 4 on the BPRS Unusual Thought Content item. After she had been seen for 4 weeks as an outpatient, the case manager was concerned about the persistence of Joan's delusions of reference about messages from the radio even though they were at a low level of intensity. The finding of persisting delusional thinking was discussed with one of the aftercare clinic psychiatrists and a decision was reached to increase Joan's daily Prolixin dosage to 30 mg. Six weeks later, another BPRS revealed no change in her ratings. With the patient's agreement, she was switched to biweekly 20 mg i.m. Prolixin to rule out compliance as a factor in her persisting symptomatology. When Joan came in for her injection, she announced she would not take her shot. She stated that the injections were embarrassing to her and that she wanted pills. After reviewing the BPRS ratings and finding no additional therapeutic impact from the increased dosage or from i.m. administration, the case manager and psychiatrist agreed to place her back on oral Prolixin at the original postdischarge dosage. (See figure 4.)

Figure 3. Graph of psychotic index for "John"



BPRS = Brief Psychiatric Rating Scale.

Conclusion

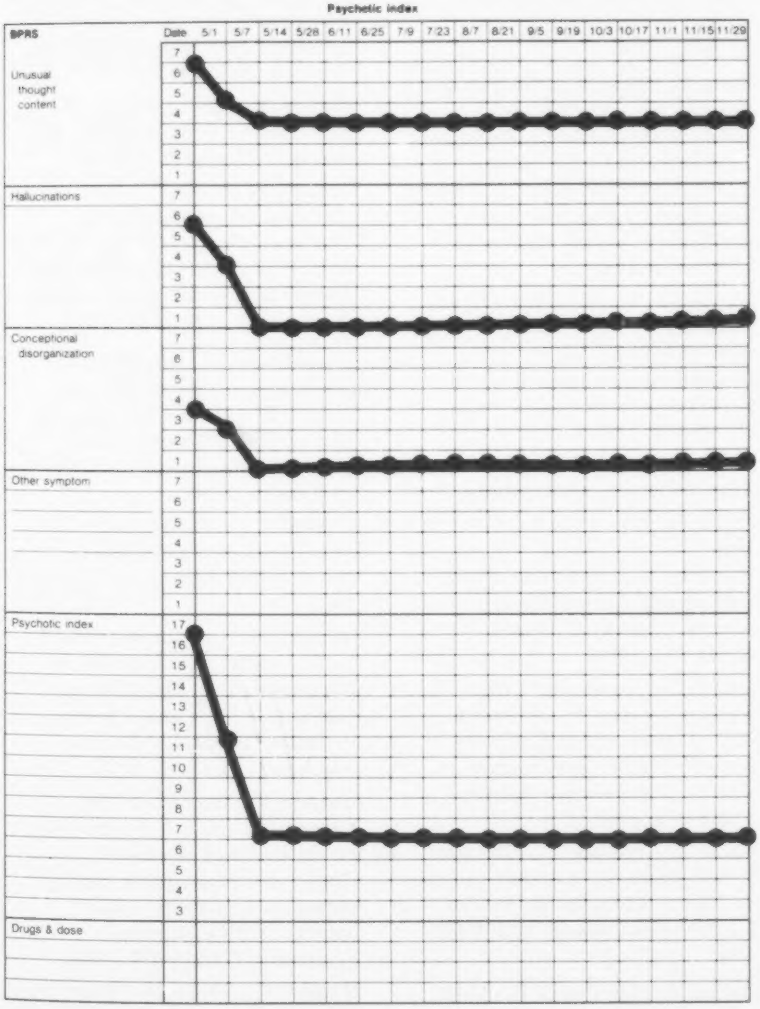
In treatment studies, psychopathology has often been the sole outcome variable used to evaluate effectiveness. In particular, relapse has been overused as the sole criterion for evaluating treatment program. Psychopathology offers only a limited view of the overall functioning of schizophrenic patients. In a study of outcome, Strauss and Carpenter (1974) followed 85 schizophrenic patients for 2 years after a hospitalization. They found that

> outcome is not a singular phenomenon but that there are several areas of outcome function—interrelated but also partly independent of each other. [p. 37]

They included social relations and employment status in their multidimensional approach to evaluating outcome. A full spectrum of functional behaviors needs to be assessed by rehabilitation workers throughout the patient's participation in a program (see Wallace, this issue). Rehabilitation staff members may view their responsibilities and efforts as more directed toward improving social functioning than symptom reduction. However, they are interrelated. Exacerbations of schizophrenic symptomatology have been clearly related to a host of social variables (Lukoff et al. 1984). Rehabilitation is one source of social stimulation that may improve clinical status or, at times, exacerbate symptoms.

Psychopathology assessment instruments can be used to improve the rehabilitation of schizophrenic patients. When psychopathology is monitored through structured interviews developed originally for research, highly symptomatic patients can be diverted from intensive rehabilitation programs, negative reactions to rehabilitation efforts can be identified, and prodromal signs of impending relapse detected. Similarly, individual and program-wide benefits from rehabilitation can also be accurately and convincingly documented.

Figure 4. Graph of psychotic index for "Joan"



BPRS = Brief Psychiatric Rating Scale.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Anderson, C.; Reiss, D.; and Hogarty, G. *Schizophrenia and the Family.* New York: Guilford Press, 1986.

Andreasen, N. Negative symptoms in schizophrenia: Definition and reliability. *Archives of General Psychiatry,* 39:784–788, 1982.

Andreasen, N., and Olsen, S. Negative *v.* positive schizophrenia: Definition and validation. *Archives of General Psychiatry,* 39:789–794, 1982.

Battle, C.; Imber, S.; Hoehn-Saric, R.; Stone, A.; Nash, E.; and Frank, J. Target complaints as criteria of improvement. *American Journal of Psychotherapy,* 20:184–192, 1966.

Bennett, D. Social forms of psychiatric treatment. In: Wing, J.K., ed. *Schizophrenia: Towards a New Synthesis.* New York: Grune & Stratton, Inc., 1978. pp. 211–232.

Bigelow, L., and Murphy, D.L. *"Guidelines and Anchor Points for Modified BPRS."* Unpublished manuscript, NIMH Intramural Research Program, Saint Elizabeths Hospital, 1978.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias.* (1911) Translated by J. Zinkin. New York: International University Press, 1950.

Bleuler, M. The long-term course of the schizophrenic psychoses. *Psychological Medicine,* 4:244–254, 1974.

Brown, G., and Birley, J. Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behavior,* 9:203–214, 1968.

Brown, G.; Birley, J.; and Wing, J. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, 121:241–258, 1972.

Carpenter, W.T, Jr., and Heinrichs, D. Early intervention, time-limited, targeted pharmacotherapy of schizophrenia. *Schizophrenia Bulletin*, 9:533–542, 1983.

Ciompi, L. Catamnestic long-term study on the course of life and aging of schizophrenics. *Schizophrenia Bulletin*, 6:606–618, 1980.

Crow, T. The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin*, 11:471–485, 1985.

Derogatis, L.; Lipman, R.; and Covi, L. SCL-90: An outpatient psychiatric rating scale—Preliminary report. *Psychopharmacology Bulletin*, 9:13–17, 1973.

Docherty, J.; van Kammen, D.; Siris, S.; and Marder, S. Stages of onset of schizophrenic psychosis. *American Journal of Psychiatry*, 136:420–426, 1978.

Falloon, I. Relapse: A reappraisal of assessment of outcome in schizophrenia. *Schizophrenia Bulletin*, 10:293–299, 1984.

Falloon, I.; Boyd, J.; and McGill, C. *Family Care of Schizophrenia: A Problem-Solving Approach to the Treatment of Mental Illness*. New York: Guilford Press, 1984.

Goldberg, S.; Schooler, N.; Hogarty, G.; and Roper, M. Prediction of relapse in schizophrenic outpatients treated by drug and sociotherapy. *Archives of General Psychiatry*, 34:171–184, 1977.

Guy, W. *ECDEU Assessment Manual for Psychopharmacology*. DHEW Pub. No. (ADM) 76–338. Rockville, MD: National Institute of Mental Health, 1976.

Herz, M., and Melville, C. Relapse in schizophrenia. *American Journal of Psychiatry*, 137:801–805, 1980.

Herz, M.; Szymanski, H.; and Simon, J. Intermittent medication for stable schizophrenic outpatients. *American Journal of Psychiatry*, 139:918–922, 1982.

Hogarty, G. Depot neuroleptics: The relevance of psychosocial factors. *Journal of Clinical Psychiatry*, 45:36–42, 1984.

Kane, J.; Rifkin, A.; Woerner, M.; Reardon, G.; Sarantakos, S.; Schiebel, D.; and Ramos-Lorenzi, J. Low-dose neuroleptic treatment of outpatient schizophrenics: I. Preliminary results for relapse rates. *Archives of General Psychiatry*, 40:893–896, 1983.

Krawiecka, M.; Goldberg, D.; and Vaughan, M. A standardized psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, 55:299–308, 1977.

Leff, J., and Wing, J. Trial of maintenance therapy in schizophrenia. *British Medical Journal*, 3:599–604, 1971.

Liberman, R., and Foy, D. Psychiatric rehabilitation of chronic mental patients. *Psychiatric Annals*, 13:539–545, 1983.

Lukoff, D.; Snyder, K.; Ventura, J.; and Nuechterlein, K. Life events, familial stress, and coping in the developmental course of schizophrenia. *Schizophrenia Bulletin*, 10:258–292, 1984.

Lukoff, D.; Wallace, C.; Liberman, R.; and Burke, K. A holistic program for chronic schizophrenic patients. *Schizophrenia Bulletin*, 12:274–282, 1986.

Marder, S.; Van Putten, T.; Mintz, J.; Labell, M.; McKenzie, J.; and Faltico, G. Maintenance therapy in schizophrenia: New findings. In: Kane, J., ed. *Drug Maintenance Strategies in Schizophrenia*. Washington, DC: American Psychiatric Press, 1984. pp. 31–49.

May, P.R.A. Strategies for applied researchers in chronic schizophrenia: Choosing the right treatment and measuring outcome. In: Baxter, C., and Melnechuck, T., eds. *Perspectives in Schizophrenia Research*. New York: Raven Press, 1980. pp. 317–328.

Mendel, W. *Schizophrenia: The Experience and Its Treatment*. San Francisco: Jossey-Bass, 1976.

Mintz, J.; Luborsky, L.; and Christoph, P. Measuring the outcomes of psychotherapy: Findings of the Penn psychotherapy project. *Journal of Consulting and Clinical Psychology*, 47:319–334, 1979.

Nuechterlein, K.; Miklowitz, D.; Ventura, J.; Stoddard, M.; and Lukoff, D. "Manual for Assessing Outcome in Schizophrenic and Bipolar-Manic Patients." Unpublished manuscript, 1985.

Overall, J., and Gorham, D. The Brief Psychiatric Rating Scale. *Psychological Reports*, 10:799–812, 1962.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: II. Relationships between predictor and outcome variables. *Archives of General Psychiatry*, 31:37–42, 1974.

Vaughn, C., and Leff, J. The influence of family and social factors on the course of psychiatric illness: A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129:125–137, 1976.

Vaughn, C.; Snyder, K.; Jones, S.; Freeman, W.; and Falloon, I. Family factors in schizophrenic relapse: A

California replication of the British research on expressed emotion. *Archives of General Psychiatry,* 41:1169–1177, 1984.

Wallace, C. The social skills training project of the Mental Health Clinical Research Center for the Study of Schizophrenia. In: Curran, J., and Monti, P., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment.* New York: Guilford Press, 1982. pp. 57–89.

Wing, J. Social treatments of mental illness. In: Shepherd, M., and Davies, D., eds. *Studies of Psychiatry.* London: Oxford University Press, 1968.

Wing, J. Social influences on the course of schizophrenia. In: Wynne, L.; Cromwell, R.; and Matthysse, S., eds. *The Nature of Schizophrenia: New Approaches to Research and Treatment.* New York: John Wiley & Sons, 1978. pp. 599–616.

Wing, J.; Cooper, J.; and Sartorius, N. *The Description and Classification of Psychiatric Symptoms: An Instruction Manual for PSE and Catego System.* London: Cambridge University Press, 1974.

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Appendix A. Manual for Expanded Brief Psychiatric Rating Scale (BPRS)

Developed by David Lukoff, Keith H. Nuechterlein, and Joseph Ventura

The following guidelines are designed for use with an outpatient psychiatric population. This manual contains an interview schedule, symptom definitions, and specific anchor points for rating symptoms. The ratings for items 1-10 and 19-22 are based on the patient's answers to the interviewer's questions. The time frame for these items is the past 2 weeks. Items 11-18, 23, and 24 are based on the patient's behavior during the interview and the time frame covered is the interview period only. When psychotic symptoms (e.g., hallucinations and unusual thought content) have had a period of exacerbation lasting at least 1 day, the rating should reflect mainly the peak period. When the anchor point definitions contain an *"or,"* the patient is assigned the *highest* rating that applies, e.g., if a patient has hallucinations persistently throughout the day (a rating of 7) but the hallucinations only interfere with functioning to a limited extent (a rating of 5), a rating of 7 is given. An additional guideline which is often helpful involves the distinction between pathological and nonpathological intensities of symptoms. Ratings of 2-3 indicate a nonpathological intensity of a symptom whereas ratings of 4-7 indicate a pathological intensity of that symptom.

Rate items 1-10 on the basis of patient's self-report

1. **Somatic concern:** Degree of concern over present bodily health. Rate the degree to which physical health is perceived as a problem by the patient, whether complaints have realistic bases or not

2-3	Mild	Occasional complaint or expression of concern
4-5	Moderate	Frequent expressions of concern or exaggerations of existing ills. Some preoccupation. Not delusional
6-7	Severe	Preoccupied with physical complaints *or* somatic delusions

Have you been concerned about your physical health?
Have you had any physical illness or seen a medical doctor?

2. **Anxiety:** Reported apprehension, tension, fear, panic or worry. Rate *only* patient's statements—not observed anxiety which is rated under **tension**.

2	Very mild	Reports feeling worried more than usual or some discomfort due to worry
3	Mild	Worried frequently but can turn attention to other things
4	Moderate	Worried most of the time and cannot turn attention to other things easily but no impairment in functioning *or* occasional anxiety with automatic accompaniment but no impairment in functioning
5	Moderately severe	Frequent periods of anxiety with autonomic accompaniment *or* some areas of functioning are disrupted by anxiety or constant worry
6	Severe	Anxiety with autonomic accompaniment most of the time *or* many areas of functioning are disrupted by anxiety or constant worry
7	Extremely severe	Constantly anxious with autonomic accompaniment *or* most areas of functioning are disrupted by anxiety or constant worry

Have you felt worried or anxious?
Do unpleasant thoughts constantly go round and round in your mind?

Did your heart beat fast (or sweating, trembling, choking)?
Has it interfered with your ability to perform your usual activities/work?

3. **Depression:** Include *mood*—sadness, unhappiness, anhedonia; and *cognitions*—preoccupation with depressing topics (can't switch attention to TV, conversations), hopelessness, loss of self-esteem (dissatisfied or disgusted with self). Do not include vegetative symptoms, e.g., motor retardation, early waking

2	Very mild	Reports feeling sad/unhappy/depressed more than usual
3	Mild	Same as 2, but can't snap out of it easily
4	Moderate	Frequent periods of feeling very sad, unhappy, moderately depressed, but able to function with extra effort
5	Moderately severe	Frequent periods of deep depression *or* some areas of functioning are disrupted by depression
6	Severe	Deeply depressed most of the time *or* many areas of functioning are disrupted by depression
7	Extremely severe	Constantly deeply depressed *or* most areas of functioning are disrupted by delusional thinking

Have you felt unhappy or depressed?
How much of the time?
Are you able to switch your attention to more pleasant topics when you want to?
Have your interests in work, hobbies, social or recreational activities changed?
Has it interfered with your ability to perform your usual activities/work?

4. **Guilt:** Overconcern or remorse for past behavior. Rate *only* patient's statements—do not infer guilt feelings from depression, anxiety, or neurotic defenses

2–3	Mild	Worries about having failed someone or at something. Wishes to have done things differently
4–5	Moderate	Preoccupied about having done wrong or injured others by doing or failing to do something
6–7	Severe	Delusional guilt *or* obviously unreasonable self-reproach

Have you been thinking about past problems?
Do you tend to blame yourself for things that have happened?
Have you done anything you're still ashamed of?

5. **Hostility:** Animosity, contempt, belligerence, threats, arguments, tantrums, property destruction, fights, and any other expression of hostile attitudes or actions. Do not infer hostility from neurotic defenses, anxiety, or somatic complaints. Do not include isolated appropriate anger

2	Very mild	Irritable, grumpy
3	Mild	Argumentative, sarcastic, *or* feels angry
4	Moderate	Overtly angry on several occasions *or* yelled at others
5	Moderately severe	Has threatened, slammed about or thrown things
6	Severe	Has assaulted others but with no harm likely, e.g., slapped, pushed, *or* destroyed property (knocked over furniture, broken windows)
7	Extremely severe	Has attacked others with definite possibility of harming them or with actual harm, e.g., assault with hammer or weapon

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How have you been getting along with people (family, board-and-care residents, co-workers)?
Have you been irritable or grumpy lately?
Have you been involved in any arguments or fights?

6. **Suspiciousness:** Expressed or apparent belief that other persons have acted maliciously or with discriminatory intent. Include persecution by supernatural or other nonhuman agencies (e.g., the devil)

2–3	Mild	Seems on guard. Unresponsive to "personal" questions. Describes incidents where other persons have harmed or wanted to harm him/her that sound plausible. Patient feels as if others are laughing at or criticizing him/her in public
4–5	Moderate	Says other persons are talking about him/her maliciously *or* says others intend to harm him/her. Beyond likelihood of plausibility but not delusional
6–7	Severe	Delusional. Speaks of Mafia plots, the FBI, or others poisoning food

Do you ever feel uncomfortable as if people are watching you?
Is anyone trying to harm or interfere with you in any way?
Are you concerned about anybody's intentions toward you?
Have you felt that any people are out to get you?

7. **Unusual thought content:** Unusual, odd, strange, or bizarre thought content. Rate the degree of unusualness, not the degree of disorganization of speech. Delusions are patently absurd, clearly false, or bizarre ideas verbally expressed. Include thought insertion, withdrawal, and broadcasting. Include grandiose, somatic, and persecutory delusions even if rated elsewhere

2	Very mild	Ideas of reference (people stare/laugh at him/her). Ideas of persecution (people mistreat him/her). Unusual beliefs in psychic powers, spirits, UFO's. Not strongly held. Some doubt
3	Mild	Same as 2 with full conviction but not delusional
4	Moderate	Delusion present but not strongly held—functioning not disrupted; *or* encapsulated delusion with full conviction—functioning not disrupted
5	Moderately severe	Full delusion(s) present with some preoccupational *or* some areas of functioning disrupted by delusional thinking
6	Severe	Full delusion(s) present with much preoccupation *or* many areas of functioning disrupted by delusional thinking
7	Extremely severe	Full delusion(s) present with almost total preoccupation *or* most areas of functioning disrupted by delusional thinking

Have things or events had special meanings for you?
Did you see any references to yourself on TV or in the newspapers?
Do you have a special relationship with God?
How do you explain the things that have been happening (specify)?
Have you felt that you were under the control of another person or force?

8. **Grandiosity:** Exaggerated self-opinion, self-enhancing conviction of special abilities, powers, or identity as someone rich or famous. Rate only patient's statements about self, not demeanor

2	Very mild	Feels great and denies obvious problems
3	Mild	Exaggerated self-opinion beyond abilities and training
4	Moderate	Inappropriate boastfulness, claims to be "brilliant," understands how everything works

5	Moderately severe	Claims to be great musician who will soon make recordings or will soon make patentable inventions—but not delusional
6	Severe	Delusional—claims to have special powers like ESP, to have millions of dollars, made movies, invented new machines, worked at jobs when it is known that he was never employed in these capacities
7	Extremely severe	Delusional—claims to have been appointed by God to run the world, controls the future of the world, is Jesus Christ, or President of the U.S.

Is there a special purpose or mission to your life?
Do you have any special powers or abilities?
Have you thought that you might be somebody rich or famous?

9. **Hallucinations:** Reports of perceptual experiences in the absence of external stimuli. When rating degree to which functioning is disrupted by hallucinations, do not include preoccupation with the content of the hallucinations. Consider only disruption due to the hallucinatory experience. Include thoughts aloud-*gedankenlautwerten*

2	Very mild	While resting or going to sleep, sees visions, hears voices, sounds, or whispers in absence of external stimulation, but no impairment in functioning
3	Mild	While in a clear state of consciousness, hears nonverbal auditory hallucinations (e.g., sounds or whispers) or sees illusions (e.g., faces in shadows) on no more than two occasions and with no impairment in functioning
4	Moderate	Occasional verbal, visual, olfactory, tactile, or gustatory hallucinations (1–3 times) but no impairment in functioning *or* frequent nonverbal hallucinations/visual illusions
5	Moderately severe	Daily *or* some areas of functioning are disrupted by hallucinations
6	Severe	Several times a day *or* many areas of functioning are disrupted by hallucinations
7	Extremely severe	Persistent throughout the day *or* most areas of functioning are disrupted by hallucinations

Have you heard any sounds or people talking to you or about you when there has been nobody around?
Have you seen any visions or smelled any smells others don't seem to notice?
Have these experiences interfered with your ability to perform your usual activities/work?

10. **Disorientation:** Does not comprehend situations or communications. Confusion regarding person, place, or time

2–3	Mild	Occasionally seems muddled, bewildered, or mildly confused
4–5	Moderate	Seems confused regarding person, place, or time. Has difficulty remembering facts—e.g., where born—or recognizing people. Mildly disoriented as to time or place
6–7	Severe	Grossly disoriented as to person, place, or time

May I ask you one or two standard questions we ask everybody?
How old are you?
What is the date?
What is this place called?

Rate items 11–18 on the basis of observed behavior and speech

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11. **Conceptual disorganization:** Degree to which speech is confused, disconnected, or disorganized. Rate tangentiality, circumstantiality, sudden topic shifts, incoherence, derailment, blocking, neologisms, and other speech disorders. Do not rate *content* of speech. Consider the first 15 minutes of the interview

2	Very mild	Peculiar use of words, rambling but speech is comprehensible
3	Mild	Speech a bit hard to understand or make sense of due to tangentiality, circumstantiality, or sudden topic shifts
4	Moderate	Speech difficult to understand due to tangentiality, circumstantiality, or topic shifts on many occasions *or* 1–2 instances of severe impairment, e.g., incoherence, derailment, neologisms, blocking
5	Moderately severe	Speech difficult to understand due to circumstantiality, tangentiality, or topic shifts most of the time *or* 3–5 instances of severe impairment
6	Severe	Speech is incomprehensible due to severe impairments most of the time
7	Extremely severe	Speech is incomprehensible throughout interview

12. **Excitement:** Heightened emotional tone, increased reactivity, impulsivity

2–3	Mild	Increased emotionality. Seems keyed up, alert
4–5	Moderate	Reacts to most stimuli whether relevant or not with considerable intensity. Short attention span. Pressured speech
6–7	Severe	Marked overreaction to all stimuli with inappropriate intensity, restlessness, impulsiveness. Cannot settle down or stay on task

13. **Motor retardation:** Reduction in energy level evidenced in slowed movements and speech, reduced body tone, decreased number of spontaneous body movements. Rate on the basis of observed behavior of the patient only. Do *not* rate on the basis of patient's subjective impression of his/her own energy level. Rate regardless of medication effects

2–3	Mild	Noticeably slowed or reduced movements or speech compared to most people
4	Moderate	Large reduction or slowness in movements or speech
5	Moderately severe	Seldom moves or speaks spontaneously *or* very mechanical stiff movements
6	Severe	Does not move or speak unless prodded or urged
7	Extremely severe	Frozen, catatonic

14. **Blunted affect:** Restricted range in emotional expressiveness of face, voice, and gestures. Marked indifference or flatness even when discussing distressing topics

2–3	Mild	Some loss of normal emotional responsiveness
4	Moderate	Emotional expression very diminished, e.g., doesn't laugh, smile, or react with emotion to distressing topics except on 2 or 3 occasions during interview
5	Moderately severe	Emotional expression extremely diminished, e.g., doesn't laugh, smile, or react with emotions to distressing topics except for a maximum of 1 time during interview
6	Severe	Mechanical in speech, gestures, and expression
7	Extremely severe	Frozen expression and flat speech. Shows no feeling

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15. **Tension:** Observable physical and motor manifestations of tension, nervousness, and agitation. Self-reported experiences of tension should be rated under the item on anxiety

2–3	Mild	Seems tense. Tense posture, nervous mannerisms some of the time
4–5	Moderate	Seems anxious. Fearful expression, trembling, restless
6–7	Severe	Continually agitated, pacing, hand wringing

16. **Mannerisms and posturing:** Unusual and bizarre behavior, stylized movements, or acts, or any postures which are clearly uncomfortable or inappropriate. Exclude obvious manifestations of medication side effects

2–3	Mild	Eccentric or odd mannerisms or activity that ordinary persons would have difficulty explaining, e.g., grimacing, picking
4–5	Moderate	Mannerisms or posturing maintained for 5 seconds or more that would make the patient stand out in a crowd as weird or crazy
6–7	Severe	Posturing, smearing, intense rocking, fetal positioning, strange rituals that dominate patient's attention and behavior

17. **Uncooperativeness:** Resistance, unfriendliness, resentment, or lack of willingness to cooperate with the interview. Rate only uncooperative behavior observed during interview, not uncooperativeness with relatives

2–3	Mild	Gripes or tries to avoid complying but goes ahead without argument
4–5	Moderate	Verbally resists, or negativistic but eventually complies. Some information withheld
6–7	Severe	Refuses to cooperate. Physically resistant

18. **Emotional withdrawal:** Deficiency in patient's ability to relate emotionally during interview situation. Use your own feeling as to the presence of an "invisible barrier" between patient and interviewer

2–3	Mild	Tends not to show emotional involvement with interviewer but responds when approached
4–5	Moderate	Emotional contact not present most of the interview. Responds only with minimal affect
6–7	Severe	Actively avoids emotional participation. Unresponsive or yes/no answers. May leave when spoken to or just not respond at all

19. **Suicidality:** Expressed desire, intent, or actual actions to harm or kill self

2	Very mild	Occasional feelings of being tired of living. No overt suicidal thoughts
3	Mild	Occasional suicidal thoughts without intent or specific plan. *Or* feels he would be better off dead
4	Moderate	Suicidal thoughts frequent, without intent or plan
5	Moderately severe	Many fantasies of suicide by various methods. May seriously consider making specific attempt with specific time or worked out plan. *Or* impulsive suicide attempt using nonlethal method or in full view of potential saviors.
6	Severe	Wants to kill self. Searches for appropriate means and time. *Or* potentially medically serious suicide attempt with patient knowledge of possible rescue
7	Extremely severe	Specific suicidal plan and intent (e.g., "as soon as ________, I will do it by doing X"). *Or* suicide attempt characterized by plan patient thought was lethal or attempt in secluded environment

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Have you felt that life wasn't worth living?
Have you thought about harming or killing yourself?
Do you have a specific plan?

20. **Self-neglect:** Hygiene, appearance, or eating behavior below usual expectations, below socially acceptable standards, or life threatening.

2	Very mild	Hygiene/appearance somewhat below usual standards, e.g., shirt out of pants, buttons unbuttoned
3	Mild	Hygiene/appearance much below usual standards, e.g., clothing disheveled and stained, hair uncombed
4	Moderate	Hygiene/appearance below socially acceptable standards, e.g., large holes in clothing, bad breath, hair uncombed, oily, eating irregular and poor
5	Moderately severe	Hygiene highly erratic and poor, e.g., extreme body odor, eating very irregular and poor, e.g., eating only potato chips
6	Severe	Hygiene and eating potentially life threatening, e.g., eats and/or bathes only when prompted
7	Extremely severe	Hygiene and eating life threatening, e.g., does not eat or engage in hygiene

How often do you take showers; change your clothes?
Has anyone (parents/staff) complained about your grooming or dress?
Do you eat regular meals?

21. **Bizarre behavior:** Reports of behaviors that are odd, unusual, or psychotically criminal. Not limited to interview period. Exclude mannerisms and posturing, verbalizations with bizarre content

2	Very mild	Slightly odd behavior, e.g., hoarding food in private, wears gloves indoors
3	Mild	Peculiar behavior, e.g., talking loudly in public, fails to make appropriate eye contact when talking with others
4	Moderate	Moderately unusual, e.g., bizarre dress or makeup, "preaching" to strangers, fixated staring into space while in public, collecting garbage
5	Moderately severe	Highly unusual, e.g., wandering streets aimlessly, eating nonfoods, fixated staring in a socially disruptive way
6	Severe	Unusual petty crimes, e.g., directing traffic, public nudity, contacting authorities about imaginary crimes
7	Extremely severe	Unusual serious crimes, e.g., setting fires, asocial theft, kidnapping committed in a bizarre fashion or for bizarre reasons

Have you done anything that has attracted the attention of others?
Have you done anything that could have gotten you into trouble with the police?
Have you done anything that seemed unusual or disturbing to others?

22. **Elevated mood:** A pervasive, sustained, and exaggerated feeling of well-being, cheerfulness, euphoria (implying a pathological mood), optimism that is out of proportion to the circumstances. Do *not* infer elation from increased activity or from grandiose statements alone

2	Very mild	Seems to be unusually happy, cheerful without much reason
3	Mild	Some unaccountable feelings of well-being
4	Moderate	Reports excessive or unrealistic feelings of well-being, cheerfulness, confidence, or optimism *inappropriate to circumstances*, some of the time. May frequently joke, smile, be giddy, or overly enthusiastic *or* few instances of marked elevated mood with euphoria

5	Moderately severe	Reports excessive or unrealistic feelings of well-being, confidence or optimism *inappropriate to circumstances* much of the time. May describe feeling "on top of the world," "like everything is falling in place," or "better than ever before," *or* several instances of marked elevated mood with euphoria
6	Severe	Mood definitely elevated almost constantly throughout interview and inappropriate to content, or many instances of marked elevated mood with euphoria
7	Extremely severe	Seems almost intoxicated, laughing, joking, giggling, constantly euphoric, feeling invulnerable, all inappropriate to immediate circumstances

Have you been feeling cheerful and on top of the world without any reason?
How long does that last?
Have you felt so good or high that other people make comments to you about it?

23. **Motor hyperactivity:** Increase in energy level evidenced in more frequent movement and/or rapid speech. (Note: In making this rating, consider the 15-minute period of most severe symptomatology)

2	Very mild	Some restlessness, difficulty sitting still, lively facial expressions, or somewhat talkative
3	Mild	Occasionally very restless, definite increase in motor activity, lively gestures, 1-3 brief instances of pressured speech
4	Moderate	Very restless, fidgety, excessive facial expressions, or nonproductive and repetitious motor movements. Much pressured speech, up to one-third of interview
5	Moderately severe	Frequently restless, fidgety. Many instances of excessive nonproductive and repetitious motor movements. On the move most of the time. Frequent pressured speech, difficult to interrupt. Rises on 1-2 occasions to pace
6	Severe	Excessive motor activity, restlessness, fidgety, loud tapping, noisy, etc., throughout most of the interview. Constant pressured speech with only few pauses. Speech can only be interrupted with much effort. Rises on 3-4 occasions to pace
7	Extremely severe	Constant excessive motor activity throughout entire interview, e.g., constant pacing, constant pressured speech with no pauses, interviewee can only be interrupted briefly and only small amounts of relevant information can be obtained

24. **Distractibility:** Degree to which observed sequences of speech and actions are interrupted by minimal external stimuli. Include distractibility due to intrusions of visual or auditory hallucinations. Interviewee's attention may be drawn to noise in adjoining room, books on a shelf, interviewer's clothing, etc. Do not include preoccupation due to delusions or other thoughts.

2	Very mild	Generally can focus on interviewer's questions with only 1 distraction or inappropriate shift of attention of brief duration due to minimal external stimuli
3	Mild	Same as above but occurs 2 times
4	Moderate	Responsive to irrelevant stimuli in the room or in the environment much of the time
5	Moderately severe	Same as above, but now interferes with comprehensibility of speech
6	Severe	Extremely difficult to conduct interview or pursue a subject due to preoccupation with unimportant and irrelevant stimuli or almost totally incomprehensible because attention shifts rapidly between various irrelevant external stimuli and interviewer's questions

7	Extremely severe	Impossible to conduct interview due to preoccupation with unimportant and irrelevant external stimuli

Brief Psychiatric Rating Scale

Patient's name________________________ Date____________ Interviewer's name________________________
Hospital ____________________________ Ward____________ Date of admission ________________________

Instructions: This form consists of 24 symptom constructs, each to be rated on a 7-point scale of severity ranging from "not present" to "extremely severe." If a specific symptom is not rated, mark "NA" (not assessed). Circle the number headed by the term that best describes the patient's present condition

NA	1	2	3	4	5	6	7
Not assessed	Not present	Very mild	Mild	Moderate	Moderately severe	Severe	Extremely severe

1.	Somatic concern	NA	1	2	3	4	5	6	7
2.	Anxiety	NA	1	2	3	4	5	6	7
3.	Depression	NA	1	2	3	4	5	6	7
4.	Guilt	NA	1	2	3	4	5	6	7
5.	Hostility	NA	1	2	3	4	5	6	7
6.	Suspiciousness	NA	1	2	3	4	5	6	7
7.	Unusual thought content	NA	1	2	3	4	5	6	7
8.	Grandiosity	NA	1	2	3	4	5	6	7
9.	Hallucinations	NA	1	2	3	4	5	6	7
10.	Disorientation	NA	1	2	3	4	5	6	7
11.	Conceptual disorganization	NA	1	2	3	4	5	6	7
12.	Excitement	NA	1	2	3	4	5	6	7
13.	Motor retardation	NA	1	2	3	4	5	6	7
14.	Blunted affect	NA	1	2	3	4	5	6	7
15.	Tension	NA	1	2	3	4	5	6	7
16.	Mannerisms and posturing	NA	1	2	3	4	5	6	7
17.	Uncooperativeness	NA	1	2	3	4	5	6	7
18.	Emotional withdrawal	NA	1	2	3	4	5	6	7
19.	Suicidality	NA	1	2	3	4	5	6	7
20.	Self-neglect	NA	1	2	3	4	5	6	7
21.	Bizarre behavior	NA	1	2	3	4	5	6	7
22.	Elated mood	NA	1	2	3	4	5	6	7
23.	Motor hyperactivity	NA	1	2	3	4	5	6	7
24.	Distractibility	NA	1	2	3	4	5	6	7

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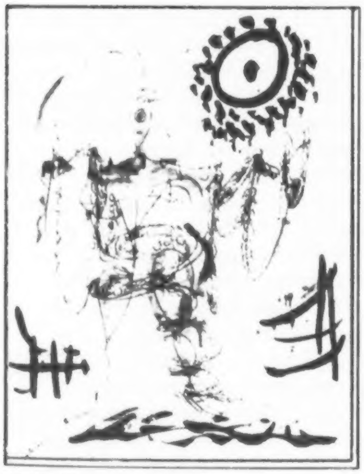

by LEONARD B. STANLEY

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Functional Assessment in Rehabilitation

by Charles J. Wallace

Abstract

The instruments that assess the functional living skills of chronically mentally ill individuals are reviewed. The instruments range from those that provide relatively global assessments of major life roles such as wage earner and student, to those that provide relatively specific assessments of behaviors such as personal hygiene and money management skills. The content, format, psychometric characteristics, advantages, and disadvantages of each instrument are presented in detail so that the reader can select the instrument that best suits the purposes of the assessment.

Anthony and Nemec (1984) have defined psychiatric rehabilitation as treatment that teaches chronically mentally ill individuals "the physical, emotional, and intellectual skills necessary to live, learn, and work in their own particular environments" (p. 377). The value of psychiatric rehabilitation is apparent when it is viewed within the context of a comprehensive model of the course and outcome of major mental illness, presented in figure 1 of the Anthony and Liberman article in this issue.

The model proposes that an episode of illness is the result of an interaction of personal and environmental factors. The relevant personal factors include not only the underlying biological and psychological abnormalities that define an individual's vulnerability to develop an illness, but also the amount and competency of his/her effort to cope with environmental demands. The relevant environmental factors include not only the number and severity of these demands, but also the amount of support available to the individual to implement his/her coping responses. Incompetent coping, a high degree of vulnerability, numerous and stressful demands, and inadequate support summate to produce an episode of illness. Psychiatric rehabilitation is designed to reduce the severity and frequency of the episodes of illness primarily by increasing the competency of an individual's coping responses. Secondarily, psychiatric rehabilitation may reduce illness either by reducing the number and severity of environmental demands, or by increasing the amount of environmental support.

As indicated in figure 1, the process of psychiatric rehabilitation involves assessment of an individual's competencies, identification of the environmental demands and support, and implementation of treatment methods that will improve competencies, reduce demands, and increase supports. The success of this process depends as much on the reliability and validity of the assessment instruments as on the effectiveness and efficiency of the available treatment methods. The assessment information pinpoints the incompetent coping responses for which treatment will be necessary, and provides an empirical basis for evaluating the success of the process both for individuals and for entire programs (Anthony and Farkas 1982; Liberman 1982; Taylor, Liberman, and Agras 1982; Anthony and Cohen 1984; Anthony, Cohen, and Nemec 1986).

While this article reviews scales and instruments constructed for multidimensional assessment, most rehabilitation efforts proceed from individualized evaluation and goals using more focal, target behaviors.

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Rehabilitation interventions usually focus on idiosyncratic behavioral targets that are then monitored for decisions to continue, change, or terminate the interventions. Methods of assessing and recording various targeted behavioral excesses and deficits have been reviewed elsewhere (Wallace 1981) and will not be covered in this article.

The purpose of this article is to review the assessment instruments that are suitable for measuring the functional living competencies of chronically mentally ill individuals. Excluded from this review are instruments whose primary purpose is to assess psychopathology such as the Inpatient Multidimensional Psychiatric Scale (Lorr and Klett 1966) and the Brief Psychiatric Rating Scale (Overall and Gorham 1962); instruments whose value is primarily "historical" such as the Normative Social Adjustment Scale (Barrabee, Barrabee, and Finesinger 1955) and the Social Ineffectiveness Scale (Parloff, Kelman, and Frank 1954); instruments that have been constructed for use in a particular treatment setting such as Behavior on Ward Scale (Rostow and Smith 1975); and instruments that provide only global assessments of competencies such as the Strauss-Carpenter Outcome Scale (Strauss and Carpenter 1972) and the Global Assessment Scale (Endicott et al. 1976).

Also excluded are instruments that assess the functional living competencies of children and developmentally disabled individuals (for a comprehensive review, see Walls et al. 1977). Since these instruments assess relatively basic skills, they are appropriate only for chronically mentally ill individuals who are extremely regressed. For example, The Pyramid Scales (Cone 1985) assess the competencies of children and developmentally disabled individuals in 20 areas of functioning such as auditory responsiveness (e.g., "responds to sudden loud noise"), social interaction (e.g., "asks time-related questions such as what time is it"), eating (e.g., "chews all food unassisted"), and dressing (e.g., "zips, snaps, and buttons without help"). Clearly these assessments are useful only for severely regressed, institutionalized individuals. Furthermore, since most of these instruments have been developed to provide local administrative agencies with information, such as summaries of patient characteristics and evaluations of treatment programs, their information is of limited relevance to formulating a rehabilitation treatment plan. Finally, there is little information about their reliability and validity when administered to the chronically mentally ill.

Figure 1. Process of psychiatric rehabilitation

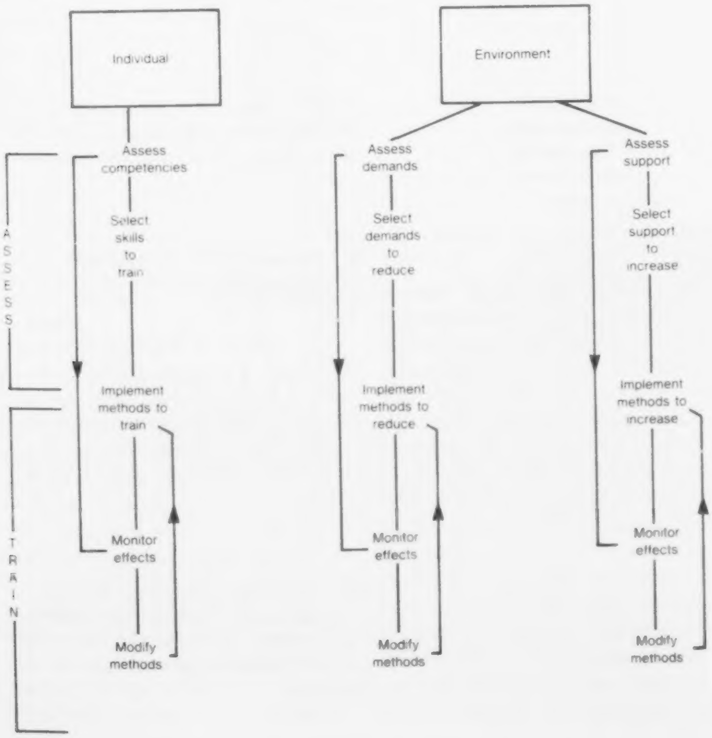
Individual
Environment
ASSESS
Assess competencies
Select skills to train
Implement methods to train
TRAIN
Monitor effects
Modify methods
Assess demands
Select demands to reduce
Implement methods to reduce
Monitor effects
Modify methods
Assess support
Select support to increase
Implement methods to increase
Monitor effects
Modify methods

Table 1 summarizes the major characteristics of the instruments to be reviewed.

Katz Adjustment Scale (KAS)

Description. The KAS (Katz and Lyerly 1963) was designed to "describe and classify patients in accordance with their behavior before entrance to the hospital, and in the community followup evaluation and comparison of psychiatric treatments" (p. 503). Two versions of the KAS are available: the KAS-R, which consists of 205 items completed by a patient's significant other, and the KAS-S, which consists of 138 items completed by the

Table 1. Major characteristics of instruments measuring functional living skills of the chronically mentally ill

Instrument	Areas assessed	Time frame of assessment	Data collection method
Katz Adjustment Scale (KAS)—Katz & Lyerly (1963)	Symptoms, expected activities, recreation/leisure activities	Past "few" weeks	Form R; 205-item questionnaire completed by significant other Form S; 138-item questionnaire completed by patient
Personal Adjustment & Role Skills Scale (PARS)—Ellsworth et al. (1968)	Symptoms, substance abuse, general community functioning	Past month	5 versions of PARS, all questionnaires of 29 to 79 items completed by significant other
Social Behaviour & Adjustment Scale (SBAS)—Platt et al. (1980)	Symptoms, role performance, objective burden, household distress	Past month	Semistructured interview conducted with significant other that covers 329 items
Psychiatric Status Schedule (PSS)—Spitzer et al. (1970)	Symptoms, instrumental role performance	Past week (symptoms) Past month (role performance)	Patient version: 321 items rated during structured interview; informant version: items rated during structured interview
Psychiatric Evaluation Form (PEF)—Endicott & Spitzer (1972*b*)	Symptoms, instrumental role performance	Past week (symptoms) Past month (role performance)	28 items completed by experienced clinician using all sources of information
Current & Past Psychopathology Scale (CAPPS)—Endicott & Spitzer (1972*a*)	PEF + past adjustment, symptoms, & personality	PEF + from age 12 to 1 month before evaluation	PEF + 13 items of current symptoms + 130 items of Psychiatric History Scale completed by experienced rater
Social Adjustment Scale (SAS)—Weissman et al. (1971)	General & interpersonal performance & satisfaction in 6 instrumental roles	Past 2 months (SAS, SAS-II) Past 2 weeks (SAS-SR)	SAS: 42 items rated during interview with patient; SAS-SR: 42-item questionnaire completed by patient; SAS-II: 52 items for chronic patients rated in interview

Table 1. Major characteristics of instruments measuring functional living skills of the chronically mentally ill—*Continued*

Instrument	Areas assessed	Time frame of assessment	Data collection method
Social Stress & Functioning Inventory (SSFIPD)—Serban (1978)	Performance & stress in 21 areas of community living	Not reported	174 performance items and 130 stress items rated during interview with patient
Denver Community Mental Health Questionnaire (DCMHQ)—Ciarlo & Riehman (1977)	Distress, isolation, drug & alcohol abuse, productivity, use agencies, satisfaction with service	Past 24 hours to past month	61 items rated during interview with patient
Community Adaptation Schedule (CAS)—Burnes & Roen (1967)	Behavior, affect, desire for change in 34 areas of functioning	Current	212-item questionnaire completed by patient
Rehabilitation Evaluation (REHAB)—Baker & Hall (1983)	"Deviant" behavior & "general" behaviors (speech, interaction, etc.)	Past week	7 deviant behavior items and 15 general behavior items completed by trained institutional staff

patient. Each version is divided into five forms.

Form 1 of the KAS–R obtains estimates from the significant other of the frequency with which 127 psychopathological (e.g., talks to self) and interpersonal (e.g., is dependable) behaviors have occurred within the "past few weeks." Scores on the items may be summed to obtain totals for the occurrence of minor symptoms, major symptoms, and interpersonal disturbance. However, the more typical scoring procedure involves summing across 86 items to obtain totals for 13 clusters: belligerence, verbal expansiveness, negativism, helplessness, suspiciousness, anxiety, withdrawal-retardation, general psychopathology, nervousness, confusion, bizarreness, hyperactivity, and stability (Hogarty and Katz 1971). Scores on these 13 clusters may be combined to obtain totals for three factors: social obstreperousness, acute psychoticism, and withdrawal-depression. The cluster scores may also be used to assign patients to one of six symptom patterns: agitated-belligerent-suspicious, withdrawn-periodically agitated, acute panic state, withdrawn-helpless-suspicious, agitated-helpless, and agitated-expansive-bizarre-suspicious (Katz 1966).

Forms 2 and 3 of the KAS–R obtain information from the significant other about the actual and desired frequency of the patient's performance of 16 "socially expected" activities such as attending church and visiting friends. Total scores are obtained by summing the actual and desired frequencies; the difference between them provides an index of the significant other's dissatisfaction with the patient's current performance. Forms 4 and 5 are much the same as forms 2 and 3, except that they survey the actual and desired frequency of performance of 23 leisure and self-improvement activities such as shopping and working around the house.

Form 1 of the KAS–S, which is a modified version of the Hopkins Symptom Distress Checklist, obtains information from the patient about the discomfort experienced for each of 55 symptoms. Forms 2, 3, 4, and 5 are the same as the corresponding KAS–R forms, except that the items are modified for reporting by the patient rather than by the significant other.

Psychometric Characteristics. The reliability and validity of the KAS, particularly form 1 of KAS–R, have been extensively investigated. Internal consistency reliabilities (coefficient α) of 12 of the 13 clusters of form 1 of the KAS–R were reported by Katz and Lyerly (1963) to range from .41 to .81 (the stability cluster was a later addition). Crook, Hogarty, and Ulrich (1980) reported a high degree of agreement between mothers and fathers of recently hospitalized patients for the more behaviorally based clusters (e.g., $r = .84$ for belligerence) and forms (e.g., $r = .85$ for form 2), with less agreement for the more inferential clusters (e.g., $r = .33$ for nervousness) and forms ($r = .08$ for form 3).

Hogarty and Katz (1971) and Crook, Hogarty, and Ulrich (1980) noted results that indicated that the ratings may be biased by the relationship of the significant other to the patient. Hogarty and Katz (1971) found that the ratings made by mothers of adolescents were significantly less pathological than ratings made by siblings, while Crook, Hogarty, and Ulrich (1980) reported that the ratings made by cottage mothers of delinquent adolescents were more pathological than ratings made by the cottage fathers. However, whether these differences reflect rater bias or actual differences in behavior directed to the various raters is impossible to determine.

The KAS has been found to discriminate among various groups of individuals including patients and community residents (Hogarty and Katz 1971), hospitalized and day-treatment patients (Hogarty et al. 1968), different types of depressed patients (Paykel 1972), and drivers involved in fatal single-car accidents compared to the general population (Schmidt et al. 1972). The KAS has been used in cross-cultural research (Katz, Gudeman, and Sanborn 1969; Katz 1981), and in epidemiological studies of the incidence of psychiatric disorder in the general population (Hogarty, Katz, and Lowery 1967).

The KAS has been used extensively to compare the effectiveness of various treatments, and it has been recommended by Waskow and Parloff (1975) for inclusion in the National Institute of Mental Health (NIMH) core battery of psychotherapy outcome measures. Not only have the six symptom patterns been associated with differential responsiveness to antipsychotic medication (Katz 1966; Katz, Lowery, and Cole 1967), but scores on the R forms have demonstrated the expected outcomes of day and hospital treatment of various diagnostic groups, with and without medication (Hogarty, Guy, and Gross 1969; Michaux et al. 1969; Caffey, Galbrecht, and Klett 1971; Hogarty, Goldberg, and Schooler 1974; Goldberg et al. 1977; Hargreaves et al. 1977).

Advantages and Disadvantages. Perhaps the major advantage of the KAS–R is that it has proved its value in a far broader range of treatment and diagnostic studies than any similar instrument. Additionally, it is easy to administer and score, and requires only 25–45 minutes to complete. Its coverage of psychopathology is particularly thorough, and its indices of significant others' dissatisfaction with current performance of socially expected and leisure activities are unusual and potentially useful.

The major disadvantage of both the R and S versions is that neither provides the detailed information necessary to construct a rehabilitation treatment plan. Both versions highlight broad areas of functioning (e.g., leisure activities) in which performance may be inadequate and rehabilitation necessary. However, neither pinpoints the causes of this inadequate performance (e.g., lack of interest, lack of skill, lack of knowledge, or inadequate resources) that need to be identified to design an effective rehabilitation plan. Furthermore, the psychometric adequacy of the S version has not been investigated, and there is no systematic method of detecting rater bias on either version. The range of socially expected and leisure activities is appropriate primarily for chonically ill patients, and the coverage of major life roles such as student and homemaker is extremely limited. Thus, the KAS is primarily useful either as a screening instrument, a broad measure for program evaluation, or an outcome measure for comparing the effects of various treatments.

Personal Adjustment and Role Skills Scale (PARS)

Description. Since its initial description (Ellsworth et al. 1968), five versions of the PARS have been published. Each has been a relatively brief questionnaire completed by a significant other about the patient's functioning within the previous month. The PARS-I consisted of 39 items measuring behaviors specifically relevant to the outcome of treatment with male veteran patients diagnosed as schizophrenic. The frequency of occurrence of each behavior was rated on a four-point scale, and the item scores were summed to obtain totals on six factor-analytically derived scales: dependent-confusion, agitation-depression, unacceptable behavior, friendship skills, participation in organizations, and employment.

Fifty items were added to the PARS-I to increase the range of male veteran patients for whom the scale would be applicable. Ten of the added items were eliminated either because they had low test-retest reliability, or because they did not discriminate pretreatment from posttreatment adjustment. A factor analysis of the remaining 79 items of the PARS-II yielded seven scales: interpersonal involvement, confusion, anxious-depression, agitation, alcohol abuse, outside social involvement, and employment.

To widen the applicability of the PARS to veteran and nonveteran patients of both sexes, two versions of the PARS-III were constructed: a 57-item version for males (pared from 115 items), and a 57-item version for females (pared from 120 items). Items in both versions measured parenting behaviors, and the response scale was expanded to five points. Factor analyses of the two versions yielded seven scales for males identical to those of the PARS-II and seven scales for females (interpersonal involvement, confusion, agitation, alcohol and drug abuse, household management, outside social activities, and employment, if applicable).

The PARS-IV was then constructed specifically to measure treatment outcomes for male veteran patients participating in a comprehensive study comparing the effects of hospital and community-based treatment. Twenty-five items were selected from the PARS-III, four items were added to measure performance of household chores, and the response scale was contracted to its original four points.

Finally, the PARS-V was constructed based on results of the PARS-III. Items were eliminated that either had low reliability (stability or internal consistency), poor loadings on the seven factors of the male and female versions of the PARS-III, or did not discriminate between pretreatment and posttreatment adjustment or among various diagnostic groups. Factor analyses of the resulting 39 items of the male version and the 37 items of the female version produced scales identical to those of the PARS-III, and norms were developed for interpreting pre-post changes as well as total scores on each scale.

Psychometric Characteristics. Including or excluding items based on their reliability and validity has ensured that all forms of the PARS have excellent psychometric characteristics. For example, the internal consistency reliabilities (coefficient a) of the scales of the PARS-V range from .77 to .94 for females and .66 to .94 for males, while stability coefficients range from .72 to .97 for females and .80 to .92 for males. Ellsworth (1975) found substantial agreement between PARS-III ratings of patients and significant others, particularly on the employment and substance abuse scales. Agreement was considerably lower for the symptom scales measured before treatment, although agreement was quite high for posttreatment symptom scales. Fontana and Dowds (1975) reported similar results for the PARS-II.

Like the KAS, the PARS has been used in a number of studies comparing the effectiveness of various treatments, and it has also been recommended by Waskow and Parloff (1975) for inclusion in the NIMH core battery of psychotherapy outcome measures. For example, Ellsworth et al. (1979) found that certain environmental and "setting" variables discriminated between hospital wards whose treatment outcomes were more or less effective as measured by the PARS-IV. Ellsworth (1978) reported that the PARS-V reflected the differential effectiveness of hospital versus community treatment, with hospital treatment being more effective for female patients than community treatment even though the hospitalized patients were worse at pretreatment. Ellsworth (1970*a*, 1973) demonstrated that feedback to hospital wards about the relationship between the posthospital outcomes of their patients and ward characteristics such as staff time spent with patients resulted in changes to the wards' structure and programs. However, the correlation between 1-month PARS-III posthospital adjustment and rehospitalization within 6 months was minimal (Ellsworth 1975), and the factors of the PARS-I did not discriminate patients who were rehospitalized after 6 months from those who were not (Arthur, Ellsworth, and Kroeker 1968).

Advantages and Disadvantages. Much like the KAS, the several versions of the PARS have proven their value in a wide variety of studies. Their psychometric characteristics are excellent; they are quite easy to administer and score; and they are well accepted by respondents. Ellsworth (1975) has reported that an initial mailing of the PARS followed by a second mailing and a telephone reminder resulted in a return rate of 92 percent.

However, their brevity limits the amount of information they provide. Their coverage of psychopathology is less than that of the KAS, and they do not provide measures of significant others' expectations. Furthermore, because their coverage of areas of community functioning is general, detailed information sufficient to formulate a rehabilitation treatment plan cannot be obtained. Thus, like the KAS, the several versions of the PARS are useful either as screening instruments, measures of program evaluation, or dependent variables comparing broadly defined outcomes of different treatments.

Social Behaviour and Adjustment Scale (SBAS)

Description. The SBAS (Platt et al. 1980) was designed to provide a comprehensive assessment of both a patient's performance of major life roles and the effects of that performance on the members of the household in which the patient resides. The information is obtained during a 60- to 90-minute interview conducted with a significant other about the patient's behavior during the previous month. The interview covers 329 questions divided into six sections.

The 22 questions of section 1 obtain information about demographic characteristics of the patient, the household, and the significant other. The 66 items of section 2 obtain information about the severity, time of onset, and distress experienced by the significant other of 22 symptoms such as misery, irritability, and self-neglect. The 48 items of section 3 obtain information about the patient's current performance, changes in that performance, time of onset of those changes, and distress experienced by the significant other for 12 socially expected activities such as child care, interpersonal relationships, and work-study behaviors. The 104 items of section 4 obtain information about the current conditions of the household such as the emotional well-being of its members, changes in those conditions, time of onset of these changes, extent to which they were related to the patient's illness, and degree of distress experienced by the significant other. The 66 items of section 5 obtain information about the frequency of occurrence of 11 stressful life events, the household member to whom they occurred, and the extent to which these events were related to the patient's illness. The 23 items of section 6 obtain information about the assistance received by household members from relatives, friends, and social agencies, as well as the size and type of the family's housing.

The scores on the six sections are combined to yield measures of the patient's disturbed behavior, performance of socially expected behaviors, and the objective burden represented by the patient. In addition, measures are obtained about the distress experienced by the household as a result of the disturbed behavior, changes in performance of socially expected activities, and the objective burden.

Psychometric Characteristics. Because of its relatively recent development, there is far less information about the psychometric characteristics of the SBAS than of the KAS or the PARS. Platt et al. (1980) reported that the interrater reliability (intraclass correlation coefficient) of the initial version of the SBAS ranged from .92 to .99 for the six summary scales. Interrater reliabilities (weighted kappa) of 83 percent of the individual items exceeded .70, although only 53 percent of the items measuring disturbed behavior and 66 percent of the items measuring distress caused by performance of socially expected activities exceeded .70. Platt et al. (1980) indicated that the current version of the SBAS has been modified to improve the reliabilities of these items.

Hirsch et al. (1979) found that the initial version of the SBAS reflected improvement, particularly in disturbed behavior, resulting from either long- or short-stay hospitalization. The improvement was corroborated by changes in the Present State Examination, and was maintained during a 3-month followup period. No differences were found in initial and followup improvement for long- versus short-stay patients.

Advantages and Disadvantages. Not only is the SBAS extremely thorough in its coverage of psychopathology and performance of socially expected behaviors, but its measurement of the objective and subjective burden caused by the illness is unique. Furthermore, it provides data about the support offered to and the demands imposed upon the patient by the immediate household environment. Its psychometric characteristics appear to be acceptable, and the interview format allows a wealth of information to be collected that might be missed with a paper-and-pencil assessment.

However, an interview is a costly method of obtaining information since it requires trained personnel whose accuracy must be continually monitored. Indeed, extensive training is needed to achieve reliable administration and scoring of the SBAS. Furthermore, the significant other must be very knowledgeable about the patient's functioning, and all of the information may not be relevant to designing a rehabilitation treatment plan. Certain areas of inadequate social and instrumental performance may be pinpointed, but additional assessments are likely to be necessary to determine the specific causes of the inadequacies.

Psychiatric Status Schedule (PSS), Psychiatric Evaluation Form (PEF), and Current and Past Psychopathology Scale (CAPPS)

Description. The PSS (Spitzer et al. 1970), the PEF (Endicott and Spitzer 1972*b*), and the CAPPS (Endicott and Spitzer 1972*a*) were designed to assess a common set of variables using different techniques. The PSS consists of 321 items, administered to a patient during a 30- to 50-minute structured interview, which survey psychopathology and performance of major instrumental roles. The psychopathology items are focused on behaviors and events occurring in the week before the interview; the instrumental role items are focused on behaviors and events occurring during the previous month. The items are scored true or false, and are worded with sufficient specificity to require only minimal judgment to rate each. A version of the PSS is available for administration to significant others, although it is used far less frequently than the patient form.

The PSS psychopathology items are variations of those of the Mental Status Examination (Spitzer et al. 1964), and are combined to yield scores on 15 first order factors and 4 second order factors. The first order factors are depression-anxiety, social isolation, suicide/self-mutilation, somatic concern, speech disorientation, inappropriate affect-appearance-behavior, agitation-excitement, interview belligerence-negativism, disorientation-memory impairment, retardation-lack of emotion, antisocial impulses or acts, overt anger, grandiosity, suspicion-persecution-hallucinations, and denial of illness. Three additional factors can be scored, although they lack statistical verification: daily routine-leisure impairment, alcohol abuse, and drug abuse. The four second order factors are subjective distress, behavioral disturbance, impulse-control disturbance, and reality-testing disturbance.

The PSS instrumental role items yield scores, as appropriate, about the adequacy of performance as a wage earner, housekeeper, student or trainee, spouse, and parent. These scores can be combined to obtain an overall instrumental role score.

The PEF consists of 28 items that measure psychopathology (20 items), instrumental role performance (5 items), and, for new admissions, duration of the current episode, presence/absence of precipitating stress, and the reason for admission. The PEF is suitable for experienced clinicians who rate each item on a six-point scale of severity using all sources of information (patient, staff, significant others, and case notes). Nineteen of the 20 psychopathology items are much the same as the first order factors of the PSS, including the focus on behaviors and events during the week before completion of the PEF; the 20th item measures the overall severity of the illness. The 19 items can be combined to obtain scores on six scales derived from factor analysis: disorganization, subjective distress, antisocial behavior, withdrawal, alcohol abuse, and grandiosity-externalization. The five role performance items can be combined to obtain an overall role performance score.

The CAPPS is a superset of the PEF designed to measure not only current psychopathology and instrumental role performance, but also social adjustment, psychopathology, and personality characteristics from age 12 to 1 month before completion of the CAPPS. The current section of the CAPPS includes the 28 items of the PEF plus 13 items that provide more detailed coverage of psychopathology such as phobia, guilt, and conversion reaction. These 41 items are combined to yield current severity scores on eight scales derived from factor analysis: reality testing-social disturbance, depression-anxiety, impulse control, somatic concern, disorganization, obsessive-guilt-phobic, elation-grandiosity, and summary role performance.

The "past" section consists of the 130 items of the Psychiatric History Scale (Spitzer, Endicott, and Fleiss 1967), which are combined to yield scores on 18 factor analytically derived scales: depression-anxiety, impulse control, social-sexual relations, reality testing, dependence, somatic concern, obsessive-compulsive, anger-excitability, manic, sexual disturbance, memory-orientation, disorganization, organicity, neurotic childhood, phobia, retardation, stubborn, hysterical symptoms, and intellectual performance.

Psychometric Characteristics. The reliability and validity of all three tests have been extensively investigated. For example, Spitzer et al. (1970) reported that internal consistency reliabilities (coefficient α) of the PSS ranged from .80 to .89 for the four second order factors, .34 to

.93 for the 18 first order factors, and .65 to .80 for the six role scales. Interrater reliabilities (intraclass correlation coefficients) for the PSS ranged from .90 to .98 for the second order factors, .57 to .99 for the first order factors, and .66 to .98 for the role scales. Interrater reliabilities (intraclass correlation coefficients) for the CAPPS administered by experienced raters ranged from .68 to 1.0 for individual items, and .74 to 1.0 for the summary scales.

Spitzer et al. (1967) found that the first and second order factors of the PSS discriminated among inpatients, outpatients, and nonpatients, and reflected the changes in functioning resulting from 4 weeks of hospital treatment. The PSS has also reflected the symptomatic improvement resulting from participation in day treatment programs (Herz et al. 1971; Washburn et al. 1976). Correlations of the first and second order PSS factors with other measures of psychopathology such as the Minnesota Multiphasic Personality Inventory, the Brief Psychiatric Rating Scale, and the Beck Depression Inventory were as expected (Spitzer et al. 1970). Correlations of the role impairment scales with the other measures were not significant, however.

The PEF has also discriminated among inpatients, outpatients, and nonpatients, as well as reflecting the effects of treatment with complete and partial hospitalization. Additionally, the PEF has discriminated among patients with different illnesses such as depression, alcohol abuse, and paranoid schizophrenia. Correlations of the PEF scales with their counterparts on the PSS have generally been positive, although the additional sources of information included in the PEF ratings produce somewhat different scores than the PSS. Similarly, all but eight of the CAPPS scales discriminated nonpatients from inpatients and outpatients, with the latter two groups significantly different from one another on almost all scales. Correlations of the CAPPS scales with the MMPI were generally as expected.

Advantages and Disadvantages. The careful and thorough development of the PSS, PEF, and CAPPS has produced a trio of instruments that can be administered by a wide range of interviewers to a wide range of patients. The three have proven their usefulness in a number of studies, and the PSS has been recommended by Waskow and Parloff (1975) for inclusion in the NIMH core battery of psychotherapy outcome measures.

However, the use of an interviewer is more expensive than the administration of more objectively formatted tests, and the coverage of instrumental roles by all three measures is relatively limited. Information about the cause of inadequate role performance is scant, and the roles are suitable principally for less chronic patients. Thus, the three are suitable either as screening instruments, measures of the overall outcome of programs, or dependent variables comparing the effects of different treatments.

Social Adjustment Scale (SAS)

Description. The SAS (Weissman et al. 1971), a modification of the Structured and Scaled Interview to Assess Maladjustment (Gurland et al. 1972), was designed to assess depressed women's general performance, interpersonal performance, and satisfaction in six social instrumental roles: work, social and leisure activities, extended family relationships, relationships with spouse, parental relationships, and general family relationships. It consists of 42 items focused on behaviors and events occurring during the 2 months before the assessment. Each item is rated on a five-point scale of severity based on information obtained from the patient during a 45- to 90-minute semistructured interview. In addition, five items assess global performance in each role except general family relationships, while a final item measures performance across all roles.

Scores on the 42 items can be combined to yield an average performance per role, eliminating items that do not apply. However, the common scoring procedure involves combining scores on 33 of the items to obtain totals for six factor analytically derived scales: work performance, interpersonal friction, inhibited communications, submissive dependency, family attachment, and anxious rumination (Paykel et al. 1971). A self-report version has been developed (SAS-SR; Weissman and Bothwell 1976) that consists of the same 42 items altered to focus on behaviors and events of the previous 2 weeks rather than the previous 2 months, and with the five points of the response scale defined in quantitative terms.

Since the SAS was initially developed for use with depressed women, it is somewhat inappropriate for assessing the social adjustment of more chronically ill patients. Schooler, Hogarty, and Weissman (1979), and Glazer et al. (1980) developed the SAS-II specifically for use with ambulatory, chronic schizophrenic patients. The SAS-II consists of 52 items that measure interpersonal and instrumental performance in five areas: work (wage earner, homemaker, or student), relationships with household members, relationships with relatives not living in the immediate household, performance of leisure and recreational activities, and personal well-being. As is true of the SAS, information to rate each item is obtained during a semistructured interview conducted with the patient.

Psychometric Characteristics. The interrater reliability of the SAS was reported by Weissman et al. (1971) and by Paykel et al. (1971) to average .83 per item for 31 interviews (range of .33 to .97), with 86 percent of the items differing by no more than one scale point. Internal consistency reliabilities (coefficient α) of the SAS-SR scales averaged .737, with test-retest reliabilities ranging from .712 to .82 (Edwards et al. 1978).

The SAS not only discriminated depressed patients from nonpatients, but five of the six factors reflected improvement as the result of treatment with antidepressant medication and/or psychotherapy (Weissman et al. 1971). Furthermore, four of the six scales demonstrated greater changes for the women receiving psychotherapy than those receiving medication, although these differences were apparent only at 8 months posttreatment and not at 8 or 12 months posttreatment (Weissman et al. 1981). Adjustment, however, was not correlated with relapse rates (Klerman et al. 1974).

The SAS-SR has also reflected the effects of medication and/or psychotherapy with depressed women (Prusoff et al. 1980), and has been administered to several types of individuals including undergraduates, women attending consciousness-raising seminars, patients receiving day treatment services, and patients recovering from cardiac surgery (Weissman et al. 1978). Correlations between the SAS-SR scales and SAS interviewer ratings ranged from .40 to .76 for the six scales, with a median of .54. Correlations of the SAS-II scales with ratings of adjustment made by significant others ranged from .27 to .81 (Glazer et al. 1980).

Advantages and Disadvantages. Given their common focus on social adjustment, the SAS, SAS-SR, and the SAS-II provide information that is particularly relevant for designing a rehabilitation treatment program. The SAS and SAS-SR are appropriate for higher functioning patients, while the SAS-II is appropriate for more chronically ill patients. The psychometric characteristics of all three measures appear to be acceptable, and the SAS and SAS-SR have reflected changes due to treatment.

Perhaps the major disadvantage of the SAS and the SAS-II is the cost of obtaining information using a trained interviewer. Furthermore, the information from all three measures would need to be supplemented by more detailed assessments to specify the skills to be trained in the rehabilitation treatment program.

Social Stress and Functioning Inventory for Psychotic Disorders (SSFIPD)

Description. The SSFIPD (Serban 1978) was designed to assess patients' objective level of functioning and subjectively experienced stress in each of 21 areas of adjustment. The 21 areas are grouped into four major "sectors": instrumental performance (education, work, housekeeping, dependence on welfare, management of finances, general living circumstances), family interactions (parents, relatives, marriage, children), social interaction (dating, sex, friends, neighbors, others, leisure activities, religion), and social maladjustment (drinking, addictive drugs, psychedelic drugs, antisocial acts).

Objective level of functioning is measured by 174 items that assess five variables: actual level of functioning, comparison of functioning with that of significant others, attempts at self-improvement, impairment due to uncontrolled events, and personal appraisal of one's own functioning. Subjectively experienced stress is measured by 130 items that obtain information about the distress experienced by the patient as the result of both his/her current level of functioning and the problems encountered in each area of adjustment. Almost all items are rated on three-point scales of severity or impairment based on responses provided by the patient during a 45- to 90-minute structured interview. Scores on the items are combined to yield average functioning and stress scores per area. A form is available for use with significant others.

Psychometric Characteristics. Serban (1978) reported that the interrater reliability of the SSFIPD ranged from 85 to 91 percent agreement for 25 interviews: unfortunately, he did not indicate if these results were applicable per item or per area. Test-retest reliability determined over a 6-month interval with different interviewers ranged from .43 to .77 for seven areas, with four less than .60 (results were not reported for 14 of the areas). Ratings of functioning made on the basis of information provided by significant others were significantly correlated with ratings based on information provided by patients for 18 areas for chronically ill patients and 13 areas for acutely ill patients. Ratings of stress based on significant others' information were significantly correlated with ratings based on patients' information for only six areas (Serban 1980). This lack of agreement on ratings of stress is somewhat expected since stress is measured as a subjectively experienced phenomenon with fewer objective referents than the measurement of functioning.

Functioning in 13 of the areas significantly discriminated nonpatients from chronic patients, while stress in six areas discriminated between the groups (Serban 1975). Rehospitalization for chronic patients was associated with antisocial behavior and poor interpersonal relationships with neighbors and with the opposite sex. In contrast, rehospitalization for acute patients was associated with poor interpersonal relationships with parents and friends. Levels of stress were not associated with rehospitalization for either group.

Advantages and Disadvantages. The SSFIPD thoroughly surveys not only multiple areas of functioning, but also multiple behaviors that constitute adequate functioning. The 21 areas are relevant to designing an individual rehabilitation plan, and the focus on subjectively experienced stress is unique. However, averaging the items to obtain one functioning score per area obscures the advantage of measuring functioning in terms of the five variables, and the psychometric adequacy of the entire inventory is not well documented.

Denver Community Mental Health Questionnaire (DCMHQ)

Description. The DCMHQ was designed at the Denver Northwest Community Mental Health Center as a "tool for assessing any and all mental health center clients capable of communication" (Ciarlo and Riehman 1977). It consists of 61 items grouped into 12 scales: psychological distress, interpersonal isolation from family, interpersonal isolation from friends, productivity at work, productivity at home, dependency on public agencies, alcohol abuse, soft drug use, hard drug use, client satisfaction with services, interpersonal aggression with friends, and legal difficulties. All items are rated on four-point response scales with each point defined differently from item to item. Information to rate each is obtained during a 30- to 60-minute semistructured interview conducted with the patient.

Psychometric Characteristics. Ciarlo and Riehman (1977) reported that the internal consistency reliabilities (coefficient α) of the 12 scales ranged from .52 to .96. Interrater reliabilities ranged from .85 to 1.0 for 10 of the 12 scales (two were not used) based on 18 interviews conducted with two raters present during each interview. Ratings made by significant others correlated from .59 to .87 with ratings based on information provided by patients for nine scales (three were not used). All but one scale (interpersonal aggression with friends) discriminated patients from a random sample of 212 residents living in the Denver metropolitan area. Norms were constructed based on the responses of the 212 residents, and raw scores on the scales were converted to means of 50 (average of the community residents) and standard deviations of 5. Patients' "profiles" of functioning could then be compared to the functioning of nonpatient residents.

Advantages and Disadvantages. The DCMHQ clearly reflects its development as an instrument tailored to the services and clientele of a community mental health center. Its coverage of antisocial behaviors such as alcohol abuse is quite thorough; indeed, the majority of the items obtain information about substance abuse (32 items) and contacts with legal and public assistance agencies (9 items). Measurement of these behaviors is important since antisocial activity may result in patients' unsuccessful placement in the community despite the adequacy of their functioning in areas such as work and interpersonal relationships. Additionally, the DCMHQ is unique in its inclusion of a measure of satisfaction with services.

However, the coverage of antisocial behaviors and satisfaction with services has been achieved at the expense of the coverage of instrumental role skills. The results of the DCMHQ will need supplementation with additional information if the goal of the assessment is to design a rehabilitation treatment plan.

Community Adaptation Schedule (CAS)

Description. The CAS (Burnes and Roen 1967) was designed to assess patients' behavior, affect, and desire for changes in 34 "subsections" (e.g., employment, work potential, spouse, neighbors, recreation, transportation), grouped into six "communities" (work, family, social, larger, commercial, and professional). The CAS is a self-report questionnaire of 212 items, each with a six-point response scale defined differently from item to item. Three types of items are presented: behavior (e.g., "How many different organizations and clubs do you belong to?"), affect (e.g., "How do you feel about participating in groups?") and desire for change ("Would you join more organizations or clubs if you were invited to?"). Scores are summed to obtain a total per subsection, per community, and for all communities. Totals are also obtained for all behavior, affect, and desired change items.

Psychometric Characteristics. Burnes and Roen (1967) reported that the test-retest reliabilities of the subsections averaged .75 for nonpatients and .73 for inpatients using retest intervals of 1 week and 1 month. Internal consistency reliabilities were not reported.

The majority of community and subsection scores of the initial version of the CAS discriminated recently discharged psychiatric patients from community residents (Roen et al. 1966). Scores on all six communities, 18 of the 34 subsections, and all three types of questions of the revised CAS discriminated among 79 inpatients, 58 outpatients, and 54 mental health professionals (Burnes and Roen 1967). Cook, Looney, and Pine (1973), however, found no differences on the CAS between 23 inpatients and 23 outpatients matched for age, sex, race, and marital status, although scores on the CAS were correlated with scores on the Adjective Checklist. The latter finding was interpreted by Cook, Looney, and Pine (1973) as indicating that higher CAS scores were associated with better personal adjustment. These correlations corroborated the report by Cook and Josephs (1970) of significant correlations between CAS scores and scores on the California Psychological Inventory for 57 undergraduates. Harris and Brown (1974), however, found no significant correlations between CAS scores and scores on the Personal Orientation Inventory for 40 volunteers for a mental health paraprofessional training program. CAS scores were correlated with the Marlowe-Crowne Social Desirability Scale, replicating a significant correlation between the CAS and the Good Impressions Scale of the California Psychological Inventory reported by Cook and Josephs (1970).

Advantages and Disadvantages. The CAS is not only comprehensive in its coverage of community functioning, but its division of items into behavior, affect, and desired changes is unique. This division aids design of the rehabilitation treatment program to meet the needs and goals of the patient.

However, the psychometric adequacy of the CAS is not as well documented as that of other scales, and the significant correlations with the Marlowe-Crowne and the Good Impressions scales suggest that the CAS may be distorted by the respondent's set to answer in a socially desirable manner.

Rehabilitation Evaluation of Baker and Hall (REHAB)

Description. REHAB (Baker and Hall 1983) was designed to assess institutionalized patients' "deviant" and "general" behaviors. It consists of 22 items (plus a 23rd that assesses overall functioning) that are completed by trained staff members who base their ratings on behaviors and events that occurred during the week before the evaluation. The seven deviant behavior items measure the frequency of occurrence of "difficult or embarrassing" behaviors including incontinence, physical abuse, verbal abuse, self-mutilation, sexual offensiveness, unauthorized absence, and talking or laughing to onself. Three-point response scales are used to rate each item, and scores are combined to yield a total deviant behavior score.

The 15 general behavior items measure speech characteristics (four items), social interaction (three items), engagement in spare time activities, activity level, table manners, personal hygiene, grooming, care of personal possessions, caring for own needs, managing money, and using public facilities. Each item is rated by making a mark along a 4.25-inch line defined at the left by the worst possible performance, at the right by performance that would be considered "appropriate" for independent living in the community, and in the middle by moderately inappropriate performance. Scores on the items are combined to yield totals for five scales derived from factor analysis: social activity, speech disturbance, speech skills, self-care skills, and community skills, plus a total for all general behavior items which includes the rating given to the overall functioning item.

Psychometric Characteristics. The reliability and validity of REHAB have been thoroughly investigated since it was first developed as part of a 7-year study of the effectiveness of behaviorally based treatment methods with chronic psychiatric patients (Baker et al. 1977; Hall, Baker, and Hutchinson 1977). Acceptable content validity was ensured by including behaviors that occurred frequently in institutions (Aumack 1969), were correlated with outcomes such as discharge (Hall, Baker, and Hutchinson 1977), and had been nominated by psychiatric nurses (Hall 1977) and significant others (Creer and Wing 1974) as important in patients' care. Reliable administration and scoring were ensured by developing both an extensive training procedure and a raters' guide that thoroughly explains each item's stem and response scale.

This careful development has resulted in good reliability and validity. Baker and Hall (1983) reported that the interrater reliability of each item ranged from .61 to .92 based on independent ratings of 47 patients; additional studies have confirmed these results (e.g., Iljon-Foreman and Baker, in press). The factor analytically derived structure of the five general behavior scales has been replicated across several patient samples residing in a number of institutions, including patients attending a day hospital, floridly symptomatic inpatients, and long-stay inpatients.

The results of several discriminant function analyses indicated that scores on the five general behavior scales, as well as the total general behavior score, differentiated day hospital patients ($n = 189$), long-stay inpatients ($n = 204$), and inpatients selected for a predischarge training program ($n = 57$). Correct classifications among all pairings of the different types of patients ranged from 67 to 77 percent. REHAB scores were significantly correlated with direct observations of similar behaviors made by an independent rater, and scores on the initial version of REHAB were significantly correlated with ratings of socially embarrassing behavior and social withdrawal made on the Wing Ward Behavior Scale. However, as expected, REHAB ratings did not correlate with interview-based ratings of psychopathology or with the results of standardized psychological tests.

REHAB general behavior scores reflected improvement as the result of patients' participation in programs designed to improve their community skills. Total deviant behavior scores reflected deterioration as the result of a double-blind withdrawal of medication (Mathur and Hall 1981); interestingly, reductions in total general behavior scores predicted patients who relapsed during the withdrawal phase; relapse was determined by the nursing staff independently of REHAB ratings.

Advantages and Disadvantages. The careful development of REHAB has resulted in a low-cost measure that is reliable and useful for assessing general and deviant behaviors of chronically ill, institutionalized patients. Not only does REHAB include administration and scoring aids such as a comprehensive training procedure, raters' guide, and users' manual, but the five scale and two total scores are readily interpreted using norms based on more than 800 patients. The simplicity of the administration and scoring procedures makes it easy to conduct repeated testings to monitor the individual and program-wide effects of treatment.

However, the information provided by REHAB is not extensive enough to form the basis for a thorough rehabilitation treatment plan. Rather, the results are suggestive of possible areas for treatment, and the information must be supplemented to pinpoint the exact behaviors to be targeted in the treatment program.

Additional Instruments

There are several additional instruments that may be useful given certain caveats. These instruments either have limited distribution and inadequately documented psychometric properties, or they are readily available but provide information of limited relevance for designing a psychiatric rehabilitation program. These instruments are as follows.

Social Maladjustment Schedule (SMS). The SMS (Claire and Cairns 1978) was designed to measure objective conditions, patients' instrumental performance, and patients' satisfaction in six areas of community functioning: housing, occupation and social role, economic situation, leisure and social activities, family and domestic relationships, and marital relationships. Information to complete the SMS is obtained during a 45-minute structured interview conducted with either patients or significant others. Objective conditions are assessed with questions such as "Do you see much of your neighbors?"; answers are rated on four-point response scales defined differently from item to item (for the example item, unrestricted to severe restrictions). Instrumental performance is assessed with questions such as "How well do you get on with your neighbors?"; answers are rated on a four-point response scale that is the same for each item (no difficulty to severe difficulty). Satisfaction is assesed with questions such as "Do you feel happy about your relationship with your neighbors?"; answers are rated on a four-point response scale that is the same for each item (satisfied to severe dissatisfaction). Scores on the items are combined to yield total scores for objective conditions, instrumental performance, satisfaction, and overall maladjustment.

Interrater reliability based on interviews of 48 patients referred by general practitioners ranged from 63.6 to 100 percent agreement with four trained interviewers. Weighted kappa for 17 of the 48 items for which there were sufficient ratings to calculate the statistic ranged from .62 to .94. The overall maladjustment score discriminated psychiatrically impaired patients from those not impaired, and different patterns of objective conditions, instrumental performance, and satisfaction discriminated chronic neurotic patients from women suffering from premenstrual symptoms.

Self-Assessment Guide (SAG). The SAG (Willer and Biggen 1976) was designed to assess functioning in seven areas: physical health, general affect, interpersonal skills, personal relations, use of leisure time, control of aggression, and employment. It consists of 55 items administered to patients as a self-report questionnaire. The results of the assessment are specifically formatted to assist clinicians in documenting individual treatment plans. Pretreatment and posttreatment scores are entered into the Goal-Oriented Record (Willer and Howard 1974; Willer 1977) whose format is essentially the same as the Problem-Oriented Medical Record.

Unfortunately, Willer and Biggen (1976) do not report data about the reliability of the SAG. They do note that, unlike more commonly used measures, SAG scores discriminated patients who were rehospitalized within 6 months posttreatment from those who were not.

Community Living Assessment Scale (CLAS). The CLAS (Willer and Gustaferro 1985) was designed to measure the community functioning of patients living in residential care facilities. It consists of 68 items that assess nine areas of functioning: personal management (hygiene and grooming), nutritional management, money management, home management, medical management, time management, use of community resources, problem-solving skills, and emergency management. Each item describes a task such as "vacuums the carpet once per week with no urging"; each is rated by facility staff using a five-point response scale defined by the amount of supervision required to complete the task (does not complete task, completes task with total supervision, completes task with frequent supervision, completes task with minimal monitoring, and completes task independently). Scores for the items are combined to yield total for each area. Items that describe tasks that cannot be performed because of institutional constraints (e.g., cannot prepare food) are scored as "0" and contribute to the area score. Totals can be converted to T scores which were calculated based on a sample of 252 residents (135 males and 117 females) receiving services at a residential care facility (Transitional Services of Buffalo).

The initial version of the CLAS consisted of 368 items; 300 were eliminated due to either poor interrater reliability or low correlations with other items in the same area. Interrater reliabilities were calculated based on independent ratings of 157 patients made by two staff members. The product-moment correlations between the two raters for the remaining 68 items ranged from .76 to .84 per area. However, no additional studies were conducted to determine interrater reliabilities with raters other than the original two. The split-half reliability coefficients of the nine areas determined on the basis of ratings of 252 patients ranged from .88 to .97; the test-retest reliabilities of the nine areas based on ratings of a subsample of 95 patients repeated across a 3-month interval ranged from .68 to .79.

The nine area scores significantly discriminated a sample of 58 friends and staff of Transitional Services from a sample of 58 patients matched on age and sex. The area scores did not, however, discriminate among 97 patients who, after 2 years of care at Transitional Services, were either placed in independent living, returned to their families, discharged to nursing homes, or remained at Transitional Services. Irrespective of the patients' placement, the majority of the area scores were significantly correlated with ratings of 2-year outcome on variables such as getting along with others, level of independence, and overall rating of success. Unfortunately, the raters and the rating method were not described.

Community Adjustment Profile System (CAPS). The CAPS (Evanson et al. 1974) was designed not only to measure community functioning relevant to a broad range of patients, but also to be an integral component of a computer-based system designed to monitor the ongoing effectiveness of services. It consists of 98 items administered in a questionnaire format to significant others. Scores on the 98 items are combined to provide totals on scales of hostility, alcohol abuse, depression, work problems, assault, peculiarity, previous treatment, drug use, suicidal behavior, police contact, early deprivation, early maladjustment, and family pathology. Internal consistency reliabilities (coefficient α) of the scales ranged from .70 to .92, and norms have been calculated based on a sample of 748 patients.

Nurses Observation Scale for Inpatient Evaluation (NOSIE-30). The NOSIE-30 (Honigfeld, Roderic, and Klett 1966) was designed to measure three positive and three negative factors of inpatients' behavior: social competence, social interest, personal neatness, irritability, manifest psychosis, and retardation, plus total scores for positive, negative, and total assets. It consists of 30 well-specified items that are completed by staff using a five-point response scale that estimates the frequency of occurrence of each item. Interrater reliabilities (intraclass coefficients) have been reported to range from .57 to .83, and norms have been developed based on several very large samples of patients residing in a variety of institutions. The scale has been used in numerous studies comparing the effectiveness of many different types of medication and psychosocial interventions. It has, for example, reflected the effects of behavioral interventions such as token economies (Gripp and Magaro 1971; Schwartz and Bellack 1975; Nelson and Cone 1979), particularly for psychotic patients (Kowalski, Daley, and Gripp 1976).

Motility, Affect, Communication, Cooperation-II (MACC-II). The MACC-II (Ellsworth 1970*b*) was designed to measure four clusters of inpatients' behavior: mood, cooperation, communication, and social contact, plus a total adjustment score that is the sum of the four cluster scores (the original version of the scale assessed motility, affect, communication, and cooperation). It consists of 20 items that are completed by staff using a five-point response scale with the anchors defined differently from item to item. Reliabilities are generally high with interrater correlation coefficients ranging from .70 to .90, internal consistency coefficients from .83 to .93, and stability coefficients from .80 to .90. As expected, however, correlations between raters who have different experiences with patients are negligible (e.g., ratings made by inpatient staff and ratings made 4 weeks after discharge by community social workers). Norms were calculated based on a sample of more than 1,000 patients living in several types of institutions, and the MACC has been used in numerous studies comparing the effectiveness of several different types of interventions. Like the NOSIE-30, the MACC has reflected the effects of behavioral interventions such as token economies (Shean and Zeidberg 1971; Bennett and Maley 1973; Rostow and Smith 1975).

Life Skills Assessment (LSA). The LSA (Farkas 1980; Farkas, Rogers, and Thurer 1985) was developed to assess institutionalized patients' symptomatology and community living skills. It consists of 21 items judged by inpatient and community care staff as essential for successful hospital discharge and long-term adjustment to the demands of community living. The items are derived from the Progress Assessment Chart (Gunzberg 1968), a 100-item instrument originally designed to assess the skills of developmentally delayed individuals, and the Psychotic Inpatient Profile (Lorr and Vestre 1968). Each item is rated on a five-point scale ranging from "no performance of the skill" to "adequate and consistent performance" based on information obtained from direct observation of the patient or from interviews conducted with staff members. Each item is weighted to reflect its relative importance for discharge and community tenure; scores on each are combined to obtain a total skill score.

Farkas, Rogers, and Thurer (1985) reported that the interrater reliability (product-moment correlation coefficients) of the LSA ranged from .67 to .98 based on four administrations to the same set of 54 patients across a span of 4 years. Total scores significantly discriminated a subset of the 54 who were discharged from the hospital and remained successfully in the community during the 4 years ($n = 8$) from a subset who were never discharged ($n = 22$).

Independent Living Skills Survey (ILSS). The ILSS (Wallace, Kochanowicz, and Wallace 1985) was developed to provide a detailed assessment of the community living skills of chronically mentally ill patients residing with significant others or in residential care facilities such as family-care and board-and-care homes. Nine areas of skills are assessed: eating habits, grooming skills, domestic activities, food preparation skills, health maintenance skills, economic skills, use of public transportation, leisure activities, and job-seeking skills. The nine areas were selected based on an inspection of 15 measures of the functioning of chronically mentally ill patients and developmentally disabled patients (e.g., PARS, San Diego Skills Test, KAS, AAMD Adaptive Behavior Scale) and on information obtained during interviews conducted with five community social workers and five operators of residential care facilities.

An initial pool of 102 items was constructed, which was expanded by 10 items based on interviews conducted with additional social workers and residential care operators. The resulting 112 items, which describe specific behaviors that define each skill area, are formatted as a questionnaire to be completed by significant others or facility staff using a six-point response scale focused on behaviors and events of the previous month (never, sometimes, often, usually, always, and no opportunity to perform). An additional five-point response scale, which is designed to provide information to prioritize the areas to be trained in the rehabilitation treatment plan, measures the degree to which each behavior is a problem (never a problem, occasionally a problem, sometimes a problem, frequently a problem, and always a problem). (To provide interested readers with a possible means of pinpointing behaviors to be trained in the rehabilitation treatment plan, the items and the response scales are reproduced in the Appendix to this article. A self-report version of the scale is reproduced in Wallace et al. 1985.)

To determine the internal consistency, reliability, and concurrent validity of the ILSS, operators of 59 family-care homes completed the ILSS, the NOSIE-30, and the MACC-II for one patient whose current diagnosis was chronic schizophrenia in each facility. The diagnosis was confirmed from information obtained during an interview conducted with each patient using questions drawn from the Present State Examination and scored according to the criteria of the New Haven Schizophrenia Index. The interviews were tape-recorded and later rated by an independent rater; agreement on the diagnosis was 100 percent.

Coefficient α's of the nine areas ranged from .67 to .84, and split-half reliabilities ranged from .63 to .89. Three of the areas (eating habits, food preparation skills, and job-seeking skills) were lower in reliability than the other six areas because of restricted variance. Almost all of the patients had acceptable eating habits, very few were allowed to prepare their own meals, and almost no one was actively engaging in job-seeking behaviors.

Of the 36 correlations between the four positive assets scores of the NOSIE-30 (social competence, social interest, personal neatness, and total positive assets) and the nine skill areas of the ILSS, 20 were significant at the .01 level and four at the .05 level. The two measures shared from 10 to 37 percent of their variances in common. This was sufficient to provide evidence of the concurrent validity of the ILSS and to indicate that it is not simply a variation of the NOSIE-30. Additionally, the areas of the NOSIE-30 that measured more social assets, social interest and social competence, significantly correlated only with those ILSS areas that measured interactions with other people or interest in activities outside of the confines of the residential care facilities (health maintenance, economic, use of public transportation, leisure activities). As expected, scores on the negative factors of the NOSIE-30 did not correlate with scores on the ILSS areas except health maintenance.

Of the 36 correlations between scores on the four MACC-II scales and scores on the nine ILSS areas, six were significant at the .01 level and six at the .05 level. The communication scale of the MACC-II was significantly correlated with six areas of the ILSS, while the cooperation scale was significantly correlated with four areas.

Summary and Conclusions

Perhaps the one summary statement that applies equally well to all of the reviewed instruments is that no one of them is wholly adequate for assessing functional living skills. The many differences among them, which reflect the many different purposes for which they were constructed, preclude recommending one as the "best" tool for assessing functional living skills.

Instruments such as the PSS and the PARS provide global assessments of performance in major life roles such as spouse and worker, while instruments such as the ILSS and the CLAS provide specific assessments of molecular behaviors such as food preparation and personal hygiene skills. These different assessments reflect fundamental differences among the instruments in both the behaviors that each defines as "functional living skills" and the individuals for whom each is designed. Unfortunately, this heterogeneity makes it difficult to integrate information across instruments and to compare the effectiveness of various rehabilitation procedures.

Instruments such as the PSS and the CAS collect data from patients, instruments such as the SBAS and the KAS collect data from significant others, and instruments such as REHAB and the CLAS collect data from staff members. Each source is likely to provide somewhat different information which may be difficult to interpret as reflecting either rater bias or actual differences in behavior. Unfortunately, only a few instruments such as the PSS provide similar forms for use with different sources that assist in the comparison and integration of the information.

Instruments such as the PARS and the CAS collect data using objective questionnaires, instruments such as the SBAS and the SMS collect data using semistructured interviews, and instruments such as REHAB and the CLAS collect data using formal and informal observations. Although questionnaires are inexpensive, they collect only a limited amount of data. Although interviews are costly, they collect a wealth of data, and they involve the interviewee in the process of developing the rehabilitation treatment plan. This involvement may be particularly important and cathartic when the interviewee is a significant other.

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This heterogeneity does allow the assessor to select the instrument that best suits the purposes for which the assessment is conducted. If the purpose is to determine broad areas of living skills for which rehabilitation may be desirable, then one or more of the global instruments may be appropriate. An advantage of these instruments is that they are useful for evaluating entire treatment programs and describing the characteristics of patients who participate in them. Furthermore, they often include assessments of psychopathology and antisocial behaviors that, even at very low frequencies of occurrence, may impede successful adaptation to community life among patients who are skilled enough to prepare their own food, seek a job, care for their personal possessions, etc. If significant others are available, then a battery of instruments that use information from both patients and informants may be particularly appropriate.

If, however, the purpose of the assessment is to pinpoint behaviors that will be trained in the rehabilitation treatment plan, then one of the more specific instruments may be appropriate. Unfortunately, the psychometric adequacy of these instruments is not generally as well documented as that of the more global instruments, and the more specific the instruments, the more limited the range of patients to whom they can be successfully administered. Furthermore, assessment useful for developing and monitoring rehabilitation procedures should be guided by procedures that can be feasibly implemented. Indeed, the most appropriate assessment procedures for planning rehabilitation may involve administration of the very instruments that are part and parcel of the specific treatment procedures to be used (e.g., Wallace et al. 1985).

Perhaps most desirable would be a set of instruments that would be integrated by a common focus on the same treatment procedures but would systematically vary in their specificity. The more general instruments would assess major areas of functional living skills applicable to a wide range of patients using data collected from several different sources by means of several different methodologies such as questionnaires, direct observations, and "work samples." The information from these instruments would be examined to identify the major areas of living skills that would be the focus of a patient's rehabilitation treatment plan.

Each of these major areas would then be assessed using specific instruments appropriate for the patient's level of functioning. The resulting information would pinpoint the behaviors to be trained and be directly relevant to measuring the effects of the specific procedures of the rehabilitation treatment plan. Repeated administration of the specific instruments would be routinely conducted as treatment progressed; the effects would be apparent on the more general measures as major objectives of the rehabilitation treatment plan were achieved. In actuality, specific items and not entire instruments would be used as targeted behavioral problems or goals to be used in repeated measurement of patients' progress. One exception to this would be multidimensional instruments used in direct behavioral observation and time sampling of ongoing patients' behavior in living or working environments—such as the Time Sample Behavioral Checklist described in the article by Glynn and Mueser (this issue). Repeated observation, measurement, and recording of behaviors relevant to patients' functional status can be highly effective and sensitive ways to guide rehabilitation decisions.

References

Anthony, W.A., and Cohen, B.F. Functional assessment in psychiatric rehabilitation. In: Halpern, A., and Fuhrer, M., eds. *Functional Assessment in Rehabilitation.* Baltimore: Brookes, 1984. pp. 79-100.

Anthony, W.A.; Cohen, M.; and Nemec, P. Assessment in psychiatric rehabilitation. In: Bolton, B., ed. *Handbook of Measurement and Evaluation in Rehabilitation.* Baltimore: Brookes, 1986.

Anthony, W.A., and Farkas, M. A client-outcome planning model for assessing psychiatric rehabilitation interventions. *Schizophrenia Bulletin,* 8:13-38, 1982.

Anthony, W.A., and Nemec, P.P.B. Psychiatric rehabilitation. In: Bellack, A.S., ed. *Schizophrenia: Treatment, Management, and Rehabilitation.* New York: Grune & Stratton, 1984.

Arthur, G.; Ellsworth, R.B.; and Kroeker, D. Readmission of released mental patients: A research study. *Social Work,* 13:78-84, 1968.

Aumack, L. The Patient Activity Checklist: An instrument and an approach for measuring behavior. *Journal of Clinical Psychology,* 25:134-139, 1969.

Baker, R., and Hall, J.N. *Rehabilitation Evaluation of Hall and Baker (REHAB).* Aberdeen, Scotland: Vine Publishing Ltd, 1983.

Baker, R.; Hall, J.N.; Hutchinson, K.; and Bridge, G.W.K. Symptom changes in chronic schizophrenic patients on a token economy: A controlled experiment. *British Journal of Psychiatry,* 131:391-393, 1977.

Barrabee, P.; Barrabee, E.L.; and Finesinger, J.E. A normative social adjustment scale. *American Journal of Psychiatry*, 112:252–259, 1955.

Bennett, P.S., and Maley, R.F. Modification of interactive behaviors in chronic mental patients. *Journal of Applied Behavior Analysis*, 6:609–620, 1973.

Burnes, A.J., and Roen, S.R. Social roles and adaptation to the community. *Community Mental Health Journal*, 3:153–158, 1967.

Caffey, E.M.; Galbrecht, C.R.; and Klett, C.J. Brief hospitalization and aftercare in the treatment of schizophrenia. *Archives of General Psychiatry*, 24:81–86, 1971.

Ciarlo, J., and Riehman, J. The Denver Community Mental Health Questionnaire: Development of a multidimensional program evaluation. In: Coursey, R.D.; Specter, G.A.; Murrel, S.A.; and Hunt, B., eds. *Program Evaluation for Mental Health: Methods, Strategies, and Participants*. New York: Grune & Stratton, 1977.

Clare, A.W., and Cairns, V.E. Design, development, and use of a standardized interview to assess social maladjustment and dysfunction in community studies. *Psychological Medicine*, 8:589–604, 1978.

Cone, J.D. *The Pyramid Scales*. Available from Pro-Ed, 5341 Industrial Oaks Blvd., Austin, TX 78735, 1985.

Cook, P.E., and Josephs, P.O. The Community Adaptation Schedule and the California Psychological Inventory: A validation study with college students. *Community Mental Health Journal*, 6:366–373, 1970.

Cook, P.E.; Looney, M.A.; and Pine, L. The Community Adaptation Schedule and the Adjective Checklist: A validation study with psychiatric inpatients and outpatients. *Community Mental Health Journal*, 9:11–17, 1973.

Creer, C., and Wing, J.K. *Schizophrenia at Home*. Surbiton, England: National Schizophrenia Fellowship, 1974.

Crook, T.; Hogarty, G.E.; and Ulrich, R.F. Inter-rater reliability of informants' ratings: Katz Adjustment Scales R Form. *Psychological Reports*, 47:427–432, 1980.

Edwards, D.W.; Yarvis, R.M.; Mueller, O.P.; Zingale, H.C.; and Wagman, W.J. Test-taking and the stability of adjustment scales: Can we assess patient deterioration? *Evaluation Quarterly*, 2:275–291, 1978.

Ellsworth, R.B. Evaluating and applying information about treatment outcome. *Hospital & Community Psychiatry*, 21:115–117, 1970*a*.

Ellsworth, R.B. *The MACC Behavioral Adjustment Scale*. Rev. ed. Beverly Hills, CA.: Western Psychological Services, 1970*b*.

Ellsworth, R.B. Feedback: Asset or liability in improving treatment effectiveness? *Journal of Consulting and Clinical Psychology*, 40:383–393, 1973.

Ellsworth, R.B. Consumer feedback in measuring the effectiveness of mental health programs. In: Guttentag, M., and Struening, E.L., eds. *Handbook of Evaluation Research*. Vol 1. Beverly Hills, CA.: Sage Publications, 1975.

Ellsworth, R.B. The comparative effectiveness of community clinic and psychiatric hospital treatment. *Journal of Community Psychology*, 6:103–111, 1978.

Ellsworth, R.B.; Collins, J.F.; Casey, N.A.; Schoonover, R.A.; Hickey, R.H.; Hyer, L.; Twemlow, S.W.; and Nesselroade, J.R. Some characteristics of effective psychiatric programs. *Journal of Consulting and Clinical Psychology*, 47:799–817, 1979.

Ellsworth, R.B.; Foster, L.; Childers, B.; Arthur, G.; and Kroeker, D. Hospital and community adjustment as perceived by psychiatric patients, their families, and staff. *Journal of Consulting and Clinical Psychology*, 32:1–41, 1968.

Endicott, J., and Spitzer, R.L. Current and Past Psychopathology Scales (CAPPS): Rationale, reliability, and validity. *Archives of General Psychiatry*, 27:678–687, 1972*a*.

Endicott, J., and Spitzer, R.L. What! Another rating scale? The Psychiatric Evaluation Form. *Journal of Nervous and Mental Disease*, 154:88–104, 1972*b*.

Endicott, J.; Spitzer, R.L.; Fleiss, J.L.; and Cohen, J. The Global Assessment Scale—A procedure for measuring overall severity of psychiatric disturbance. *Archives of General Psychiatry*, 33:766–771, 1976.

Evanson, R.C.; Sletten, I.W.; Hedlund, J.L.; and Faintich, D.M. CAPS: An automated evaluation system. *American Journal of Psychiatry*, 131:531–534, 1974.

Farkas, M.D. "The Effects of Training Psychiatric Staff in Human Relations and Programming Skills: Developing a Rehabilitation Model for Chronic Clients During Deinstitutionalization." Unpublished doctoral dissertation, Boston University, Boston, MA, 1980.

Farkas, M.D.; Rogers, E.S.; and Thurer, S. *Rehabilitation Outcome for the Recently Deinstitutionalized Psychiatric Patient: The Ones We Left Behind*. Available from Center for Psychiatric Rehabilitation, Boston University, 1019 Commonwealth Ave., Boston, MA 02215, 1985.

Fontana, A.F., and Dowds, B.N. Assessing treatment outcome: Adjustment in the community. *Journal of Nervous and Mental Disease,* 161:221-230, 1975.

Glazer, W.M.; Aaronson, H.S.; Prusoff, B.A.; and Williams, D.H. Assessment of social adjustment in chronic ambulatory schizophrenics. *Journal of Nervous and Mental Disease,* 168:493-497, 1980.

Goldberg, S.C.; Schooler, N.R.; Hogarty, G.E.; and Roper, M. Prediction of relapse in schizophrenic patients treated by drug and sociotherapy. *Archives of General Psychiatry,* 34:171-184, 1977.

Gripp, R.F., and Magaro, P.A. A token economy program evaluation with untreated ward comparison. *Behaviour Research and Therapy,* 9:137-149, 1971.

Gunzberg, H. *Social Competence and Mental Handicap: An Introduction to Social Education.* London: Baliere, Tindell, & Lassell, 1968.

Gurland, B.J.; Yorkston, N.J.; Stone, A.R.; Frank, J.D.; and Fleiss, J.L. The Structured and Scaled Interview to Assess Maladjustment (SSIAM): I. Description, rationale, and development. *Archives of General Psychiatry,* 27:259-264, 1972.

Hall, J.N. The content of ward rating scales for long stay patients. *British Journal of Psychiatry,* 130:287-293, 1977.

Hall, J.N.; Baker, R.; and Hutchinson, K. A controlled evaluation of token economy procedures with chronic schizophrenic patients. *Behaviour Research and Therapy,* 15:261-283, 1977.

Hargreaves, W.A.; Glick, I.D.; Drues, J.; Showstack, J.A.; and Feigenbaum, E. Short vs. long hospitalization: A prospective controlled study: IV. Two-year followup results for schizophrenics. *Archives of General Psychiatry,* 34:305-311, 1977.

Harris, D.E., and Brown, T.R. Relationship of the Community Adaptation Schedule and the Personal Orientation Inventory: Two measures of positive mental health. *Community Mental Health Journal,* 10:111-118, 1974.

Herz, M.I.; Endicott, J.; Spitzer, R.L.; and Mesnikoff, A. Day versus inpatient hospitalization: A controlled study. *American Journal of Psychiatry,* 127:1371-1382, 1971.

Hirsch, S.R.; Platt, S.; Knights, A.; and Weyman, A. Shortening hospital stay for psychiatric care: Effect on patients and their families. *British Medical Journal,* 1:442-446, 1979.

Hogarty, G.E.; Dennis, H.; Guy, W.; and Gross, G.G. "Who goes there?"—A critical evaluation of admissions to a psychiatric day hospital. *American Journal of Psychiatry,* 124:934-944, 1968.

Hogarty, G.E.; Goldberg, S.C.; and Schooler, N.R. Drug and sociotherapy in the aftercare of schizophrenics: III. Adjustment of nonrelapsed patients. *Archives of General Psychiatry,* 31:609-618, 1974.

Hogarty, G.E.; Guy, W.; and Gross, G.G. An evaluation of community based mental health programs: Long range effects. *Medical Care,* 7:271-280, 1969.

Hogarty, G.E., and Katz, M.M. Norms of adjustment and social behavior. *Archives of General Psychiatry,* 25:470-480, 1971.

Hogarty, G.E.; Katz, M.M.; and Lowery, H.A. Identifying candidates for a community mental health program. In: Monroe, R.R.; Klee, G.D.; and Brady, E.B., eds. *Psychiatric Epidemiology and Mental Health Planning: Psychiatric Research Report No. 22.* Washington, DC: American Psychiatric Association, 1967. pp. 220-234.

Honigfeld, G.; Roderic, D.; and Klett, J.C. NOSIE-30: A treatment sensitive ward behavior scale. *Psychological Reports,* 19:180-182, 1966.

Iljon-Foreman, E., and Baker, R. A validity study of a new rating scale for psychiatric patients. *Acta Psychiatrica Scandinavica,* in press.

Katz, M.M. A typological approach to the problem of predicting response to treatment. In: Wittenborn, J.R., and May, P.R.A., eds. *Prediction of Response to Pharmacotherapy.* Springfield, IL: Charles C Thomas, Publisher, 1966. pp. 209-235.

Katz, M.M. Evaluating drug and other therapies across cultures. In: Marsella, A.J., and Pederson, B., eds. *Cross-Cultural Counseling and Psychotherapy.* New York: Pergamon Press, Inc., 1981.

Katz, M.M.; Gudeman, H.; and Sanborn, K. Characterizing differences in psychopathology among ethnic groups: A preliminary report on Hawaii-Japanese and Mainland-American schizophrenics. In: Caudill, W., and Lin, T., eds. *Mental Health Research in Asia and the Pacific.* Honolulu: East-West Center Press, 1969. pp. 148-163.

Katz, M.M.; Lowery, H.A.; and Cole, J.O. Behavior patterns of schizophrenics in the community. In: Lorr, M., ed. *Explorations in Typing Psychosis.* New York; Pergamon Press, 1967. pp. 209-230.

Katz, M.M., and Lyerly, S.B. Methods for measuring adjustment and social behavior in the community: I. Rationale, description, discriminative validity and scale development. *Psychological Reports,* 13:502-535, 1963.

Klerman, G.L.; DiMascio, A.; Weissman, M.M.; Prusoff, B.A.; and

---

Paykel, E.S. Treatment of depression by drugs and psychotherapy. *American Journal of Psychiatry*, 131: 186–191, 1974.

Kowalski, P.A.; Daley, G.D.; and Gripp, R.F. Token economy: Who responds how? *Behaviour Research and Therapy*, 14:372–374, 1976.

Liberman, R.P. Assessment of social skills. *Schizophrenia Bulletin*, 8:62–83, 1982.

Lorr, M., and Klett, C.J. *Inpatient Multidimensional Psychiatric Rating Scale*. Palo Alto, CA: Consulting Psychologists Press, 1966.

Lorr, M., and Vestre, N. *Psychotic Inpatient Profile*. Los Angeles, CA: Western Psychological Services, 1968.

Mathur, S., and Hall, J.N. Phenothiazine withdrawal in schizophrenics in a hostel. *British Journal of Psychiatry*, 138:271–272, 1981.

Michaux, W.W.; Katz, M.M.; Kurland, A.A.; and Gansereit, K.H. *The First Year Out: Mental Patients After Hospitalization*. Baltimore, MD: John Hopkins Press, 1969.

Nelson, G.L., and Cone, J.D. Multiple baseline analysis of a token economy for psychiatric in-patients. *Journal of Applied Behavior Analysis*, 12:255–271, 1979.

Overall, J.E., and Gorham, D.R. The Brief Psychiatric Rating Scale. *Psychological Reports*, 10:799–812, 1962.

Parloff, M.B.; Kelman, H.C.; and Frank, J.D. Comfort, effectiveness and self-awareness as criteria of improvement in psychotherapy. *American Journal of Psychiatry*, 111:343-351, 1954.

Paykel, E.S. Correlates of a depressive typology. *Archives of General Psychiatry*, 27:203–210, 1972.

Paykel, E.S.; Weissman, M.M.; Prusoff, B.A.; and Tonks, C.M. Dimensions of social adjustment in depressed women. *Journal of Nervous and Mental Disease*, 152:158–172, 1971.

Platt, S.; Weyman, A.; Hirsch, S.; and Hewett, S. The Social Behaviour Assessment Schedule (SBAS): Rationale, contents, scoring, and reliability of a new interview schedule. *Social Psychiatry*, 15:43–55, 1980.

Prusoff, B.A.; Weissman, M.M.; Klerman, G.L.; and Rounsaville, B.J. Research Diagnostic Criteria subtypes of depression as predictors of differential response to psychotherapy and drug treatment. *Archives of General Psychiatry*, 37:796–801, 1980.

Roen, S.R.; Ottenstein, D.; Cooper, S.; and Burnes, A. Community adaptation as an evaluative concept in community mental health. *Archives of General Psychiatry*, 15:36–44, 1966.

Rostow, C.D., and Smith, C.E. Effects of contingency management of chronic patients on ward control and behavioral adjustment. *Journal of Behavior Therapy and Experimental Psychiatry*, 6:1–4, 1975.

Schmidt, C.W., Jr.; Perlin, S.; Townes, W.; Fisher, R.S.; and Shaffer, J.W. Characteristics of drivers involved in single car accidents. *Archives of General Psychiatry*, 27:800–803, 1972.

Schooler, N.; Hogarty, G.E.; and Weissman, M.M. Social Adjustment Scale II (SAS-II). In: Hargreaves, W.A.; Atkinson, C.C.; and Sorenson, J.E., eds. *Resource Materials for Community Mental Health Program Evaluators*. DHEW No. 79-328. Washington, DC: Superintendent of Documents, U.S. Government Printing Office, 1979. pp. 290–303.

Schwartz, J., and Bellack, A.S. A comparison of token economy with standard inpatient treatment. *Journal of Consulting and Clinical Psychology*, 43:107–109, 1975.

Serban, G. Relationship of mental status, functioning and stress to readmission of schizophrenics. *British Journal of Social and Clinical Psychology*, 14:291–301, 1975.

Serban, G. Social Stress and Functioning Inventory for Psychotic Disorders (SSFIPD): Measurement and prediction of schizophrenics' community adjustment. *Comprehensive Psychiatry*, 19:337–347, 1978.

Serban, G. *Adjustment of Schizophrenics in the Community*. New York: SP Medical and Scientific Books, 1980.

Shean, G.D., and Zeidberg, Z. Token reinforcement therapy: A comparison of matched groups. *Journal of Behavior Therapy and Experimental Psychiatry*, 2:94–105, 1971.

Spitzer, R.L.; Endicott, J.; and Fleiss, J.L. Instruments and recording forms for evaluating psychiatric status and history: Rationale, method of development and description. *Comprehensive Psychiatry*, 8:321–343, 1967.

Spitzer, R.L.; Endicott, J.; Fleiss, J.L.; and Cohen, J. The Psychiatric Status Schedule: A technique for evaluating psychopathology and impairment in role functioning. *Archives of General Psychiatry*, 23:41–55, 1970.

Spitzer, R.L.; Fleiss, J.L.; Burdock, E.I.; and Hardesty, A.A. The Mental Status Schedule: Rationale, reliability and validity. *Comprehensive Psychiatry*, 5:384–395, 1964.

Spitzer, R.L.; Fleiss, J.L.; Endicott, J.; and Cohen, J. Mental Status Schedule: Properties of factor-analytically derived scales. *Archives of*

General Psychiatry, 16:479–493, 1967.

Strauss, J.S., and Carpenter, W.T., Jr. The prediction of outcome in schizophrenia: I. Characteristics of outcome. *Archives of General Psychiatry*, 27:739–746, 1972.

Taylor, C.B.; Liberman, R.P.; and Agras, W.S. Treatment evaluation and behavior therapy. In: Lewis, J.M., and Usdin, G., eds. *Treatment Planning in Psychiatry*. Washington, DC: American Psychiatric Press, 1982. pp. 151–224.

Wallace, C.J. Assessment of psychotic behavior. In: Hersen, M., and Bellack, A., eds. *Behavioral Assessment: A Practical Handbook*. New York: Pergamon Press, Inc., 1981. pp. 328–388.

Wallace, C.J.; Boone, S.E.; Donahoe, P.C.; and Foy, D.W. The chronically mentally disabled: Independent living skills training. In: Barlow, D.H., ed. *Clinical Handbook of Psychological Disorder*. New York: Guilford Press, 1985. pp. 462–501.

Wallace, C.J.; Kochanowicz, N.; and Wallace, J. "Independent Living Skills Survey." Unpublished manuscript, Mental Health Clinical Research Center for the Study of Schizophrenia, West Los Angeles VA Medical Center, Rehabilitation Medicine Service (Brentwood Division), Los Angeles, CA 90073, 1985.

Walls, R.T.; Werner, T.J.; Bacon, A.; and Zane, T. Behavior checklist. In: Cone, J.D., and Hawkins, R.P., eds. *Behavioral Assessment: New Directions in Clinical Psychology*. New York: Brunner/Mazel, 1977. pp. 77–146.

Washburn, S.; Vannicelli, M.; Longbaugh, R.; and Scheff, B.A. A controlled comparison of psychiatric day treatment and inpatient hospitalization. *Journal of Consulting and Clinical Psychology*, 44:655–675, 1976.

Waskow, I.E., and Parloff, M.B., eds. *Psychotherapy Change Measures*. Washington, DC: Superintendent of Documents, U.S. Government Printing Office, 1975.

Weissman, M.M., and Bothwell, S. The assessment of social adjustment by patient self-report. *Archives of General Psychiatry*, 33:1111–1115, 1976.

Weissman, M.M.; Klerman, G.L.; Prusoff, B.A.; Sholomkas, D.; and Padin, N. Depressed outpatients: Results one year after treatment with drugs and/or interpersonal psychotherapy. *Archives of General Psychiatry*, 38:51–55, 1981.

Weissman, M.M.; Paykel, E.S.; Siegal, R.; and Klerman, G.L. The social role performance of depressed women: Comparisons with a normal group. *American Journal of Orthopsychiatry*, 41:390–405, 1971.

Weissman, M.M.; Prusoff, B.A.; Thompson, W.D.; Harding, P.S.; and Meyers, J.K. Social adjustment by self-report in a community sample and in psychiatric outpatients. *Journal of Nervous and Mental Disease*, 166:317–326, 1978.

Willer, B.S. Individualized patient programming: An experiment in the use and evaluation of feedback for hospital psychiatry. *Evaluation Quarterly*, 1:587–608, 1977.

Willer, B.S., and Biggen, P. Comparison of rehospitalized and nonrehospitalized psychiatric patients on community adjustment: Self-assessment guide. *Psychiatry*, 39:239–244, 1976.

Willer, B.S., and Guastaferro, J.R. *Community Living Assessment Scale*. Available from Transitional Services, Inc., 2075 Main St., Buffalo, NY 14208, 1985.

Willer, B.S., and Howard, P. Development of a computer assisted progress record for psychiatry. In: Garwick, G., ed. *Goal Attainment Review*. Vol. 1. Minneapolis, MN: Program Evaluation Resource Center, 1974.

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Appendix. Independent Living Skills Survey

The purpose of this questionnaire is to obtain your view of the social adjustment of your son/daughter, relative, or resident of your home or facility. Social adjustment can be measured by how much a person is able to do for himself or herself in self-care such as eating and grooming, domestic activities, health care, money management, transportation, leisure activities, and work. Each of these areas of independent living is covered by this questionnaire.

Please start by using the two scales listed below to score each item. You may find it helpful to detach the scales so that you have them in front of you while you make your ratings on each of the 112 items listed under the right sections of the questionnaire. Rate each of the 112 items according to how often the particular behavior has occurred in the **past month** and how much of a problem the behavior (or lack of it) has been for you, the family, or the facility in which the person lives **during the past month.**

At the end of the questionnaire is a scale for rating the person's overall social adjustment. Please mark an "X" on the line at the point that best describes him/her **during the last month.**

Name of person being rated ______________________________
Name of person doing the rating ____________________________
Relationship of rater to person being rated ______________________
Date____________________

Scale 1: Frequency of occurrence of behavior
For each item in this questionnaire, please record in **Column 1** on the questionnaire, how often each behavior occurred during the past month. If there is no opportunity for the individual to perform a particular behavior (i.e., using the bus when no bus system is available), record **N/O** (no opportunity). Or, if there is no need for the individual to perform the behavior (i.e., the relative or board and care operator, for example, does all of the housework), also record **N/O.**

0	1	2	3	4
Never	Sometimes	Often	Usually	Always

Scale 2: Degree of behavioral problem
For each item in this questionnaire, please record in **Column 2** on the questionnaire, how much the person's behavior is a problem for you. Try to rate each of the items, even if the person isn't presently performing the behavior. Rate the behavior on the basis of the last month.

0	1	2	3	4
Never a problem	Occasionally a problem	Sometimes a problem	Frequently a problem	Always a problem

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Name ____________________
Date ____________________

Please score each item for the **previous month.**	1 **Occurrence during past**	2 **Degree of problem**
I. **Eating**		
1. Drinks neatly (without prompting)		
2. Uses appropriate utensils and dishware (without prompting)		
3. Eats at a reasonable pace without bolting, dawdling (without prompting)		
4. Chews food with mouth closed (without prompting)		
5. Uses napkin (without prompting)		
6. Takes food off others' plates without permission		
7. Limits food intake to appropriate amount (without prompting)		
8. Shows good nutritional habits (i.e., has a fairly well-balanced diet—doesn't live on candy and soda) (without prompting)		
9. Eats food that has been discarded (e.g., from ground, garbage cans, trash bags)		
II. **Grooming**		
1. Bathes or showers using soap at least twice a week (without prompting)		
2. Shampoos hair at least twice a week (without prompting)		
3. Uses deodorant daily (without prompting)		
4. Brushes or combs hair daily (without prompting)		
5. For males, shaves as needed or keeps beard neat (without prompting)		
6. For females, self-care during menstruation (without prompting)		
7. Gets hair cut or styled when needed (without prompting)		
8. For females, wears excessive or bizarre makeup		
9. Brushes teeth (dentures) at least once a day (without prompting)		
10. Changes underwear at least twice a week (without prompting)		
11. Undresses self at designated times (without prompting)		
12. Dresses self at designated times (without prompting)		
13. Maintains clean, neat appearance throughout the day (without prompting)		
14. Appropriately stores soiled clothing for washing by self or other (without prompting)		

Please score each item for the **previous month.**	1 **Occurrence during past**	2 **Degree of problem**
15. Wears bizarre combinations of clothing		
16. Wears clothing appropriate for weather or social occasion (without prompting)		
17. Violates general social etiquette (e.g., scratches inappropriate body parts in public, belches in public without apologies)		
18. Changes clothes excessively (several times a day)		
19. Wears excessive or multiple layers of clothing		
III. **Domestic activities**		
1. Makes bed daily (without prompting)		
2. Keeps room clean (without prompting)		
3. Changes bed linens as needed (without prompting)		
4. Picks up objects from floor (without prompting)		
5. Wipes up spills (without prompting)		
6. Uses vacuum or carpet sweeper as needed (without prompting)		
7. Dusts surfaces as needed (without prompting)		
8. Mops floor as needed (without prompting)		
9. Performs assigned household tasks (without prompting)		
10. Takes laundry to washing machine (without prompting)		
11. Uses proper amount of detergent (without prompting)		
12. Uses washing machine properly (without prompting)		
13. Dries clothes in dryer or hangs on clothesline (without prompting)		
14. Puts clean clothes away (without prompting)		
15. Folds and/or hangs up clothes (without prompting)		
16. Prepares simple foods requiring no mixing or cooking (e.g., sandwiches, cold cereal)		
17. Mixes and cooks simple foods (e.g., fries eggs, cooks TV dinners) (without prompting)		
18. Stores foods appropriately (without prompting)		
19. Can identify and discard foods that are spoiled (without prompting)		
20. Clears table (without prompting)		
21. Uses dishwasher or washes and dries dishes (without prompting)		
22. Puts dishes away (without prompting)		
23. Uses others' possessions inappropriately and/or without permission		

Please score each item for the **previous month.**	**1 Occurrence during past**	**2 Degree of problem**
IV. **Health**		
1. Reports physical problems appropriately (neither over- nor underreports)	________	________
2. Treats own minor physical problems appropriately	________	________
3. Obtains help from appropriate public agency (housing authority, Social Security, or other resource—fire or police department, social worker, doctor, dentist, relative when needed	________	________
4. Follows through on advice from resources named above	________	________
5. Contacts appropriate person (e.g., physician, social worker, board and care operator, parent) to renew a prescription for a medication presently being prescribed	________	________
6. Knows proper use of insurance or welfare coverage (e.g., Blue Cross, Medicare, Medi-Cal) for medical, psychological, or psychiatric treatment	________	________
7. Uses smoking materials safely	________	________
8. Smokes in designated areas	________	________
9. Cooperates with person who dispenses medication	________	________
10. Can reliably self-administer medication (times and dosages)	________	________
11. Contacts public agencies (e.g., police, fire, housing authority) with inappropriate complaints and/or requests (e.g., stops at police station to get a ride home, calls housing authority daily to report different minor problems)	________	________
V. **Money management**		
1. Buys the right amount of groceries (without prompting)	________	________
2. Buys own clothes (without prompting)	________	________
3. Purchases own personal items (without prompting)	________	________
4. Purchases prescribed medication (without prompting)	________	________
5. Gets clothing and household goods repaired (without prompting)	________	________
6. Maintains appropriate identification for cashing checks (without prompting)	________	________
7. Pays bills (including rent, food, clothing, transportation, recreation, and personal effects) (without prompting)	________	________
8. Cashes a pay check or SSI check (without prompting)	________	________
9. Pays bills by money order or check in addition to cash (without prompting)	________	________

Please score each item for the **previous month.**	1 Occurrence during past	2 Degree of problem
10. Budgets money (i.e., plans how funds will be spent)		
11. Contacts person responsible for financial support to ask questions and to relay relevant information (without prompting)		
12. Makes a deposit and withdrawal at a bank (without prompting)		
13. Understands the terms guardianship or conservatorship (if relevant)		
14. Purchases essential items before spending money on luxuries (without prompting)		
15. Has registered to vote		
16. Determines the day's schedule by the clock		
17. Returns defective material or exchanges goods in a store (without prompting)		
18. Counts change in store (without prompting)		
19. Uses telephone inappropriately (e.g., makes long distance calls charged to another number without permission, calls others during the middle of the night)		
VI. **Transportation**		
1. Holds a valid driver's license		
2. Uses buses (without prompting)		
3. Calls bus company to get directions (without prompting)		
4. Reads bus schedule (without prompting)		
5. Walks to places in the neighborhood (without prompting)		
6. Follows pedestrian rules (without prompting)		
7. Asks for and follows directions (without prompting)		
8. Travels long distances (by bus, train, or airplane) (without prompting)		
9. Acts appropriately on buses, trains, or airplanes (without prompting)		
VII. **Leisure**		
1. Works regularly on a hobby (without prompting)		
2. Takes a walk outside the house (without prompting)		
3. Goes to church (without prompting)		
4. Acts appropriately in church (if goes) (without prompting)		
5. Works in the garden or yard (without prompting)		

Please score each item for the **previous month.**	1 **Occurrence during past**	2 **Degree of problem**
6. Listens to the radio or watches TV (without prompting)		
7. Writes letters (without prompting)		
8. Goes to the movies and/or theater (without prompting)		
9. Attends spectator sports (without prompting)		
10. Bowls, plays pool, or other sports (without prompting)		
11. Plays cards or other table games (without prompting)		
12. Reads books or magazines (without prompting)		
13. Reads the newspaper on most days (without prompting)		
14. Attends meetings of civic or other organizations (without prompting)		
VIII. **Job-seeking or job-related skills**		
1. Reads classified ads one or more times per week to look for jobs (without prompting)		
2. Contacts potential employers to determine possible job openings (without prompting)		
3. Contacts friends/peers/social worker/employment agencies for job leads (without prompting)		
4. Participates in job interviews (without prompting)		
5. Has realistic job aspirations		
6. Leaves on time for appointments (without prompting)		
7. Leaves on time for job (without prompting)		
8. Arrives at work on time and follows daily lunch break schedule (without prompting)		

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Training Skills in the Psychiatrically Disabled: Learning Coping and Competence

by Robert Paul Liberman, Kim T. Mueser, Charles J. Wallace, Harvey E. Jacobs, Thad Eckman, and H. Keith Massel

Abstract

Social skills training methods represent a major strategy for psychiatric rehabilitation. Building skills in patients with schizophrenic and other major mental disorders is based on the assumption that coping and competence can override stress and vulnerability in reducing relapses and improving psychosocial functioning. For maximum efficiency, skills training needs to incorporate procedures and principles of human learning and information processing. Several models for skills training have been designed and evaluated, each of which has proved to be effective in raising the social competence of chronic mental patients. The "basic" model involves role playing by the patient and modeling, prompting, feedback, and reinforcement by the therapist. A "problem-solving" model of training provides general strategies for dealing with a wide variety of social situations. This model uses role playing to enhance behavioral performance but also highlights the patient's abilities to perceive and process incoming social messages and meanings. It is essential that social skills training be imbedded in a comprehensive program of rehabilitation that features continuity of care, supportive community services, therapeutic relationships, and judicious prescription of psychotropic drugs.

Given the widening consensus that most major mental disorders are chronic in course, a rehabilitation model that emphasizes the building of skills and prosthetic and supportive environments has supplanted curative and acute treatment models for schizophrenia and other disabling illnesses (Anthony 1980; Liberman 1985; Liberman and Evans 1985). The effectiveness of neuroleptic medications in controlling the positive symptoms of psychosis has enabled many patients to live in the community; however, deinstitutionalization has been accompanied by poor quality of community life (Lehman, Ward, and Linn 1982; Lehman 1983) and a "revolving door" pattern of frequent, albeit relatively brief, rehospitalizations for individuals suffering from chronic and recurrent forms of affective and schizophrenic disorders (Talbott 1978; Goldman, Gatozzi, and Taube 1981). The inadequacy of traditional methods of treatment of schizophrenia is underscored by the unacceptably high readmission rates of approximately 40 percent in a year and 75 percent in 5 years following hospital discharge (Kohen and Paul 1976). Data emerging from longitudinal studies of patients with affective disorders also suggest that chronicity and prolonged social disability mark a significant proportion of individuals suffering from major depressive episodes, including about 22 percent who suffer from "double depression."

One of the most compelling arguments for a skills training, rehabilitation model of chronic psychiatric disorders comes from the failure of antipsychotic drugs to remediate the negative symptoms of mental disorder (Schooler 1986); the serious side effects of neuroleptics which often evoke noncompliance (Van Putten 1974; Kane 1985); and the fact that medications, by themselves, cannot teach patients the coping skills they require for survival

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and maintenance in the community (Paul 1969; Liberman and Foy 1983).

The well-documented deficits in social and living skills of chronic mental patients (see articles by Anthony and Liberman, and Wallace in this issue), together with the unsatisfactoriness of current drug and psychosocial treatments for this population, provide a strong rationale for developing new interventions. Social skills training—directed to problems in the areas of instrumental roles, family relationships, vocation, and friendships and peer support—has become an innovative avenue for psychosocial intervention with severely disabled psychiatric patients. In this article, the authors chart developments in social skills training, beginning with a conceptual model that describes the influences bearing on an individual's social skills from intrapersonal as well as interpersonal sources.

Conceptual Model of Social Skills and Competence

One of the obstacles to the advancement of any new scientific field, whether applied or basic, is the lack of a coherent conceptual framework that enables investigators to (1) find guidance and direction for their research and development activities; and (2) understand the reasons for findings that mesh or appear inconsistent. While the development of social skills training as a major element of psychiatric rehabilitation grew out of the pragmatic "trial and error" traditions of behaviorism and human resource development, it now appears timely to organize and galvanize skills training approaches with an overarching theoretical model. One such model, designed by rehabilitation researchers at the UCLA Clinical Research Center for Schizophrenia and Psychiatric Rehabilitation, is articulated in figure 1.

The model has four major components—social schemata, social skills, coping efforts, and social competence. Basic psychobiological and social cognitive processes constitute an individual's *social schemata*, providing the "raw material" for learning social skills. *Social skills* are the cognitive, verbal, and nonverbal behaviors that must be used interpersonally to achieve needs for community survival and a reasonable quality of life. Social skills include accurate perception of incoming social messages, social problem-solving capacity, and "sending" skills. *Coping* efforts are the individual's attempt to put into practice the social skills that exist within his or her repertoire. The impact of the person's social skills on the relevant interpersonal field or environment, favorable or unfavorable, defines that person's *social competence*.

A social schema is a modifiable information structure that is a prototype in memory of a frequently experienced situation (Rumelhart 1981; Glaser 1984). The individual uses this prototype to interpret instances of related knowledge and to integrate new information. A schema is, in essence, an individual's theory or model that "enables him/her to make assumptions about events that generally occur in a particular situation" (Glaser 1984, p. 100).

When applied to a particular social situation, a schema is the individual's assumptions about the qualities that define a competent performance in that general class of situations, the skills required for that performance, and the responses that can be expected of the environment. The more extensive the individual's experience in that general class of situations, the more finely differentiated his/her schema will be, and the more likely he/she will perform competently.

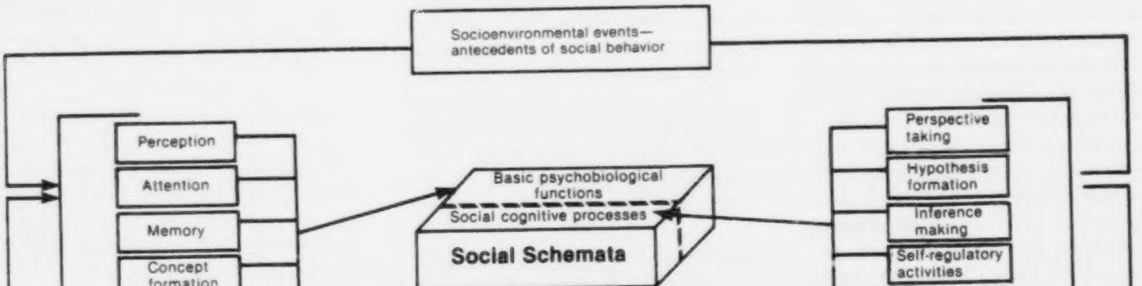
To develop social schemata, certain basic psychobiological functions are necessary, including perception, attention, memory, affect, and concept formation. A deficiency in one of these will severely limit the range and distinctiveness of the individual's schemata and any subsequent coping efforts. In addition, developing social schemata requires several higher order cognitive processes, including the ability to take the perspective of others' intentions, and regulate one's own behaviors. A deficiency in one of these will result in impoverished social schemata, lower skill level, and impaired social coping.

Thus, social schemata that are poorly developed because of inexperience, a deficiency in the psychobiological functions, or a deficiency in higher order cognitive processes may result in social incompetence. Inexperience may lead to incorrect assumptions about the nature of a competent performance, the required skills, or the type and extent of the environmental responses. The individual's social behaviors may be hasty and poorly fashioned; on the other hand, he/she may be inactive because of an overestimate of the required skills or an underestimate of his/her abilities. Deficiencies in the higher order cognitive skills may lead to invalid hypotheses about others' behaviors. Misinterpretations may result, and behaviors may be performed that are inappropriate to the individual's and others' actual goals and intentions. Deficiencies in the basic psychobiological functions may lead to either an inattentiveness to the relevant stimuli or an inability to store them for later processing.

Basic psychobiological functions, behavioral competencies, and social cognition together exert reciprocal influences on self-efficacy and interpersonal problem-solving skills. Self-efficacy is governed both by social schemata and successful social outcomes (Bandura 1977). The social schemata carried by an individual at any time will affect the person's ability to evaluate correctly a social situation that is about to be entered, to determine whether the requisite skills to be effective in the situation are in one's repertoire, to know whether a means to implement the skills is at hand, and to evaluate motivation or desire for using the skills in action. These cognitive and motivational factors represent self-efficacy, and they determine the likelihood and persistence of coping efforts in the situation.

Coping efforts then lead to the use of interpersonal problem-solving skills in the situation. These skills, also affected by the individual's social schemata, include social perception (*receiving* skills), generation of alternatives and their evaluation (*processing* skills), and verbal and nonverbal behavioral responses

Figure 1. Conceptual model for constituent variables related to social skills and social competence


Socioenvironmental events—antecedents of social behavior
Perception
Attention
Memory
Concept formation
Affect
Basic psychobiological functions
Social cognitive processes
Social Schemata
Perspective taking
Hypothesis formation
Inference making
Self-regulatory activities
Set & social rules
Reciprocal influences
Reciprocal influences
Self-Efficacy
Coping efforts
Interpersonal Problem Solving Skills
Correctly identify situation & actors?
Appropriate skills for situation?
Appropriate implementation strategy for situation?
Desire for action?
Receiving skills
Correctly identify desired outcomes
Correctly identify other's goals, needs, and emotions
Processing skills
Generate alternatives and their consequences
Evaluate feasibility and likely attainment of goal
Sending skills
Implement to maximize outcome
Verbal components
Nonverbal components
Content
Timing
Social Competence
Socioenvironmental contingences of reinforcement—consequences of social behavior

Social schemata influence social coping and social skills and are organized from basic psychobiological processes and social cognitions. The effectiveness of an individual's social skills in attaining desirable instrumental and affiliative goals determines social competence.

(*sending* skills). Thus, social schemata (basic psychobiological functions plus social cognitive processes) interact with both self-efficacy and interpersonal problem-solving skills to determine the success or failure of the individual's actual efforts in social transactions. The more success the person experiences with more of his/her needs met through social contacts, the greater the person's social competence. Competence is defined by the outcomes of social interactions and the degree to which a person is able to cope and use interpersonal skills to obtain instrumental and affiliative needs.

In figure 1, the role of the social environment is shown as a frame or border around the intrapersonal elements of social competence. Life events and social stimuli trigger the operation of an individual's social schemata, leading to self-evaluation of efficacy and coping with the event or social situation. The contingencies of reinforcement and social "rules" and expectancies, at the other end of the process, determine whether the individual has used schemata, coping, and skills appropriately and effectively. Successful social outcome, or competence, in turn, has an impact on social schemata, self-efficacy, and social skills. The feedback loops in figure 1 illustrate the bidirectional manner in which these processes influence one another.

Implication of Conceptual Model

The theoretical processes in figure 1 are heuristic for both researchers and clinicians. For researchers, they suggest hypotheses to test; for example, will patients suffering from schizophrenia who have deficits in attention, memory, concept formation, and affect exhibit less self-efficacy, coping, and interpersonal problem-solving skills? Can direct training of basic psychobiological functions improve social skills and social competence? Can self-efficacy and coping be shown to increase or decrease depending on the outcomes of social transactions?

For clinicians, several modifications of customary therapy processes are suggested for patients with deficits in basic psychobiological functions. For example, in psychotic patients with anhedonia and amotivational states, it is desirable to build into the therapy or skills training procedure extrinsic sources of motivation or incentives. The use of tokens, food, or monetary reinforcement is helpful in the social learning of such patients (Paul and Lentz 1977). In patients lacking perceptual and attentional abilities, offering such "prostheses" as boldly written posters and handouts, use of videotape models, and repetitions of instructions and feedback improve engagement in social skills training (Liberman, Nuechterlein, and Wallace 1982).

The conceptual framework serves to identify three major focal points for the design and testing of social skills training methods. The first option is to train the basic psychobiological and cognitive functions that form the person's social schemata. For example, attention span and higher order abstraction have been improved in preliminary studies carried out in experimental psychopathology laboratories (Brenner 1986; Spaulding et al., this issue). A second approach is to train the *receiving*, *processing*, and *sending* skills of individuals using behavior rehearsal, coaching, reinforcement methods, modeling, and homework. The third strategy that derives from the model is to reprogram the individual's natural environment such that skills—however well developed—can be assured of support and favorable response by others. This strategy is often used in combination with the first two approaches.

Direct Training of Social Cognitive Skills

For regressed, thought-disordered, and highly distractible schizophrenic patients, directive training of basic cognitive processes is a prerequisite to broader induction of social skills. A significant minority of schizophrenic patients have such severe cognitive, memory, and attentional impairments that they cannot productively participate in group-based training of social skills that requires sustained attention, ability to follow general instructions, and voluntary involvement in role playing. Furthermore, conventional social skills training methods assume that patients can generalize social concepts after minimal exposure to multiple elements of a stimulus class: for example, to learn that self-disclosure is appropriate in a situation where one's conversational partner has self-disclosed and is asking open-ended questions that solicit personal information about oneself. As can be seen in figure 1, deficiencies in basic psychobiological functions and social cognitive processes—including concept formation and inference making—markedly interfere with generalization of such social experiences.

The use of behavioral learning procedures to teach distractible and incoherent patients better cognitive skills is promising. One group of behavior analysts trained simple greeting responses in regressed State hospital patients by systematically introducing and fading prompts and reinforcers in a discrete trials format

(Kale et al. 1968). Overlearning and repetitious training of exemplars of greetings resulted in generalization of the greeting behavior to persons not involved in the training. Several replications and extensions of this approach, using single subject experimental designs, confirmed the ability of regressed patients to learn social responses under highly constrained and directive training protocols (Wallace and Davis 1974; Fichter et al. 1976; Wallace et al. 1980). Training included frequent and immediate reinforcement for minimal social verbalizations. Both positive (e.g., tokens exchangeable for various edibles or cigarettes) and negative (e.g., escape from the noxious social interaction) reinforcers were used to build conversational skills. These studies suggested that patients could extend their social skills development as a consequence of initial training.

Three groups of workers are currently expanding this work by developing programmatic approaches to training attentional, perceptual, and self-regulatory activities. In each group, the focus is on training patients who have been refractory to various psychosocial and pharmacotherapeutic interventions. A group in Switzerland has designed a five-step program that begins with training of attentional skills and, in the course of 3 months, teaches patients basic conversational skills (Brenner 1986). Spaulding and his co-workers in Nebraska have focused their training efforts on simple vocational skills as a means of improving attention span (see Spaulding et al., this issue). A group at the Camarillo-UCLA Clinical Research Center have developed an attention-focusing procedure for teaching conversational skills (Liberman et al. 1985).

Attention-Focusing, Training of Conversational Skills

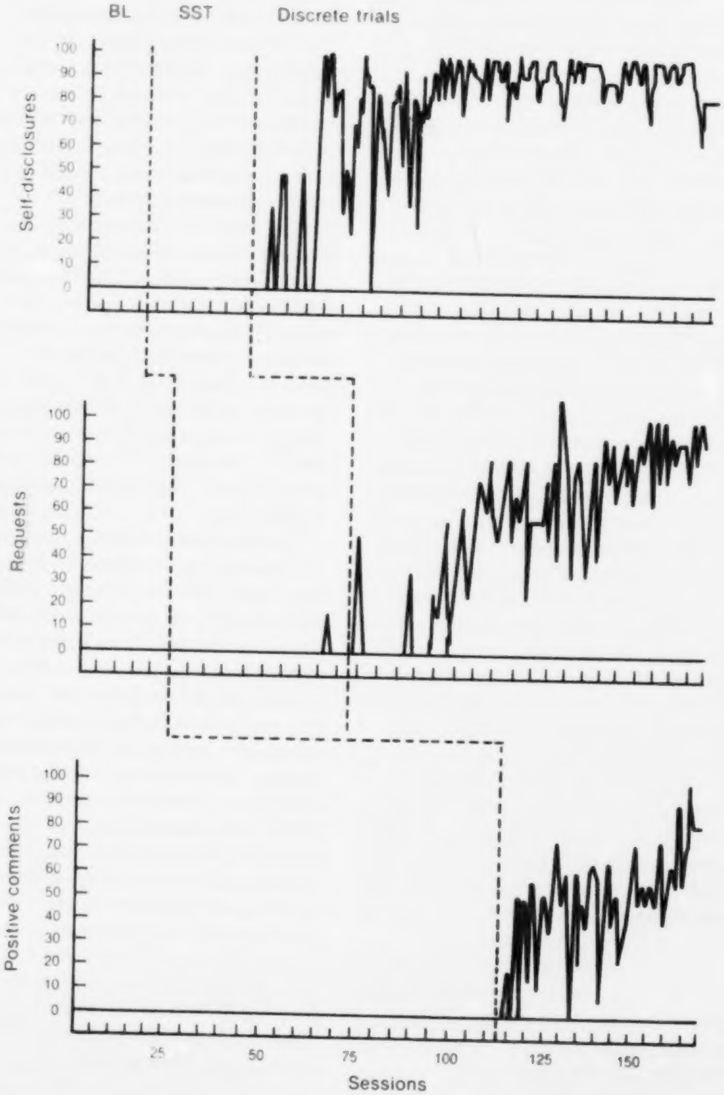
The attention-focusing procedure involves systematic repetitions of training content, a graduated prompting sequence, and consistent and immediate reinforcement—all applied within a discrete trials format. This training procedure readily elicits the patient's attention on the relevant training curriculum while minimizing demands on cognitive and information-processing capacities. The repetitious but brief training sessions reduce the chances of distractibility by careful manipulation of the training components in each learning trial. When this type of intensive training is provided twice daily for 20 minutes each, repertoires of conversational skills can be established.

The discrete trials format requires a trainer and a training confederate, who could be a higher functioning patient or an aide. The confederate serves as the patient's conversational partner and presents conversational "openers" to the patient. The trainer provides instructions, prompts, and reinforcement. In a typical trial—there may be up to 20 trials in a single training session—the training confederate makes a statement to the patient, such as "I went shopping last night." If the patient makes an appropriate response (e.g., "What did you buy?"), the trial is terminated and reinforcement is provided. If there is no response or an inappropriate response, the trainer delivers a prompt to the patient—"Ask him a question." If the patient still does not ask a question of the confederate, a second prompt is given—"One good question is 'What did you buy?' " If the patient still does not ask a question, that prompt is repeated. The trial ends after the third prompt, whether or not a correct response was made.

The patient demonstrates internalization of the learning by making four consecutive, correct, unprompted responses in successive trials. Once a particular behavioral response is learned, another response is brought into the training trials and training proceeds using prompts and reinforcement until the patient evinces four correct responses without prompting in successive trials. Repertoires are built by adding length and complexity to the interactional sequences of responses being taught. For example, conversational skills of a functional type require the use of sequences of responses such as asking a question, followed by a positive comment, followed by self-disclosure, followed by asking a question, etc.

The attention-focusing procedure for teaching conversational skills was tested with three patients who were trained to (1) ask questions, (2) give compliments, and (3) make requests to engage in activities with others. A multiple baseline experimental design was used in which the discrete trials training for each of the three conversational skills was sequentially introduced over time after a baseline period and a second period of attempting to train these skills using conventional role-playing methods. As shown in figure 2 for one of the three patients, acquisition of the conversational skills occurred only after the discrete trials, attention-focusing procedure was instituted. These findings were replicated in the other two patients as well. Moreover, generalization of the skills to nonconfederates on the ward was observed for all three patients with the use of a minimal prompt-reinforcement protocol by nursing staff.

Figure 2. Conversational skills of a chronic schizophrenic patient as a function of social skills training



A multiple baseline (BL) analysis of the conversational skills of a chronic, thought-disordered schizophrenic male as a function of basic social skills training (SST) and attention-focusing skills training (discrete trials). Efficacy of training is done by the percentage of correct, unprompted conversational responses made during the training sessions. These data are reproduced from an unpublished doctoral disseration of H. Keith Massel which is available from the author at the Camarillo-UCLA Research Center, Box A, Camarillo, CA 93011.

Training of Social and Independent Living Skills

While not requiring the intensive, discrete trials method of training needed by severely regressed and inattentive patients, most individuals with chronic schizophrenia do learn social and independent living skills only through involvement in structured and directive instructional sessions (Liberman et al. 1975; Goldstein, Sprafkin, and Gershaw 1976). The teaching procedures are based on learning principles and include goal setting, focused instructions, modeling, behavior rehearsal, prompting, social reinforcement, shaping successive approximations to desired behaviors, in vivo practice of skills, and homework assignments.

Social skills can be defined as interpersonal behaviors that are: (1) instrumental for maintaining and optimizing independence and community survival; and (2) socioemotional for establishing, maintaining, and deepening supportive personal relationships. While early definitions of social skills were limited strictly to the domain of overt motoric behavior, cognitive and affective behavioral modalities are now included as relevant dimensions of this construct.

Social skills training resembles a classroom teaching environment more than a traditional therapy setting. Sessions require the active participation of the patient(s) and the therapist; may be conducted with individual patients or in groups; and may be as brief as 10 minutes a day or as long as 2 hours, depending on the attentional capacities of the patients. Massed practice (i.e., multiple training sessions per week) is preferred to learning less intensively over a longer period. Agendas specifying the behavioral goals are planned with a patient's input and implemented using specific procedures following written guidelines derived from a trainers' manual. Role playing (behavior rehearsal) is the main vehicle for both assessing and teaching social skills, which are targeted for intervention on the basis of their functional relation toward attaining a specific goal important to the patient. Three types of behavior are usually targeted for modification: *response topography* behaviors, such as voice volume, fluency, eye contact; *content* behaviors, such as making a positive statement or requesting additional information; and *cognitive problem-solving* skills (Wallace et al. 1985).

Basic Model of Social Skills Training

To give a more graphic picture of how social skills training is actually done, the next two sections provide details on the procedures used by trainers/therapists conducting the most commonly used "basic model" and the more recently developed "problem-solving model." The basic model is shown as a clinical decision-tree in the flow chart of figure 3. The procedures can be described in five steps.

1. The trainer identifies the interpersonal problem of a patient by asking the following questions: *What* emotion, need, or communication is lacking or not being appropriately expressed, and how often does the behavior occur? *With whom* does the patient desire to improve social contact? *When* does the problem occur? *Where* does the problem occur? A wide variety of techniques can be used to assess interpersonal problems, including naturalistic observation, self-report measures, reports of significant others, and role-play performance. As in other behavior therapy interventions, assessment is an ongoing process that occurs before, during, and after social skills training.

2. In specifying the goals of training, the trainer and patient together target new behaviors that either rectify deficits in performance or modulate excessive or overly intense emotional expressiveness. Targeted behaviors are chosen with the patient on the basis of their functional relation to achieving a positive goal that is attainable within the patient's learning capacities. Goals should be articulated as specific behaviors that are high in frequency, to provide more opportunity for practice and feedback. The relevant domains of goal setting—behaviors and emotions, relationships, and settings—which combine to set the stage for overcoming the problem and moving toward improved social and emotional functioning are outlined in table 1.

3. The patient is engaged in a role play of the problem situation, using other members of the group to play relevant roles. These scenes are usually events that have occurred in the recent past or are likely to occur in the near future. The first role play of each problem situation is a "dry run" done "naturally" by the patient, who is simply instructed to act as he would if he were in the actual situation. Following the "dry run," which may be videotaped for immediate feedback, the patient's assets, deficits, and excesses in his role-play performance are noted. The patient is praised for appropriate behaviors and efforts, and positive feedback is solicited from other group members.

4. In a series of role plays, direct instructions, *modeling* or behavioral demonstration, *shaping* (reinforcing successive approximations), and *coaching* (verbal or nonverbal prompts given by a therapist to elicit specific behaviors in a role play) are used to modify the patient's behaviors toward the goal. Elements of the total "gestalt" are added one by one, such as eye contact, facial expression, vocal tone and loudness, posture, and speech content. After each role play, good elements of the patient's performance are praised by the therapist and group; corrective feedback is given about deficits.

Table 1. Some characteristics of goal setting in social skills training

Targeted behaviors

Asking for or giving information
Initiating or terminating conversations
Maintaining conversations
Response topography (eye contact, voice volume, affect)

Targeted emotions

Affection, love
Anger, annoyance, hostility
Assertiveness, dominance
Frustration
Happiness, pleasure, delight
Interest, empathy
Sadness, grief

Interpersonal targets

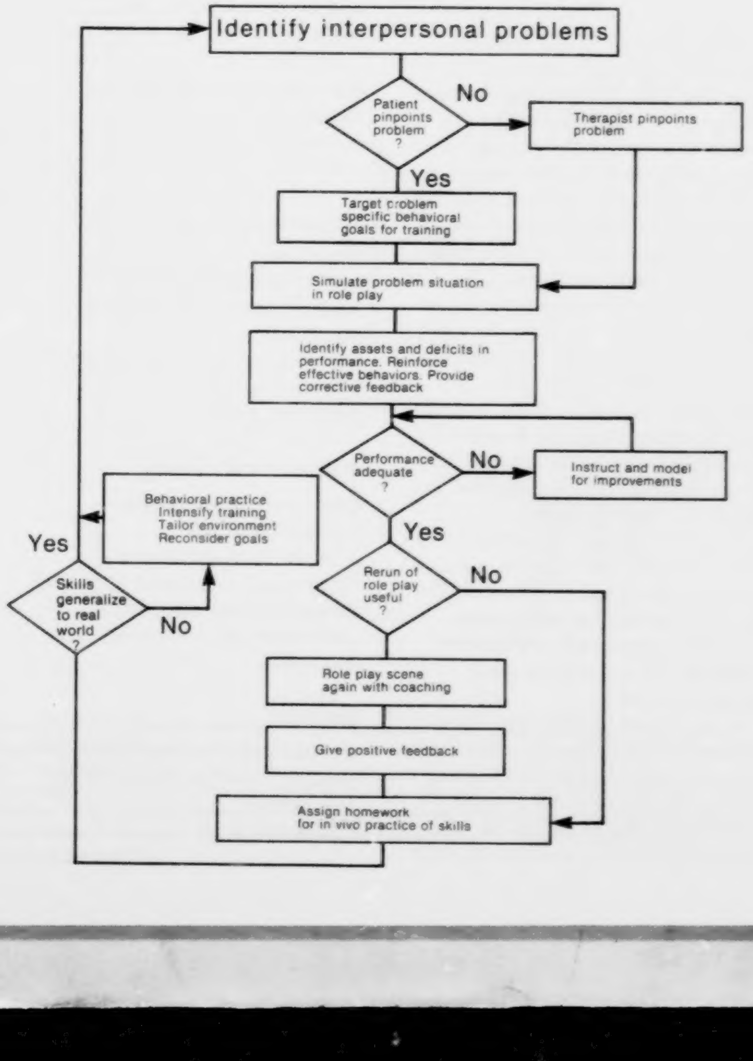
Employers or employees
Family members
Friends, acquaintances
Hospital or board-and-care staff
Sales persons, agency bureaucrats
Strangers

Settings

Home, board-and-care facility
Hospital, mental health center
Job, school
Public place

5. The trainer promotes generalization of the newly learned behavior to situations outside the training sessions by giving homework assignments to practice the skills in the natural environment and giving positive feedback for successful transfer of skills. Generalization of skills is improved when training is not separated from the patient's everyday world, but integrated into it. Whenever possible, therapy should be taken out of the clinician's office and practiced in homes, wards, schools, stores, restaurants, and other environments where it is desirable to perform the target behaviors. Transfer of skills can also be facilitated by repeated practice and overlearning, teaching the patient to use self-evaluation and self-reinforcement, fading the structure and frequency of training, and ensuring that the natural environment is indeed socially responsive and reinforcing to the patient's skill performance. Friends, family members, nursing staff personnel, and peers can aid this process by prompting and reinforcing new social behaviors until they are established.

Figure 3. Flow chart depicting the steps used in conducting basic social skills training


Identify interpersonal problems
Patient pinpoints problem ?
No
Therapist pinpoints problem
Yes
Target problem specific behavioral goals for training
Simulate problem situation in role play
Identify assets and deficits in performance. Reinforce effective behaviors. Provide corrective feedback
Performance adequate ?
No
Instruct and model for improvements
Yes
Behavioral practice
Intensify training
Tailor environment
Reconsider goals
Yes
Skills generalize to real world ?
No
Rerun of role play useful ?
No
Role play scene again with coaching
Give positive feedback
Assign homework for in vivo practice of skills

Problem-Solving Training Model

Many chronic psychiatric patients are deficient in basic problem-solving skills (Edelstein et al. 1980; Wallace et al. 1980). Recently, training within an information-processing framework has been shown to be effective for those patients capable of learning problem-solving strategies (Foy, Wallace, and Liberman 1983). In this model patients are taught to improve their perception of incoming stimuli from immediate interpersonal situations, process those stimuli meaningfully to select an appropriate response, and send an effective verbal and nonverbal response back to the other person.

Interpersonal communication is viewed as a three-stage process requiring *receiving*, *processing*, and *sending* skills (Wallace et al. 1980). Receiving skills refer to the accurate perception, interpretation, and comprehension of relevant situational parameters. Processing skills involve weighing and selecting response options, and determining an implementation strategy. Sending skills are the verbal and nonverbal behaviors emitted in the interpersonal situation that are necessary steps toward attaining the specified goal.

As in the basic model, an interpersonal scene is role played, and preferably videotaped. After each role play, the therapist asks specific questions to assess the patient's receiving and processing skills, exemplified by the list contained in table 2. After the patient has shown acceptable receiving and processing skills, his sending skills are assessed by reviewing his videotaped role play.

Clinical researchers at the Clinical Research Center for Schizophrenia and Psychiatric Rehabilitation at the Brentwood Psychiatric Division of the West Los Angeles VA Medical Center have developed a comprehensive social skills program for chronic psychiatric patients based on the problem-solving model. This program includes "modules" that are being developed in areas such as medication management, leisure and recreation, self-care and personal hygiene, food preparation, money management, and friendship and dating. Training is done in small groups, meeting in 1½-hour sessions, one to three times weekly for a period of 2 to 3 months, depending on the patient's clinical status and premorbid level of adjustment.

Table 2. Receiving and processing skill questions asked in training social problem solving skills

Receiving questions

What did the other person say?
What was the other person feeling?
What where the patient's short-term goals?
What were the patient's long-term goals?
Did the patient obtain his goals?

Processing questions

What other alternatives could the patient use in this situation?
If the patient were to do (an alternative), what would the other person feel?
If the patient were to do (an alternative), what would he feel?
Would the (alternative) help the patient achieve his short-term goals?
Would the (alternative) help the patient achieve his long-term goals?

One of the modules, Medication Management, has been empirically evaluated and field tested in 30 facilities nationwide. An example of evaluation data, collected before, after, and at 3 months' followup, is shown in figure 4. The evaluation of behavioral skills in the four topical areas relevant to reliable use of neuroleptic drugs was carried out through role-play simulation of the criterion situation. Significant improvements in knowledge of medication benefits, self-administration of medication, coping with side effects, and negotiating medication matters with a physician were found. Erosions in knowledge were less than 12 percent at the 3-month followup point. The Medication Management Module is described at length in another publication (Wallace et al. 1985) and is available from the Clinical Research Center.

Skills Training in Vocational Rehabilitation

Despite the obvious advantages of employment for psychiatric patients, unemployment has been reported to be as high as 70 percent in the chronically mentally ill in the United States (Goldstrom and Manderscheid 1982). Unemployment among mental patients reflects both their difficulty in obtaining and maintaining jobs. While approximately 25 percent of patients return to work within 6 months of hospital discharge, only 15 percent are still employed by the 1-year followup point (Anthony, Cohen, and Vitalo 1978).

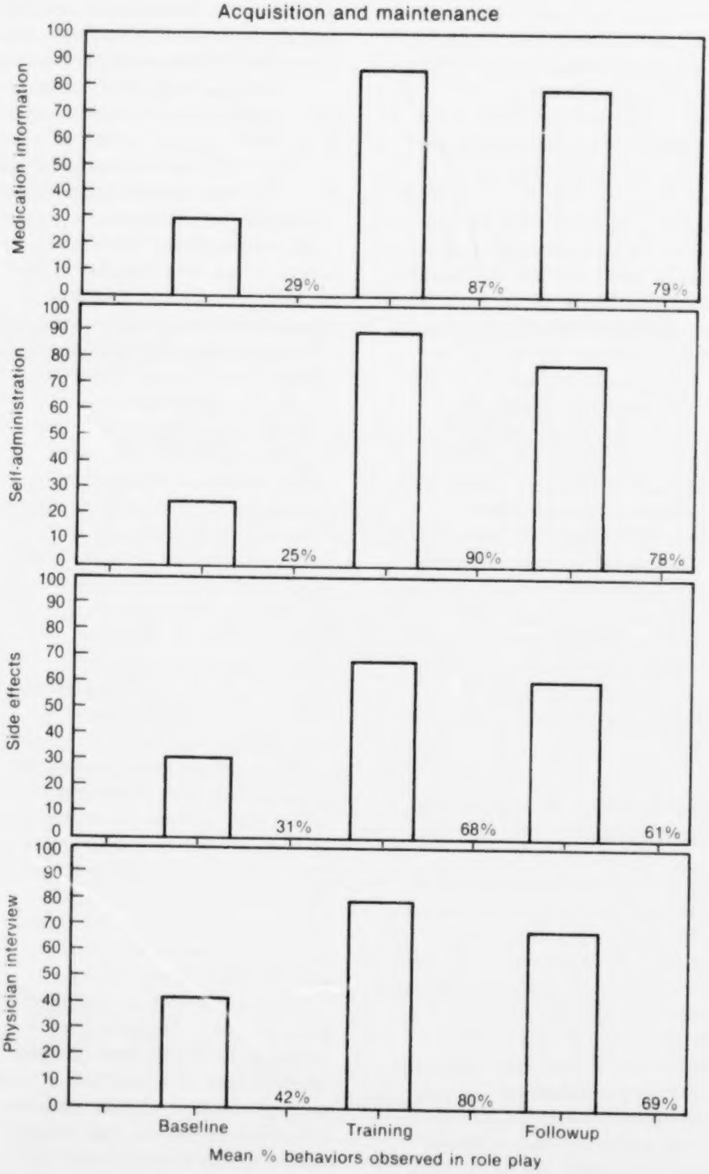
The experience of being chronically unemployed is highly aversive to individuals with psychiatric disorders, common stereotypes and misconceptions to the contrary. In a survey of 500 chronic mental patients residing in Los Angeles board-and-care homes, Lehman, Ward, and Linn (1982) found that lack of work was one of the greatest complaints related to poor quality of life. Even chronically impaired patients supported by Social Security pensions had not relinquished their aspirations for a job, and their dissatisfaction with unemployment and leisure time was significantly greater than that of a cross-section of the normal population.

Since many patients who are capable of assuming full-time employment will not have jobs waiting for them when they leave the hospital, job placement is an important element in their rehabilitation and integration back into the community. How patients present themselves at job interviews is a critical determinant of whether they obtain work. Psychiatric patients often have special problems responding to questions about their personal circumstances and recent past. Studies have demonstrated that psychiatric patients can benefit from training in interview skills (Kelly et al. 1979). Additional skills for obtaining employment include knowing how to solicit job leads, and having the motivation and persistence to sustain a long, frustrating job search. One fruitful program for overcoming the obstacles to employment has been the establishment of a Job-Finding Club for recovering psychiatric patients whose symptoms are well enough controlled for them to work full time.

Job-Finding Club. The Job-Finding Club combines several successful techniques in a packaged module first developed by Azrin (Azrin and Phillip 1979; Azrin and Besalel 1980). Key elements of the module include: (1) the use of an environment conducive to motivating patients in their job search, (2) use of reinforcement strategies, (3) a breakdown of the tasks involved in finding a job, and (4) the training of skills needed to find a job. To adapt this model to the needs of the psychiatrically disabled, it was necessary to increase the structure and motivation inherent in the program, including daily goal-setting activities, monetary rewards, and remedial training in job-seeking skills. The Job-Finding Club for psychiatric patients was designed and evaluated at the Brentwood Division of the West Los Angeles VA Medical Center (Jacobs et al. 1984). Patients participated in the program full time (6 hours per day) while they either lived in the hospital or in the community. While time for participating in the program is unlimited, patients spent an average of 24 days in the Club before locating employment. There are three distinct parts to the Club: training in job-seeking skills, the job search itself, and followup and job maintenance.

Training in job-seeking skills. During the first week of the program, patients participate in an intensive 6 hour per day workshop designed to assess and train basic job-finding skills. The curriculum includes identifying sources of job leads, contacting job leads, writing job resumes, filling out employment applications, participating in job interviews, and use of public transportation. Instruction is competency-based, with trainers using programmed materials, didactic instruction, role plays, and in vivo training exercises. Whenever possible, the program uses materials

Figure 4. Evaluation of the Medication Management Module



A group of 3 chronic schizophrenic patients on maintenance neuroleptic medication were rated for the presence or absence of skills taught in each of 4 areas. Ratings were made in novel role-played situations that were similar to the situations in which the patients were trained. Adapted from Wallace et al. 1985.

and situations that the client will face during the job search, such as filling out actual job applications and contacting sources for job leads. Patients' progress is closely monitored and additional instruction is provided as necessary to meet individual needs.

Job search. After completing the 5-day workshop on skills required for job seeking, patients begin their job search. The program provides areas for telephoning, secretarial support, and identifying current job leads. These leads are gleaned from newspaper want ads, employment notices, civil service announcements, the yellow pages, and visits to job placement counselors from State employment agencies. A daily, intensive goal-setting session is conducted with each patient to plan his job-search activities. During this session counselors and patients identify the most advantageous options for the day's search. They also develop outcome expectations for the daily activities, set a time frame for accomplishing the task, and carry out problem solving of potential obstacles that may be encountered during the day. Patients keep a log of their daily job-seeking activities to account for their time in the program.

Job maintenance. Club graduates may attend a weekly session that teaches strategies to deal with problems that may threaten job security. Training follows a problem-solving model specifying solutions to an identified issue and then role playing these solutions with feedback before the patient uses the approach in his work setting. Problem issues are identified by the participants and may include learning how to get along with co-workers on the job, improving daily living conditions, and managing residual psychiatric symptoms. Graduates may also return to the program if they lose their jobs or wish to upgrade their positions.

Evaluation. The effectiveness of the Club was supported by the outcome of the first 97 patients enrolled in the program. The majority of these patients had hospital admission diagnoses of schizophrenia and a history of psychiatric hospitalization. The average patient had been unemployed for over 1½ years before his present hospitalization. The results of the Job-Finding Club during the first 8 months of operation revealed that 66 percent of all the patients who entered the Club either obtained employment or were enrolled in full-time job-training programs. Most jobs were secured in clerical and sales positions (34 percent); service occupations (25 percent); technical, managerial, or professional positions (11 percent); and machine trades (7 percent).

Six-month followup data were collected on the 66 patients who entered jobs or job-training programs from the Job-Finding Club. The percentage of employed people was 75 percent over the half year following leaving the Club. In comparison, out of the 25 patients who left the Job-Finding Club without successful placement, none of them had found a job 6 months later (Jacobs et al. 1984). Job outcomes have remained stable since the Club's initiation 3 years ago. Out of a total referred patient group of approximately 300, 65 percent of the participants obtained jobs or entered full-time vocational educational programs. Patients with positive symptoms had greater difficulties finding a job, as did older patients. Previous work history and education did not predict success in finding a job, but did affect the type of employment secured. Thus, the Job-Finding Club is a viable program for training and preparing psychiatric patients in the skills necessary to find employment in the competitive workplace.

Generalization of Social Skills Training

The most challenging obstacle to rehabilitation practitioners involved in training skills with chronic mental patients is the difficulty of transferring trained skills into patients' natural living, working, and learning environments. Some patients show no transfer of skills taught them in training sessions while others evince incomplete generalization or poor maintenance of skills. If skills training is to be a clinically effective method for rehabilitating patients, then it will be necessary to improve the generalization of skills from the clinic or hospital into the patient's natural environment.

Tactics aimed at facilitating generalization from hospital to community have been evaluated in a case study by Liberman, King, and DeRisi (1976). Maintaining consistency in reinforcement contingencies from hospital to community settings, using natural reinforcers, pinpointing functional behaviors as therapeutic goals, overlearning, and training natural caregivers were methods that promoted generalization.

Several specific suggestions for promoting generalization (Stokes and Baer 1977) have not as yet been fully tested in work with chronic mental patients. The social skills trainer might consider:

- Cueing patient's significant others (families, friends, agency workers, employers) to reinforce the patient's gradually improving skills.

- Training many exemplars of the situations in which the skills need to be used; in particular, diversifying situations during training that prepare patients for a variety of real-life settings.
- Loosening control over the stimuli and responses involved in training; in particular, training different examples concurrently, varying instructions, social reinforcers, and backup reinforcers.
- Blurring the contingencies and expectations operating during training; in particular, concealing, when possible, the point at which those contingencies stop operating, possibly by delaying reinforcement.
- Using stimuli in the training setting that are likely to be found in generalization settings; in particular, using peers as tutors may aid transfer of skills.
- Reinforcing accurate self-reports of desirable behavior, applying self-recording and self-reinforcement techniques whenever possible.
- When examples of generalization occur, reinforcing at least some of them at least sometimes.

Social skills training techniques, while effective in helping patients acquire interpersonal skills, require the inclusion of procedures that specifically facilitate the generalization of the learned skills into the patient's real-life settings. Generalization does not ordinarily occur spontaneously; it must be planned and programmed. The organization of the real-life settings, orchestrated by the clinician conducting the skills training, to promote support by significant others for the patient's new-found skills is a key element in the overall training process.

Homework assignments to practice skills in natural environments can be made more successful with the addition of modest efforts at instructing key others to prompt and reinforce the trainee for practicing skills. A study of conversational skills in chronic psychiatric inpatients of a State hospital revealed little or no generalization from the training room to the wards and grounds of the hospital. When systematic homework assignments were provided with backup reminders and praise from ward staff for completing the practice, generalization was found, even with total strangers (Martinez-Diaz et al. 1983).

Training Recreational and Leisure Skills

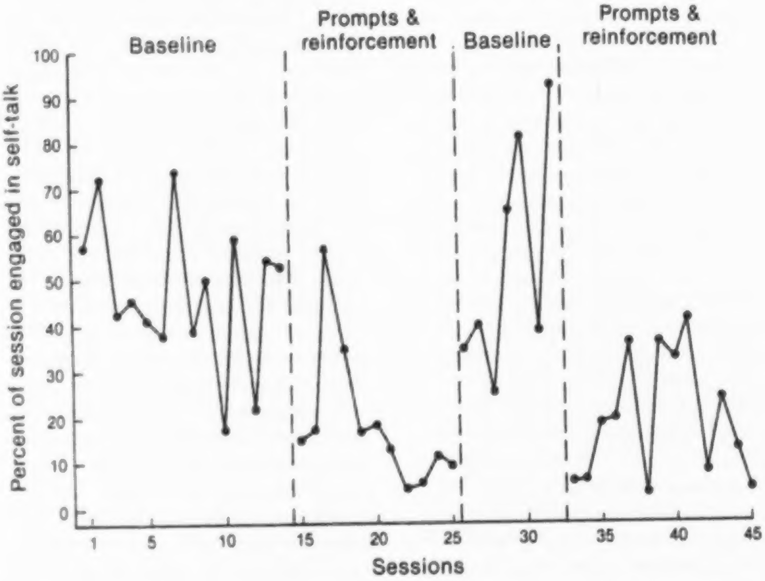
For those patients unable to seek sheltered or competitive employment because of persisting and intrusive psychopathology, the skills training approach can still provide benefits. Such patients need to be engaged in activities that might be considered precursors of more demanding social skills and that could "displace" some of their psychopathology. Unless their symptoms and bizarre behaviors are at least partly reduced in frequency and intensity, their self-efficacy and coping efforts will be obliterated and their constructive participation in various forms of skills training will be blocked.

Consider the difficulty an individual would have entering relevant social situations for transacting with others if the person had a high rate of talking to himself or showed strange posturing. It is hard enough to overcome social anxiety and longstanding social deficits, but the presence of stigmatizing signs of severe mental disorder will blunt coping efforts and suppress self-efficacy as depicted in figure 1. Can individuals be trained to overcome these stigmata of their disorders and thereby to become candidates for social skills training?

Behavioral observations, made objectively with time-sampling rating codes, have repeatedly documented the inverse relation between the frequency of idiosyncratic behaviors (e.g., pacing, repetitive gestures, posturing, and self-verbalizations) and the amount of structured programming present in the living environment. Whether studied in hospitals, mental health centers, or board-and-care homes, patients exhibit significantly more deviant behaviors during unstructured, "free" time (Liberman et al. 1974; Paul and Lentz 1977; Rosen et al. 1981).

The corollary of these observations is the displacement of bizarre behaviors by engaging patients in recreational and social activities. A series of controlled studies has been carried out at the Camarillo-UCLA Clinical Research Center that examined the influence of a variety of leisure activities in the reduction of bizarre motoric and verbal behaviors (Wong et al. 1985). Unstructured time on the inpatient unit during which patients had access to recreational materials (e.g., crafts, books, playing cards, and music) but were not prompted for their use was compared to other times when the patients were prompted and reinforced for participation in these activities. Obsessive-compulsive ruminations, posturings, inappropriate laughter, mumbling to self, and other bizarre behaviors were significantly reduced when patients were engaged in structured recreational activities. The data in fig. 5 from a case study of a patient with hallucinatory speech illustrate the displacement of dysfunctional behaviors with recreational therapy. Without the structured prompting and reinforcement, the patient's engagement in activities waned and his hallucinatory speech markedly increased. Further analysis of the effective components of recreational therapy showed that instructions and prompting were crucial, but reinforcement was not, aside from the intrinsic pleasure patients obtained from engaging in the activities. A caveat here is to ensure that the patient's inventory of past and current recreational interests is taken seriously in planning the recreational task.

Figure 5. Displacement of hallucinatory speech behavior with recreational therapy



A chronic schizophrenic male with high frequency hallucinations marked by self-vocalizations was exposed to recreational materials and activities with ("independent activity") and without ("baseline") supervision by a therapist who provided prompts and reinforcement for task engagement during the supervised periods.

Conclusion

Over the past 15 years, more than 50 studies have been published on social skills training with psychiatric patients (for reviews, see Wallace et al. 1980; Brady 1984). These studies provided the first evidence that social skills training was a feasible treatment strategy, and laid the empirical foundation for recent innovations in training techniques. Many of the early studies suffered from methodological shortcomings, including lack of (1) diagnostic assessment, (2) specifying concomitant psychotropic medications, and (3) widely accepted outcome measures, such as symptomatology or relapse rate.

The results of these studies can be summarized by three conclusions:

1. Psychiatric patients can be trained in behaviors that will improve their social skills in specific interpersonal situations.

2. Patients show moderate to substantial generalization of trained behaviors to untrained scenes and items (Goldsmith and McFall 1975; Kelly, Urey, and Patterson 1980; Liberman et al. 1984). The problem of behavioral generalization to different and novel situations appears to be greater for complex behaviors (e.g., requests for behavior change) than simple behavior such as eye contact. This presents a special problem for social skills training with chronic mental patients living in the community, for whom complex social behaviors may be necessary to use accessible resources and generate social support.

3. Comprehensive, intensive social skills training can reduce clinical symptoms and relapse in psychiatric patients. Among neuroleptic-stabilized schizophrenic inpatients or outpatients, intensive social skills training significantly lowered symptoms and delayed relapse (Liberman, Falloon, and Wallace 1984; Wallace and Liberman 1985; Hogarty et al., in press). Similarly, schizophrenic patients who participated in a day hospital program and concurrently received social skills training showed symptom reductions that were more durable over a 6-month followup period than patients who were in the day program but did not receive social skills training (Bellack et al. 1984). Social skills training alleviates depression for unmedicated depressed outpatients (McKnight et al. 1984), has clinical effects equivalent to those of antidepressant medication, and is associated with a lower rate of dropout from treatment (Bellack, Hersen, and Himmeloch 1983).

Many psychiatric patients manifest learning disabilities that require highly directive behavioral techniques for teaching social skills. Chronic patients often have information-processing and attentional deficits, and show hyperarousal or hypoarousal during psychophysiological testing. These patients may experience overstimulation from emotional stressors or even from therapy sessions that are not adequately structured and modulated. Chronic patients often fail to be motivated by customary forms of social and tangible rewards available in traditional therapy. The acquisition of other social skills is impaired in many patients by their lack of conversational skills, an important building block to the attainment of social competence. Patients with schizophrenia often have deficiencies in their social perception and their ability to generate response alternatives for coping with everyday problems such as making an appointment or getting help with annoying drug effects. Patients with verbal dysfluencies, minimal vocal intonation, and poor eye contact may be further impaired in their social learning. The wide variation in cognitive impairment and social deficits among psychiatric patients points to the importance of tailoring social skills training procedures to the needs of individual patients.

The aim of rehabilitation is not to cure disease, but to enhance a deficient individual's functioning, level of adaptation, and quality of life. The necessary design and validation of fully effective technology for skills training in social, family, and vocational domains will require tools from a variety of disciplines—psychopharmacology, social and cognitive psychology, developmental psychology, behavior analysis, psychodynamics, systems theory, and environmental psychology. For example, it has been demonstrated that patients learn social problem-solving skills better when they are taking optimal doses of neuroleptics, when their families are involved in educational activities aimed at improving family emotional climates, when they are exposed to repeated practice with slow and gradual steps, when visual as well as auditory cues and instructions are used, when positive feedback and corrective feedback are liberally provided, when social modeling is provided that uses peers as well as therapists, and when skills training is personalized and overlearned.

Just as neuroleptic drugs require consistent and indefinite use to maintain their therapeutic and prophylactic effects, psychosocial treatments require consistency and long-term application. Remediating psychological and social deficits in patients with schizophrenic disorders involves the same persistent application of the environment as remedying the patient's neurochemical disturbances with medication.

Despite the best efforts at training social, vocational, family, and independent living skills, some patients with major mental disorders require a responsive and compensatory environment to allow them to function with a reasonable quality of life in the community. Designing supportive environments to compensate for the residual behavioral and social deficiencies of chronic mental patients can be viewed as akin to the manufacture of prosthetic and orthotic devices for the physically disabled. Just as a paraplegic individual depends on a wheelchair, hydraulic lifts, and sidewalk ramps to exercise independence, mentally disabled individuals require self-help social clubs, transitional employment programs, and sheltered workshops.

References

Anthony, W.A. A rehabilitation model for rehabilitating the psychiatrically disabled. *Rehabilitation Counseling Bulletin*, 24:6–21, 1980.

Anthony, W.A.; Cohen, M.R.; and Vitalo, R. The measurement of rehabilitation outcome. *Schizophrenia Bulletin*, 4:365–383, 1978.

Azrin, N.H., and Besalel, V.A. *Job Club Counselors Manual: A Behavioral Approach to Vocational Counseling*. Baltimore: University Park Press, 1980.

Azrin, N.H., and Phillip, R.A. The job club method for the job handicapped: A comparative outcome study. *Rehabilitation Counseling Bulletin*, 23:144–155, 1979.

Bandura, A. Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84:191–215, 1977.

Beard, J.J.; Malamud, T.J.; and Rossman, E. Psychiatric rehabilitation and long-term rehospitalization rates. *Schizophrenia Bulletin*, 4:622–635, 1978.

Bellack, A.S.; Hersen, M.; and Himmelhoch, J.M. A comparison of social skills training, pharmacotherapy and psychotherapy for depression. *Behaviour Research and Therapy*, 21:101–107, 1983.

Bellack, A.S.; Turner, S.M.; Hersen, M.; and Luber, R.F. An examination of the efficacy of social skills training for chronic schizophrenic patients. *Hospital & Community Psychiatry*, 35:1023–1028, 1984.

Brady, J.P. Social skills training for psychiatric patients. *American Journal of Psychiatry*, 141:491–498, 1984.

Brenner, H.D. Zur bedeutung von basisstorungen für behand lung und rehabilitation. In: Böker, W., and Brenner, H.D., eds. *Bewaltigung der Schizophrenie.* Bern: Huber, 1986. pp. 142-158.

Edelstein, B.A.; Couture, E.; Cray, M.; Dickens, P.; and Lusebrink, N. Group training of problem solving with psychiatric patients. In: Upper, D., and Ross, S.M., eds. *Behavioral Group Therapy: An Annual Review.* Vol. 2. Champaign, IL: Research Press, 1980.

Fichter, M.; Wallace, C.J; Liberman, R.P.; and Davis, J.R. Improving social interaction in a chronic psychotic using nagging (discriminated avoidance): Experimental analysis and generalization. *Journal of Applied Behavior Analysis,* 9:377-386, 1976.

Foy, D.W.; Wallace, C.J.; and Liberman, R.P. Advances in social skills training for chronic mental patients. In: Craig, K.D., and McMahon, R.J., eds. *Advances in Clinical Behavior Therapy.* New York: Brunner/Mazel, Inc., 1983.

Glaser, R. Education and thinking: The role of knowledge. *American Psychologist,* 39:93-104, 1984.

Goldman, H.H.; Gatozzi, J.; and Taube, R. The national plan for the chronically mentally ill. *Hospital & Community Psychiatry,* 32:16-28, 1981.

Goldsmith, J.B., and McFall, R.M. Development and evaluation of an interpersonal skill-training program for psychiatric inpatients. *Journal of Abnormal Psychology,* 84:51-58, 1975.

Goldstein, A.P.; Sprafkin, R.P.; and Gershaw, M.J. *Skill Training for Community Living: Applying Structured Learning Therapy.* New York: Pergamon Press, 1976.

Goldstrom, I., and Manderscheid, R. The chronically mentally ill: A descriptive analysis from the uniform client data instrument. *Community Support Services Journal,* 2:4-9, 1982.

Hogarty, G.E.; Anderson, C.M.; Reiss, D.J.; Kornblith, S.J.; Greenwald, D.P.; Javna, C.J.; and Madonia, M.J. Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia. *Archives of General Psychiatry,* in press.

Jacobs, H.E.; Kardashian, S.; Kreinbring, R.K.; Ponder, R.; and Simpson, A.R. A skills-oriented model for facilitating employment among psychiatrically disabled persons. *Rehabilitation Counseling Bulletin,* 28:87-96, 1984.

Kale, R.J.; Kaye, J.H.; Wheland, P.A.; and Hopkins, B. The effects of reinforcement on the modification, maintenance and generalization of social responses of mental patients. *Journal of Applied Behavior Analysis,* 1:307-314, 1968.

Kane, J.M. Compliance issues in outpatient treatment. *Journal of Clinical Psychopharmacology,* 5:22S-27S, 1985.

Kelly, J.A.; Laughlin, C.; Claiborne, M.; and Patterson, J. A group procedure for teaching job interviewing skills to formerly hospitalized psychiatric patients. *Behavior Therapy,* 10:299-310, 1979.

Kelly, J.A.; Urey, J.R.; and Patterson, J.T. Improving heterosocial conversational skills of male psychiatric patients through a small group training procedure. *Behavior Therapy,* 11:79-83, 1980.

Kohen, W., and Paul, G.L. Current trends and recommended changes in extended care placement of mental patients. *Schizophrenia Bulletin,* 2:575-594, 1976.

Lehman, A.F.; Ward, N.C.; and Linn, L.S. Chronic mental patients: The quality of life issue. *American Journal of Psychiatry,* 134:1271-1276, 1982.

Liberman, R.P. Psychosocial therapies for schizophrenia. In: Kaplan, H.I., and Sadock, B.J., eds. *Comprehensive Textbook of Psychiatry.* Baltimore: Williams & Wilkins, 1985. pp. 724-734.

Liberman, R.P.; DeRisi, W.J.; King, L.W.; and Wood, D. Behavioral measurement in a community mental health center. In: Davidson, P.; Clark, F.; and Hamerlynck, C., eds. *Evaluating Behavioral Programs in Community, Residential and Educational Settings.* Champaign, IL: Research Press, 1974. pp. 103-139.

Liberman, R.P., and Evans, C.C. Behavioral rehabilitation for chronic mental patients. *Journal of Clinical Psychopharmacology,* 5:8S-14S, 1985.

Liberman, R.P.; Falloon, I.R.H.; and Wallace, C.J. Drug-psychosocial interventions in the treatment of schizophrenia. In: Mirabi, M., ed. *The Chronically Mentally Ill: Research and Services.* New York: SP Medical & Scientific Books, 1984. pp. 175-212.

Liberman, R.P., and Foy, D.W. Psychiatric rehabilitation for chronic mental patients. *Psychiatric Annals,* 13:539-545, 1983.

Liberman, R.P.; King, L.W.; and DeRisi, W.J. Behavior analysis and therapy in community mental health. In: Leitenberg, H., ed. *Handbook of Behavior Analysis and Modification.* Englewood Cliffs, NJ: Prentice Hall, 1976. pp. 566-603.

Liberman, R.P.; King, L.W.; DeRisi, W.J.; and McCann, M. *Personal*

Effectiveness: Guiding People to Assert Themselves and Improve Their Social Skills. Champaign, IL: Research Press, 1975.

Liberman, R.P.; Kuehnel, T.G.; Phipps, C.C.; and Cardin, V.A. *Resource Book for Psychiatric Rehabilitation: Elements of Service for the Mentally Ill.* Los Angeles: University of California Press, 1985.

Liberman, R.P.; Lillie, F.J.; Falloon, I.R.H.; Harpin, E.J.; Hutchinson, W.; and Stoute, B.A. Social skills training for relapsing schizophrenics: An experimental analysis. *Behavior Modification,* 8:155–179, 1984.

Liberman, R.P.; Nuechterlein, K.H.; and Wallace, C.J. Social skills training and the nature of schizophrenia. In: Curran, J.P., and Monti, P.M., eds. *Social Skills Training: A Practical Handbook for Assessment and Treatment.* New York: Guilford Press, 1982. pp. 5–56.

Martinez-Diaz, J.A.; Massel, H.K.; Wong, S.E.; Wiegand, W.; Bowen, L.; Edelstein, B.A.; Marshall, R.D.; and Liberman, R.P. "Training of Generalization of Conversation Skills in Chronic Schizophrenics." Presented at the World Congress on Behavior Therapy, Washington, DC, December 1983.

McKnight, D.L.; Nelson, R.O.; Hayes, S.C.; and Jarrett, R.B. Importance of treating individually assessed response classes in the amelioration of depression. *Behavior Therapy,* 15:315–335, 1984.

Paul, G.L. The chronic mental patient: Current status, future directions. *Psychological Bulletin,* 71:81–94, 1969.

Paul, G.L., and Lentz, R. *Psychosocial Treatment of Chronic Mental Patients.* Cambridge, MA: Harvard University Press, 1977.

Rosen, A.J.; Sussman, S.; Mueser, K.T.; Lyons, J.S.; and Davis, J.M. Behavioral assessment of psychiatric inpatients and normal controls across different environmental contexts. *Journal of Behavioral Assessment,* 3:25–36, 1986.

Rumelhart, D.E. *Understanding Understanding.* La Jolla, CA: University of California, Center for Human Information Processing, 1981.

Schooler, N.R. The efficacy of antipsychotic drugs and family therapies in the maintenance treatment of schizophrenia. *Journal of Clinical Psychopharmacology,* 6:11S–19S, 1986.

Spaulding, W.; Storms, L.; Goodrich, V.; and Sullivan, M. Applications of experimental psychopathology to psychiatric rehabilitation. *Schizophrenia Bulletin,* 12:560–577, 1986.

Stein, L.I., and Test, M.A. *Alternatives to Mental Hospital Treatment.* New York: Plenum Press, Inc., 1978.

Stokes, T.F., and Baer, D.M. An implicit technology of generalization. *Journal of Applied Behavior Analysis,* 10:349–369, 1977.

Talbott, J.A. *The Chronic Mental Patient: Problems, Solutions, and Recommendations for a Public Policy.* Washington, DC: The American Psychiatric Association, 1978.

Van Putten, T. Why do schizophrenic patients refuse to take their drugs? *Archives of General Psychiatry,* 31:67–72, 1974.

Wallace, C.J.; Boone, S.E.; Donahoe, C.P.; and Foy, D.W. Psychosocial rehabilitation for the chronic mentally disabled: Social and independent living skills training. In: Barlow, D., ed. *Behavioral Treatment of Adult Disorders.* New York: Guilford Press, 1985. pp. 462–501.

Wallace, C.J., and Davis, J.R. The effects of information and reinforcement on the conversational behavior of chronic psychiatric patient dyads. *Journal of Consulting and Clinical Psychology,* 42:656–666, 1974.

Wallace, C.J., and Liberman, R.P. Social skills training for patients with schizophrenia: A controlled clinical trial. *Psychiatry Research,* 15:239–247, 1985.

Wallace, C.J.; Nelson, C.J.; Liberman, R.P.; Aitchinson, R.H.; Lukoff, D.; Elder, J.P.; and Ferris, C. A review and critique of social skills training with schizophrenic patients. *Schizophrenia Bulletin,* 6:42–63, 1980.

Wong, S.E.; Massel, H.K.; Mosk, M.D.; and Liberman, R.P. Behavioral approaches to the treatment of schizophrenia. In: Burrows, G.D.; Norman, T.R.; and Rubinstein, G., eds. *Handbook of Studies on Schizophrenia.* Amsterdam: Elsevier Science Publishers, 1986. pp. 239–247.

Wong, S.E.; Terranova, M.D.; Marshall, B.D.; Banzett, L.K.; and Liberman, R.P. "Reducing Bizarre Stereotypic Behavior in Chronic Psychiatric Patients: Effects of Supervised and Independent Recreational Activities." Presented at the Ninth Annual Convention of the Association for Behavior Analysis, Milwaukee, WI, May 1983.

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Announcement

The **Hancock House Center for Movement Arts and Therapies** and the **University of Wisconsin Arts Extension**, Madison, Wisconsin, are cosponsoring a weekend symposium entitled "**Psychoanalytic Approaches to Dance/Movement Therapy.**" The event is scheduled for **April 4-5, 1987**, at the Wisconsin Center, Madison, Wisconsin. Three major dance/movement therapists have developed astute and innovative theories of interpretation and intervention that they will present. The presenters are Penny L. Bernstein, Ph.D., A.D.T.R. (Jungian); Liljan Espenak, M.A., A.D.T.R. (Adlerian); and Elaine V. Siegel, Ph.D., A.D.T.R. (Freudian). The symposium will enable participants to study with each of these presenters, and to hear them discuss their work with other well-known verbal psychotherapists in a panel chaired by Jarl Dyrud, a psychiatrist practicing in Chicago.

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Social Learning for Chronic Mental Inpatients

by Shirley Glynn and Kim T. Mueser

Abstract

With chronic institutionalized psychiatric patients, an intensive social learning program resulted in greater increases in adaptive functioning, reductions in bizarre behavior, less prescribed medication, and over 98 percent of patients being successfully discharged into the community. The social-learning program was more cost-effective than custodial hospital care, and nonprofessional staff were able to apply the treatment with a high degree of competence. Especially effective elements of the social-learning program included (1) integrated procedures emphasizing the acquisition of patient skills and the reduction of bizarre, inappropriate behavior; (2) a token economy structure; and (3) consistent pacing and generalization training. More recent research in the area has sought to identify the remediative aspects of social-learning programs, to specify patient variables that are related to improvement in a token economy, and to offer patients more specialized interventions (e.g., social skills training) in conjunction with a standard token economy. With the demonstrated and operationalized efficacy of social-learning procedures, the rehabilitation of chronic psychiatric patients has become more feasible.

The rehabilitation of long-term institutionalized psychiatric patients poses an ongoing challenge to mental health practitioners. Almost 10 years after its publication, the comparative study of a social-learning program for chronic psychiatric patients reported by Paul and Lentz (1977) remains unmatched in both its comprehensiveness and methodological rigor (see Rhoades 1981). Social-learning procedures appear to be the most cost-effective approach to meeting the treatment needs of low-functioning, institutionalized psychiatric patients. The inadequacy of existing services for the chronically mentally ill is reflected in at least three disturbing facts: (1) many psychiatric patients are unable to maintain community tenure; (2) even "rehabilitated" chronic patients often have severe deficits in self-care and social skills; and (3) patients in traditional institutional settings are understimulated and frequently lead barren lives.

Rehospitalization rates for psychiatric patients range from 35 to 50 percent within the first year postdischarge (e.g., Friedman, Von Mering, and Hinko 1966; Wolkon, Karman, and Tanaka 1968). About 60 percent of public psychiatric beds are occupied by patients who have been continuously hospitalized for more than 1 year, and as many as 90 percent of these patients are unresponsive to pharmacological treatment (Paul 1984). However, even the ability to live in the community is not a guarantee of satisfactory social functioning. Psychiatric patients frequently reside in dismal social conditions in the community (Kirk and Therrien 1975), and the functioning of many chronic patients living in community residential settings actually declines over time (Kohen and Paul 1976; Dickey et al. 1981; Spivack et al. 1982).

The inadequacy of chronic patients' social skills may limit their ability to cope with and blunt the noxious effects of stressors, making them more vulnerable to symptom exacerbations (Liberman 1982). While social deficits of patients in the community lead to social rejection

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and a higher risk of rehospitalization, they are also indicative of the impoverished quality of their lives (Lehman, Ward, and Linn 1982; Lehman 1983). In fact, social competence is the best single predictor of successful community tenure following discharge from psychiatric facilities (Paul, in press a). Treatment programs that strive to remediate the social deficits of chronic patients may be expected to improve their clinical condition, social adjustment, and potential for community tenure.

Just as overstimulating, stressful environments worsen the symptoms of many psychiatric patients, understimulating and unstructured environments also promote maladaptive behavior (Wing and Freudenberg 1961; Rosen et al. 1981; Wong et al. 1985). Many programs for the chronically ill include few structured activities and minimal patient-staff contact. Patient-staff interaction in State and VA hospitals accounts for less than 11 percent of patient waking hours, and less than 15 percent of staff working hours (Paul 1986*a*). Increased interaction of appropriate types and levels between patients and staff in structured or semistructured environments could lessen inappropriate behaviors that present obstacles to community survival (Mechanic 1969; Paul 1986*b*).

Thus, the high proportion of chronic psychiatric patients with severe social deficits who reside in understimulating environments that maintain, rather than rehabilitate, suggests the need for programs that actively seek to improve these patients' skills competency (Bellack and Mueser, in press). The social-learning program of Paul and Lentz (1977, in preparation), because of its demonstrated efficacy and relevance to community as well as hospital-based programs for disabled chronic mental patients, deserves a renewed focus and descriptive evaluation.

Psychosocial Programs

The study of Paul and Lentz (1977) compared traditional hospital treatment and two comprehensive psychosocial treatment programs for psychiatric inpatients that actively sought to enhance patients' functional independence: a social-learning program, based on learning principles such as associative learning, problem solving, reinforcement, modeling, punishment, and shaping; and a milieu therapy program based on communication theory and the therapeutic community model that promotes prosocial behavior through communicating attainable expectations, stimulating patient involvement, and group cohesiveness. Both psychosocial programs aimed at resocializing patients by developing self-maintenance and interpersonal skills; decreasing bizarre, inappropriate behaviors; and facilitating discharge and maintenance in the community. Successful community tenure was defined as a spectrum of levels of functioning including independent living, residence in board-and-care homes, and gainful or sheltered employment. Declining-contact aftercare in the community was an important treatment component that was provided to patients discharged from traditional hospital wards as well as to those discharged from the psychosocial units. The declining-contact aftercare consultation was provided directly to discharged patients and their significant others for those living independently and to the facilities for those living in community placements.

The comparative study was conducted with chronically institutionalized, severely debilitated, aggressive, and psychotic psychiatric patients. The specific, comprehensive social-learning and milieu programs that were developed and evaluated employed the same clinical staff members, who rotated between the two programs on a daily basis to equate time and exposure. These programs engaged patients in structured therapeutic and training activities much of their waking hours (85 percent), unlike the traditional hospital group (5 percent). The psychosocial programs were conducted concurrently for 4½ years with a minimum 18-month declining-contact followup while the control hospital program continued "as usual" with monitoring of patients for 6 years.

Patients and Staff. The patients were 18 to 55 years old, equally divided in gender, and had been residing continuously for at least 2 years in one of four State hospitals in Illinois. The great majority of patients were divorced or had never been married, and all were low in socioeconomic status. All had diagnoses of chronic "process" schizophrenia and had few or no friends or relatives in the community. Overall, the patient group had spent an average of 17 years—about two-thirds of their adult lives—in psychiatric hospitals. As a group, the patients demonstrated higher levels of inappropriate, "crazy" behavior and greater deficits in self-care, interpersonal, and communication skills than any previous psychiatric group that had been subject to programmatic study. This population had received many prior somatic treatments and all had received chemotherapy, although they were clearly "hard-core" chronic mental patients for whom neuroleptic medications are largely not beneficial (Paul, Tobias, and Holly 1972; Gardos and Cole 1976). Initially, three patient groups of 28 each were equated on 16 variables and randomly assigned to the three treatment programs. "New" patients were admitted from the original chronic population to maintain 28 inpatients in each program with equation at the entry assessment for additional patients as well.

The clinical staff who implemented the social-learning and milieu programs were 20 percent professionals (e.g., psychologists, social workers, nurses, and occupational therapists) and 80 percent nonprofessionals (aide-level with a high school degree). The staffing level and staff-patient ratios were purposely equal to those in the traditional hospital treatment, and all were considerably lower than the average staffing ratios in public mental hospitals nationwide.

Treatment Setting. The two inpatient psychosocial programs were located in identical adjacent 28-bed units in a regional mental health center. These units were coeducational, and had been designed to facilitate staff-patient interaction. Unique features of the units included a kitchen, bedrooms that varied from six-bed dormitory-type rooms to private ones, a living room, a TV room, a classroom-lounge, and a sunken "activity pit" in the center of an open area of the ward. Administrative structure and patient care services were totally decentralized to treatment units.

The regional State hospital that served as the control inpatient treatment housed approximately 1,000 residents, and contained a wide variety of departments, treatment facilities, and support services, including occupational, industrial, and educational programs as well as recreational and art facilities. The 28 comparison beds and patients were located on wards that were segregated by sex, and varied in size from 16 to 84 beds. The declining-contact aftercare for discharged patients from both center programs and the hospital comparison group was provided by the psychosocial staff, who traveled to the community residence of ex-patients for on-site consultations.

The social-learning and milieu therapy programs shared several features and basic aims. Both inpatient psychosocial programs used a level system where residents promoted to higher steps were gradually exposed to less structured situations, earned greater privileges, and carried added responsibility. Both programs covered all aspects of patients' lives and functioning with consistent interactional-educational conceptual models, including integrated principles, procedures, and assessments 24 hours per day, 7 days per week. Participants in the programs were referred to as "residents" rather than "patients," and an emphasis was placed on the use of descriptive, action-oriented language. A list of 10 rules (see table 1) was conspicuously posted around both units to cue the residents to attend to the expected norms of behavior. Patient schedules included individual times and places for assessment and training of specified aspects of cognitive, social, and instrumental functioning. The programs stressed the importance of residents acquiring desirable intrapersonal, interpersonal, and instrumental aspects of functioning through continuous assessment of each resident's needs and progress, modeling appropriate social interactions, teaching vocational, housekeeping, communication, problem-solving, coping, and self-care skills, gradually reacquainting the residents with the outside world, and programming for generalization into the community. Both psychosocial programs were "high technology" in the sense of requiring very precise specification of appropriate staff interactions with patients demon-

Table 1. Psychosocial program rules

As a resident of the rehabilitation unit, you are expected to:

1. Care for yourself and present a desirable appearance
2. Perform your own housekeeping chores and share in those of the unit
3. Think, talk, and act straight (in ways that make sense to others)
4. Show gentlemanly or ladylike behavior
5. Show respect for the rights of others, for yourself, and for your property
6. Interact cooperatively and actively with residents, staff, and others
7. Attend and participate in all scheduled activities (be where you're supposed to be)
8. Acquire and demonstrate work habits and skills that will provide for an income after you leave
9. Move through the 4 step levels of the program and return permanently to the outside community
10. *Not* act "crazy"!

From Paul, G., and Lentz, R. *Psychosocial Treatment of Chronic Mental Patients: Milieu vs. Social-Learning Programs*. Cambridge, MA: Harvard University Press, 1977. p. 47.

strating a variety of behaviors in a number of settings. What discriminated between the social-learning and milieu therapy inpatient programs was the specific nature and content of staff/patient interactions.

Social-Learning Inpatient Program. The social-learning inpatient program was based on a token economy motivational structure. Residents in the first three step levels earned tokens (colored strips of plastic) redeemable for goods or privileges contingent upon engaging in appropriate behaviors. Consistent verbal and nonverbal staff-patient interactions were paired with token exchanges. As patients progressed to higher steps, the number of tokens they could earn grew, as did the availability of increasingly more desirable privileges. For example, tokens could be earned by attending and participating in meetings and classes, engaging in appropriate informal conversations, cleaning the unit, good mealtime behavior, maintaining one's personal appearance, and working in vocational training positions; tokens could be spent on more pleasant sleeping quarters, passes, privacy time, food, appointments with staff members, and promotion to a higher step. Throughout the program, the particular performances necessary to obtain a set number of tokens were clearly specified in objective, readily observable terms for each patient, as were the privileges for which the tokens could be exchanged. As patients were promoted, gradual delays of reinforcement were introduced to enhance internal planning and control.

Guidelines governing the operation of the social-learning program were derived from two basic learning principles: the *law of effect*, and the *law of association by contiguity*. The law of effect was applied in the social-learning program by arranging for desired behavior to be followed by social and material *positive reinforcement* or by the removal of *negative reinforcers*, and for undesired behavior to be followed by either punishment (*aversion*), loss of positive reinforcers (*response cost*), or no consequence at all (*extinction*). The law of association by contiguity was incorporated into the program in many ways: to assure that a resident's desirable or undesirable behavior was met immediately with the appropriate consequence; to develop new affective and cognitive learning and problem-solving skills; to establish social stimuli as positive or reinforcing events; and to shift or generalize new skills and coping responses from dependence on tokens to internal and verbal controls.

In addition to these laws, other learning principles were systematically integrated in the social-learning program to facilitate the acquisition of appropriate behavior, including *shaping* (reinforcing successive behavioral approximations to a criterion), *chaining* (teaching a new behavior by systematically linking components in a sequence), and *positive and negative prompts* (instructing desired performance through physical guidance, demonstration and modeling, and written or verbal means, along with verbal specification of resulting consequences). Direct verbal instruction and modeling were also incorporated in classes and therapy groups to provide new knowledge and skills.

Milieu Therapy Inpatient Program. The milieu program was based on a therapeutic community structure wherein all patients were members of 9- to 10-person "living groups" which were assigned the tasks of identifying problems and promoting change among individual members. Unit tasks such as meal preparation were assigned by living groups, and attempts were made to keep group members on similar daily schedules for training classes and activities. Members were also encouraged to spend free time and passes together. The major vehicle for promoting change was the social and group pressure exerted by each living group on its members. All patients and staff were also members of the unit-wide community group which, through discussion and majority vote, dealt with any decisions affecting the entire community, while an executive council with elected members from each living group was responsible for agendas.

The milieu treatment program was based on three laws: *expectancy*, *involvement*, and *group cohesiveness*. The *law of expectancy* was operationalized by the staff's use of *positive statements* that specified and encouraged performance of desirable behavior before it occurred, *positive feedback* that verbally acknowledged a recent desirable performance and/or the value of the person, and *negative feedback* that acknowledged and discouraged a recent undesirable act or verbalization. Clarification and interpretation of metacommunicative aspects of behavior were important. Since the use of *negative statements* could communicate to the resident an expectation that he or she would engage in an undesirable behavior, these were not used in either treatment program. Unlike the social-learning program, staff applied no nonsocial or material consequences whatsoever (except for "expelling" patients from the group to seclusion as a consequence for assaults) and contingent social consequences occurred only incidentally when positive feedback coincided with a particular patient performance.

On the basis of the *law of involvement*, residents in the milieu program always assumed the maximum responsibility for making decisions and implementing them, frequently practiced problem solving, and were encouraged to engage in behaviors in which they had previously experienced failure. *Group cohesion* was enhanced by the entire community and living-group structure, encouraging an "us" and "them" attitude within the groups, and the endorsement of group pressure as a vehicle for change in group members. Milieu therapy patients attended identical classes as social-learning patients and received verbal instruction and modeling to provide new knowledge and skills; however, classes were assigned by living groups rather than by individuals.

Community Aftercare Component. The inpatient psychosocial programs focused on preparing patients for community functioning by establishing minimal levels of competence in self-care, interpersonal, communications, and instrumental role skills, and by reducing or eliminating psychotic manifestations and other extremely bizarre behavior. The inpatient programs also attempted to bridge the gap from residential treatment to community living by training for generalization to natural support systems and establishing living arrangements, training or vocational positions, and at least one supportive friend or relative in the community before discharge. An aftercare program involving scheduled contacts in the community on a declining basis by trained psychosocial staff was an equally important component to ensure generalization of inpatient gains and maintenance of functioning and community tenure. Early on, it became clear that social-learning principles were the most promising on which to base aftercare consultations; consequently, patients discharged from all three inpatient programs were provided with declining-contact followup employing social-learning principles and technology. At least 4 weekly, 5 biweekly, and 3 monthly contacts followed by semiannual contacts were scheduled, with additional contacts as needed. Declining-contact aftercare was provided for at least 2 years to each patient discharged to independent functioning and self-support, and for 18 months to board-and-care facilities that accepted community placements from any of the three inpatient programs.

Assessment and Evaluation Procedures

A comprehensive set of assessment and evaluation procedures is necessary to ensure coverage of patient, staff, and program functioning, as well as to evaluate the impact of interventions in residential and community treatment (see Paul and Mariotto 1986). In the comparative study, a battery of standardized rating instruments and structured interviews was obtained on all patients in all three treatment groups before and after entry to study programs, every 6 months thereafter during inpatient treatment, immediately before discharge, and every 6 months after discharge. Ongoing reliability checks were obtained for each measure. Along with demographic variables, these measures served to equate treatment groups on initial patient entry and to sample levels of adjustment during and after inpatient treatment on common instruments at identical time periods. Measures of staff attitudes, opinions, and personality characteristics were similarly obtained on all clinical staff.

In addition to the above assessments, three direct observational coding systems were continuously employed in both inpatient psychosocial programs. The *Clinical Frequencies Recording System (CFRS)* (see Paul and Licht, in press *a*; Paul and Shelite, in preparation) was an integral part of the application of both comprehensive social-learning and milieu programs. The CFRS consists of a set of standardized time-place-situation specific forms completed by clinical staff concurrent with therapeutic activities. CFRS forms provide 100 percent event-recording of low-frequency critical acts (e.g., assaults and other intolerable behavior) and of setting-dependent aspects of patient functioning (e.g., on time, on task, progress toward normal appearance) as well as staff responses to each patient's behavior. Higher-order CFRS scores combine recordings over forms and weekly periods to provide objective data for program evaluation on the rate with which different classes of behavior were performed. The detailed daily records ensured consistent identification and monitoring of each patient's level of achievement, subtargets for clinical programming, and consistency of staff response.

Two multivariate direct observational systems employed by a small cadre of independent noninteractive observers provided continuous objective assessment of staff and patients in both psychosocial programs through stratified hourly time sampling of all patient waking hours for the entire 4½-year inpatient phase. Full-week samples of resident and staff functioning in community board-and-care facilities were also obtained every 3 months during the followup period. The *Time-Sample Behavioral Checklist (TSBC)* (see Paul, in press *a*; Paul and Licht, in press *b*) records the context of each observation as well as the presence/absence of 69 specific codes within 7 content categories: Location, Position, Awake-Asleep, Facial Expression, Social Orientation, Appropriate Concurrent Activities, and Crazy Behaviors. Weekly summarization of TSBC data provides objective rates of occurrence for each individual code as well as higher-order components of functioning, both appropriate (e.g., Interpersonal Interaction, Total Appropriate Behavior) and inappropriate (e.g., Cognitive Distortion, Total Inappropriate Behavior) for each patient and treatment unit as well as documenting how and where time is spent.

The *Staff-Resident Interaction Chronograph (SRIC)* (see also Paul, in press *b*; Licht and Paul, in press) records the context of each observation, and the nature, content, and frequency of 21 classes of staff verbal and nonverbal response to four classes of resident functioning (Appropriate, Inappropriate Failure, Inappropriate Crazy, "Straight" Requests) or of staff-initiated interactive or noninteractive activity (Resident-"Neutral"). Weekly summarization of SRIC data provides objective rates of occurrence for each cell of the 5 (resident behavior) × 21 (staff behavior) matrix, and higher-order scores reflecting total staff activity, total staff-resident interactions, actual resident/staff ratios and attention received by all residents in a program to document the psychosocial treatment provided. SRIC summaries for individual staff or staff groups were also used for monitoring and feedback. TSBC/SRIC observers were trained to 100 percent accuracy on both instruments, and the obtained data were highly reliable.

Rehabilitation Outcomes

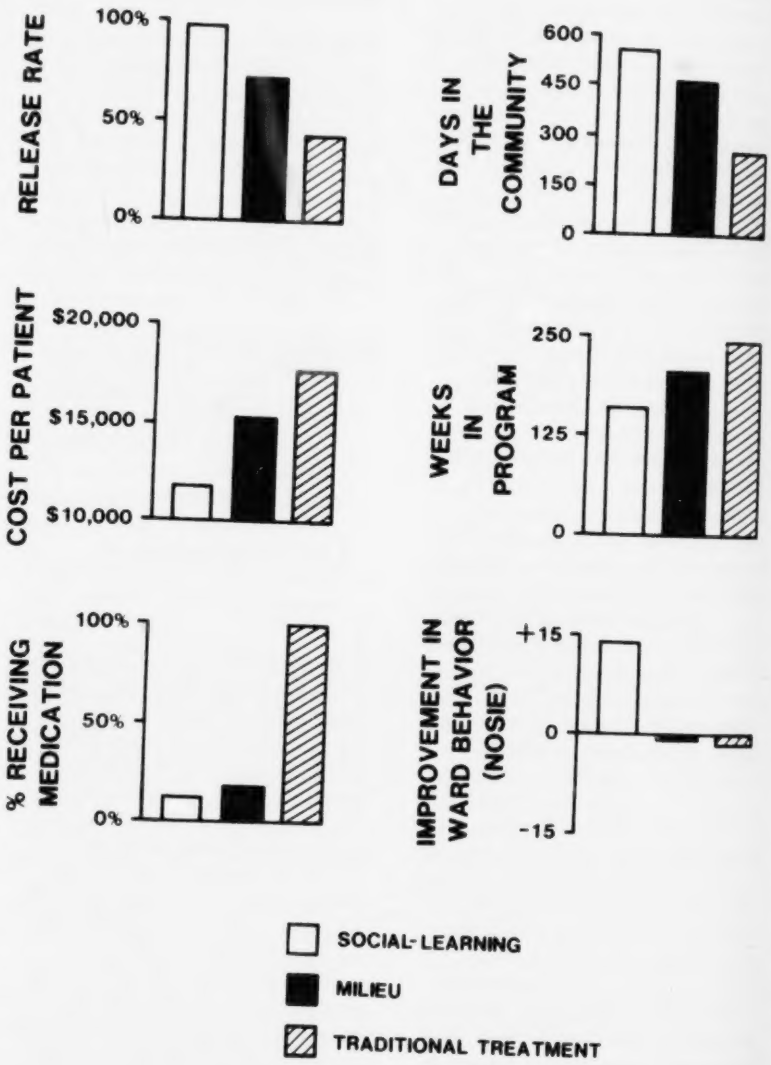
Because the patients who participated in this study exhibited such bizarre behaviors and deficits in adaptive functioning, the relative efficacy of the social-learning and milieu programs compared to traditional hospital treatment needs to be evaluated with regard to three practical goals that are critical for such a disabled population:

- Increasing patients' adaptive functioning (e.g., self-care and social skills) and decreasing patients' psychotic functioning and extremely bizarre behavior.
- Reducing the psychotropic medications necessary to stabilize patients.
- Facilitating discharge and successful maintenance in the community.

In all three domains, milieu therapy resulted in greater gains than seen in the hospital comparison group, and the social-learning program yielded strikingly better results than the milieu treatment. Furthermore, the improvement in social-learning patients was not related to any individual characteristics, pretreatment level of functioning, or diagnosis, and was obtained in the relative absence of psychotropic medication. The structured, comprehensive, and integrated procedures of the social-learning program led to the development of personal and interpersonal skills which were remarkable on an absolute level for this chronic group, the reduction of idiosyncratic and "crazy" motor and verbal behavior to fractions of initial levels, a radical decrease in the number of patients prescribed psychotropic medications, and to successful discharge or community placement without rehospitalization for the vast majority of patients.

The social-learning program treated nearly 30 percent more patients than either the milieu or hospital programs with essentially identical resources. Consequently, it was three to four times as cost-effective as traditional programs and is thus also the treatment of choice on purely economic grounds. The explicit consequences within the comprehensive, constructive program resulted in social-learning patients receiving fewer aversive or intrusive events than other inpatients, with the program ensuring that least-restrictive or intrusive procedures were invoked; thus, it is also the treatment of choice based on ethical and humanitarian concerns. The specificity of procedures and precision of feedback provided by the integrated observational assessment systems allowed both professional and nonprofessional staff to be trained to exceptional levels of competency in implementing the program. This resulted in nearly errorless performance in the ongoing conduct of the comprehensive procedures—it was accessible to and accepted by the levels of staff already employed in the usual public psychiatric hospital. Figure 1 illustrates the outcomes for the three treatment groups, including release rate, days in the community, weeks in the treatment program, percent on psychotropic medications, and changes on a common ward rating scale, the Nurses Observation Scale for Inpatient Evaluation (NOSIE-30) (Honigfeld 1966). Declining-contact aftercare for discharged patients was successful in maintaining community tenure in more than 97 percent of cases, indicating unquestioned superiority of the social-learning program over the milieu and hospital comparison groups.

Figure 1. Practical outcomes of the long-term comparative study of Paul and Lentz



Note.—On all 6 dimensions, the social-learning program achieved results better than the results of the milieu program, which were better than those of the standard treatment comparison.

Source: Paul, G., and Lentz, R. *Psychosocial Treatment of Chronic Mental Patients: Milieu vs. Social-Learning Programs*. Cambridge, MA: Harvard University Press, 1977.

Increasing Adaptive Functioning and Decreasing Bizarre Behavior. Outcomes could not be clearer regarding the absolute efficacy of the social-learning program in achieving the goals of increasing patients' adaptive functioning and decreasing patients' psychotic functioning and extremely bizarre behavior, as well as its relative superiority to the milieu program conducted by the same staff. The detailed ongoing assessment of patient functioning with the TSBC and CFRS found the milieu program to produce significant reductions in all classes of bizarre and inappropriate behavior except for dangerous and aggressive acts, for which milieu procedures were ineffective. However, the social-learning program was even more effective in reducing excesses in bizarre and psychotic behavior, with all components of maladaptive functioning showing an average reduction from incidence at entry exceeding 60 percent. Comprehensive social-learning procedures were particularly effective in eliminating belligerence and dangerous and aggressive acts—producing an average reduction of 97 percent, reflecting complete elimination of such behavior for all but one patient, whose physical condition precluded participation in those procedures known to be effective. The only area of maladaptive functioning in which the milieu therapy program was ultimately as effective as the social-learning program was in the reduction of bizarre verbal statements and inappropriate facial expressions indicative of thought disorder (e.g., delusions, hallucinations, incoherent speech)—even here, the social-learning program was more efficient, producing such reductions more rapidly and consistently.

Differential effectiveness of the social-learning program was even greater for increasing adaptive cognitive, social, and instrumental functioning. Whereas the milieu program only produced an average increase in overall competent functioning of about 25 percent above incidence at entry, the social-learning program was remarkably effective in overcoming the severe deficits in this chronic population—demonstrating improvements in overall competent functioning nearly 10 times the rate achieved by the milieu program. On an absolute level, the average social-learning patient increased interpersonal and communicative skills by over 1200 percent of their entry level.

Overall, the social-learning program produced significant improvement in functioning in every patient who was physically capable of participation, including those who had failed to improve in the milieu program. In fact, 25 percent of these severely disabled patients had improved to the point of being indistinguishable from the "normal" population. Sustained improvement in the milieu program was obtained for only 55 percent of the number improved in the social-learning program, while less than one-third that number showed any improvement in hospital programs. Further, no additional improvements were observed in milieu or hospital groups while nearly all classes of patient functioning continued to show improvement in the social-learning program until the time the inpatient units were closed. On the basis of the practical goals of increasing adaptive functioning and decreasing bizarre behavior, the social-learning program is the clear treatment of choice.

Reducing Psychotropic Medications. Since psychotropic medications frequently have deleterious side effects, and many institutionalized patients who are refractory to neuroleptic drugs received them on a "superstitious" basis, treatment programs that reduce the need for such medications are especially desirable. As might be expected given the chronic nature of the patients included in this study, the vast majority of all patients (91.7 percent) were receiving psychotropic medications at its inception. After 3½ years of operation of inpatient programs, however, 89 percent of the social-learning patients and 82 percent of the milieu patients were not prescribed such medications, and this low level was maintained through the end of the inpatient treatment phase. This low medication rate is particularly impressive in view of the fact that medications were held constant on psychosocial programs over the last year when significant improvements in functioning were obtained, and that *none* of the patients in the hospital comparison group were medication free at the end of the inpatient period. The reader will note, of course, that both alternative psychosocial programs yielded significant reductions in prescribed medications; by this criterion, both the social-learning and milieu programs were very effective, and the milieu program was nearly as effective as the social-learning program.

To assess whether psychotropic medications affected the clinical efficacy of either the social-learning or milieu treatments, Paul, Tobias, and Holly (1972) randomly assigned half of the patients in each group at pretreatment to placebo and half continued to receive neuroleptics. Physicians, staff members, and patients were blind to each patient's medication status for the following 17 weeks of the active program. The ward behavior of all patients improved over the study period, with patients in the social-learning group improving the most. After 17 weeks of the treatment, there were no differences between patients on neuroleptics and those receiving placebos. However, patients receiving neuroleptics improved at a slower rate during the first 2 months of treatment than patients receiving placebos, suggesting that medication may actually impede recovery in at least some psychiatric patients. This finding is consistent with a review of the literature by Gardos and Cole (1976) and Liberman, Falloon, and Wallace (1984) who concluded that as many as half of all medicated chronic schizophrenic outpatients were actually unresponsive to neuroleptics and would function just as well without medication, especially when involved in structured behavior therapy programs.

Facilitating Discharge and Community Tenure. A major goal in working with chronically institutionalized psychiatric patients is to improve functioning to a level enabling them to be successfully discharged and maintained in the community. On the basis of this criterion, the social-learning program was again established as significantly more successful than the milieu treatment and the traditional hospital comparison groups. Through the end of the common inpatient period, the social-learning program treated 40 patients, of whom 97.5 percent were successfully discharged with a minimum community stay of at least 90 days, as compared to 31 milieu patients with 71 percent discharged, and 29 hospital comparison patients with 44.8 percent discharged. Of the original equated groups, discharge to independent living and self-support without rehospitalization was achieved for 10.7 percent of social-learning patients, 7.1 percent of milieu patients, and none of the hospital patients—any such discharge being astounding for the severely disabled population. All other discharges were to community board-and-care facilities with, at best, employment in a sheltered workshop. Unfortunately, about 80 percent of the patients discharged to board-and-care facilities were readministered psychotropic drugs once they were in the facilities, independent of their nature or level of functioning. The investigators had hoped to extend the social-learning program to community aftercare facilities, but budgeting constraints and logistical difficulties precluded the full development of this plan and responsibility for programming had to be relinquished to the facilities. The declining-contact aftercare consultation was, however, conducted for discharges to independent living and to community board-and-care placements. These social-learning aftercare procedures were successful in maintaining community tenure for more than 97 percent of patients discharged from all three inpatient programs, without differential rehospitalization rates from the different inpatient units. Community tenure ranged from a minimum of 18 months to more than 5 years at the end of the followup period. Thus, all the evaluative goals—increasing adaptive functioning and decreasing psychotic and bizarre behavior, reducing psychotropic medications, and facilitating discharge and community tenure—identify the comprehensive social-learning program as the clear treatment of choice for the severely disabled, chronically institutionalized psychiatric patient.

Practical Acceptability of Innovations

As any practitioner can attest, treatment efficacy must be evaluated on many dimensions, including its acceptability and relevance in clinical settings. The most useful treatments must result in benefits for patients, of course, but they must also be cost-effective, ideologically acceptable, and manageable by staff. Even a surefire "cure" is of little value if it is too costly to purchase or no one can administer it! Once the success of the social-learning program in reducing symptoms and enhancing adaptive functioning among chronic mental patients is accepted, four important questions remain: (1) How much does the treatment cost? (2) Is it acceptable on ethical and humanitarian grounds? (3) Can it be delivered consistently in the face of external pressures? and (4) Is it comprehensible, manageable, and desired by the typical public hospital staff?

Cost-Effectiveness. Paul and Lentz (1977) report a detailed and conservative cost-effectiveness analysis of the comparative dollars spent and saved by the different treatment programs during the 6-year period—noting that only differential costs and savings have meaning without estimating the effects of inflation and discounting. Although staff-patient ratios were equal between psychosocial programs and the hospital comparison, staffing costs were slightly higher for the psychosocial programs as a result of including aftercare functions within inpatient staffing and a relatively greater proportion of psychologists (including Paul's consultant costs) in contrast to nurses in the psychosocial programs. However, the reduced use of psychotropic drugs in psychosocial programs resulted in lower medication costs. Thus, the daily cost of the social-learning program for staffing and medication combined was only 1.6 percent higher than that of the hospital program while the cost of the milieu program was only 2.0 percent higher in these regards. All other per diem costs of the social-learning program were equal to or less than those of the milieu program, the costs of which were in turn equal to or less than those of the hospital comparison.

The greater efficiency and effectiveness of the psychosocial programs in producing successful discharge and community tenure resulted in a considerable economic gain since aftercare is much less costly than continued hospitalization. Even when the cost side of the equation is limited to the differential expense of staff and drug costs, the social-learning program returned more than three times the dollar savings for the same dollars spent than did traditional programs during the project period, and 30 percent more than the milieu program when calculations include only the original equated groups. If the greater number of patients treated are considered, the social-learning program was nearly four times as cost-effective as the hospital comparison and 50 percent more cost-effective than the milieu program. Thus, purely on the basis of economic factors, both psychosocial programs were more effective than traditional hospital treatment, and the social-learning program again emerged as the treatment of choice.

Ethical and Humanitarian Concerns. The majority of social-learning procedures that are directed toward increasing patient motivation and decreasing inappropriate and bizarre behavior consist of positive reinforcement of adaptive competencies through application of both social and material consequences and constructive training in appropriate alternative behavior. However, the social-learning program also includes the use of primary reinforcement (e.g., food and sleep) as well as explicit behavior-contingent application of extinction, aversion, and response-cost procedures about which many commentators have ethical and humanitarian concerns.

Paul and Lentz (1977) provide a detailed analysis of all procedures within the social-learning program that could be considered aversive, restrictive, or intrusive by nearly any standard as well as comparative data on the frequency of such events in traditional hospital programs and in the milieu program, where the only specified negative consequence was for dangerous and aggressive acts. In the traditional hospital program, 20.9 percent of staff-patient interactions involved such negative components as compared to 12.3 percent within the milieu program and only 7.5 percent within the social-learning program. Further detailed analyses of the absolute level of deprivation, intrusions, or punishment found the social-learning program to be the most humanitarian of all inpatient programs in these regards. The presence of an integrated, constructive treatment program with many positive social and material consequences and a few explicit and consistent negative ones further increased the effectiveness of purely verbal procedures, even for terminating ongoing inappropriate behavior (e.g., such verbal procedures increased from a low of 94 percent effectiveness to 98 percent in the social-learning program as compared to the milieu program where verbal procedures started at 75 percent effectiveness and declined over time to only 55 percent). Thus, the comprehensiveness of the program with explicit criteria and contingency rules based on ongoing objective assessment ensured the least restrictive or intrusive means necessary for treatment of each patient nearly automatically. Consequently, the social-learning program also emerges as the treatment of choice on purely ethical and humanitarian grounds.

Treatment Consistency. Although social-learning treatment was clearly an effective intervention, its effectiveness was occasionally compromised by external events (e.g., a patient death, fiscal constraints, and hospital policy changes made at the State level). Each of these circumstances at least temporarily decreased improvements gained through behavioral programming. The most dramatic decrease occurred subsequent to a revision in Illinois State policy on the use of restrictive and aversive interventions. Previously, nursing staff had the option of placing highly assaultive and aggressive patients in seclusion for up to 48 hours, but the modified State policy limited the duration of this seclusion to a 2-hour maximum. A gradual increase in assaultive behavior followed in both psychosocial programs, with even nonassaultive patients demonstrating decreases in adaptive behavior and increases in inappropriate behavior. Fortunately, the new policy was eventually revised to allow longer duration in seclusion, and many treatment gains were reestablished.

While the circumstances surrounding this policy change may be specific to Illinois, the larger issues apply to all innovative treatment programs. They are uniquely sensitive to administrative fiats, budgetary constraints, and sociopolitical pressures. Thus, mental health professionals who wish to develop or implement novel treatment programs must be prepared to defend sedulously their rationale and treatment practices, and even then to be faced with external mandates for change. Consistent programming becomes especially problematical. However, the Paul and Lentz experience indicates that diligence in defending new programs can greatly facilitate their continued existence.

Program Accessibility to Staff. Public psychiatric hospitals must rely on nonprofessionals (e.g., high school graduates) with some additional training to provide most of the ongoing patient care. Consequently, alternative treatment approaches must be comprehensible and manageable by staff members lacking advanced training in the behavioral sciences. One of the greatest strengths of the comparative study reported by Paul and Lentz (1977) is the demonstration that nonprofessional staff could be taught to conduct two comprehensive and rigorous but distinct "high technology" psychosocial treatment programs. After less than 6 months of training, all trainees were regularly certified to work independently in all functional components of both the social-learning and milieu programs. Furthermore, continuous hourly time sampling on the SRIC verified that staff members were conducting the two programs differentially and with excellent fidelity to the models. After only 6 weeks of initial training, over 90 percent of the staff-patient interactions in the milieu program followed detailed procedures and over 98 percent of the staff-patient interactions in the social-learning treatment followed the social-learning procedures. After inservice training incorporated SRIC as well as CFRS data for staff feedback, the fidelity of staff execution of both psychosocial programs improved to more than 99.5 percent and remained there until termination (more than 2 years). Interestingly, staff opinion was somewhat divided about the alternative treatment programs. Most staff consistently judged the milieu program to be closer in ideology to their preferred form of intervention, but found the actual work on the social-learning unit to be more enjoyable and accurately judged the social-learning program to be more effective.

In summary, the social-learning program, which was the treatment of choice by all other criteria, is clearly manageable, acceptable, and accessible to the types and levels of staff already employed in public psychiatric hospitals. Nevertheless, the development and implementation of social-learning programs for chronic mental patients has critical prerequisites. These include substantial administrative support and encouragement; highly specialized and competent leadership; careful, extensive staff training; and an ongoing commitment to quality assurance. Only with these prerequisites satisfactorily met can a behavioral treatment program develop and flourish. The implications of implementing a social-learning program for chronic mental patients are outlined more fully in table 2.

Rehabilitative Characteristics of Psychosocial Programs

The focus of this section is on program characteristics that can improve the clinical outcome for institutionalized patients, drawing on the findings from the Paul and Lentz study (Paul and Lentz 1977; Paul 1984) and other relevant findings in the literature.

Commonalities Between the Social-Learning and Milieu Programs. Four common remediative aspects of the

Table 2. Implications of Paul & Lentz's (1977) report

Implications for whom	Implication
On-line staff in psychiatric facilities	Even the most disordered institutionalized schizophrenics can be treated effectively, treated and discharged to community care: work in structured/effective units is more enjoyable than unstructured ineffective units
Mental health professionals	Through psychosocial programming, most institutionalized psychiatric patients can be stabilized and discharged without psychotropic medications; comprehensive programs that effectively use nonprofessional staff are more enjoyable and better than when only professionals try to carry the treatment load
Psychiatric hospital administrators and unit directors	Nonprofessional staff can be taught to conduct a rigorous comprehensive treatment program reliably, thus increasing the effectiveness of treatment; aftercare should be integrated with inpatient programs
Mental health policy makers	Using funds to develop and implement social-learning programs at public psychiatric facilities is an investment that will be recouped by shorter hospital stays and reduced recidivism; policy decisions can have far-reaching negative effects and should be piloted carefully before they are mandated
Chronically institutionalized patients, their loved ones, and society at large	Rational, cost-effective, and humanitarian treatment programs can be implemented; the situation need not continue to be hopeless

psychosocial programs in the long-term comparative study have also received considerable theoretical and empirical support in other literature. These features are not only necessary prerequisites to the proper implementation of a social-learning program but are likely to enhance the rationality and rehabilitative efficacy of any inpatient or residential program for all patient populations in the mental health field.

Ongoing assessment systems. The CFRS, employed by clinical staff as an integral part of the conduct of treatment programs, and the TSBC and SRIC, employed by a small cadre of independent noninteractive observers, provided ongoing moment-to-moment and hour-by-hour objective assessment of patient and staff functioning in both psychosocial programs. These instruments are not only necessary to provide the detailed information for ongoing monitoring needed to implement the treatment of choice, but a thorough analysis of sources of information and methods of assessment found them to be prototypical primary strategies of a comprehensive assessment paradigm that would provide all the information needed for reasonable, cost-effective operation of residential treatment programs and facilities (Paul and Mariotto 1986). These assessment strategies provide nearly all of the information on patient, staff, and program functioning needed for rational moment-to-moment concurrent monitoring of individuals and groups, placement and disposition decisions (i.e., patient admission/assignment and discharge/transfer; staff hiring/assignment and discharge/transfer), identification of patient problems and assets, staff development/utilization, absolute and comparative program evaluation, legal and ethical regulation/documentation, need for more expensive assessment strategies such as individual interviews, and applied research.

Paul and his colleagues have developed the TSBC and SRIC into the Computerized TSBC/SRIC Planned-Access Observational Information System (Paul 1986*b*, in press *c*) to provide timely information with documented applicability to all types of residential or inpatient programs for the full range of adult patients, including mentally retarded as well as alcohol/substance abuse and acute and chronic mentally ill (Paul, in press *a*, in press *b*). Because of the quality and amount of useful assessment information and documentation provided by independent observers and the computer, clinical staff are freed from unnecessary paperwork such that the four- to five-fold increases in attention received by patients from staff in the social-learning and milieu programs can be obtained without increasing existing numbers of staff or staff/patient ratios.

Decentralized administrative structure with integrated staffing. Another common and critical aspect of the psychosocial programs in the long-term comparative study was the decentralized administrative organization to the treatment units with permanent rather than rotating staff assignments, and authority and responsibility based on demonstrated competency rather than discipline. Comprehensive psychosocial programs could be housed in a variety of physical locations, including public or private mental hospitals, inpatient units of mental health centers or general hospitals, and community residential facilities; however, the decentralized administrative/staffing organization is crucial to allow consistent staff training, monitoring, and communication as well as continuity of care—preferably extended to responsibility for postdischarge aftercare as well. Such an organization allows the maximum use of the large number of existing nonprofessional staff as direct providers of service, instead of being just "caretakers" while the professionals attempt to provide treatment. As demonstrated in the long-term study and elsewhere, positive attitudes, program identification, and improved performance of all levels of staff can result from such a structure with integrated staffing. This structure also helps buffer patients and staff from the vagaries and irrationalities of funding and external political events which can keep some component of any centralized administrative structure, where authority is based on disciplinary lines, in upheaval at all times. It is probably the only way in which the low staffing levels and resultant desirable cost-effectiveness ratios obtained in the long-term study could provide effective treatment in any program. In fact, Paul and Lentz (1977) argue that all program components and funding, except maintenance functions, should be decentralized to program subunits on a block-grant performance-contracting basis to ensure continuity of programs and to provide incentives for effective treatment without increasing costs.

Comprehensive educational/rehabilitative focus. The common focus of the psychosocial programs on establishing minimal competencies in specific components of intrapersonal, interpersonal, and instrumental skills, reducing or eliminating extreme bizarre behavior, and bridging the gap from inpatient programs to community functioning was originally derived from a review of the literature on factors leading to hospitalization or rehospitalization.

More recent literature, as well as the predictability of community functioning up to 18 months after discharge from the psychosocial programs, strongly confirms the importance of these minimal goals for any inpatient or residential program. In addition, both social-learning and milieu programs structured the daily/weekly schedule of each patient so that specific times and places ensured the opportunity for assessment and training in each component of adaptive functioning while inappropriate behavior was dealt with whenever it occurred. Comprehensive scheduling of all patient waking hours as well as the step system further ensured that all aspects of patient functioning were treated in a systematic, integrated fashion to equip patients with the skills necessary for community living and concurrently protect patients and the community from premature discharges and revolving-door existences. Consistent with a wide range of other research, such a focus on structured activities, classes, and meetings served not only a skill-building function, but also resulted in significant decreases in many aspects of bizarre and idiosyncratic behavior for milieu patients as well as for social-learning patients.

Another important aspect of the educational/rehabilitative focus of both psychosocial programs was the descriptive language, "resident" versus "patient" status, and patient accountability employed. In traditional hospital settings, "patients" are defined as "sick" and are provided with "care" until they are "well." Such conceptualizations may be useful with physical illnesses, but they may also individually reinforce the patient for maintaining the "sick role" (Goffman 1961). While a full discussion of this controversy is outside the scope of this article, it is clear that setting attainable, positive goals for change and communicating to patients that they are capable and responsible for meeting these goals is a critical first step for any effective treatment program, including ones for the chronically mentally ill.

Consistent, systematic, goal-oriented staff-patient interactions. An important commonality between the social-learning and milieu programs allowed clear, unique, explicit rules about appropriate staff behavior in interacting with patients in a variety of settings which facilitated staff training and ongoing monitoring. This specification allowed consistent and systematic staff-patient interactions. Further, these staff-patient interactions and overall program structures were always goal-oriented such that each activity in each patient's daily schedule was included only if it served a specific purpose designed ultimately to achieve discharge to the community—including gradual reexposure to the less structured settings of the external community. Although Paul and Lentz present SRIC data documenting that the type of staff-patient interaction is more important for improving most classes of patient functioning than the amount, the greater consistency and amount of staff-patient interaction achieved in both psychosocial programs clearly allow for more efficient use of limited staff resources and were effective in reactivating dormant skills and in reducing cognitive dysfunction and indicants of thought disorder in both programs.

Uniquely Effective Ingredients of the Social-Learning Program. By attending to the commonalities between the social-learning and milieu treatments, mental health practitioners working with chronically institutionalized patients can enhance the rehabilitative efficacy of their overall programs. Further improvements in patient functioning may be accomplished by implementing the unique features of the social-learning program, which was the most effective treatment of all and the clear treatment of choice for the chronically institutionalized.

Integrated procedures emphasizing the acquisition of patient competencies. Although both milieu and social-learning programs showed higher staff-patient interactions than traditional programs, no specific class of patient behavior covaried with total staff activity or attention received by patients. Rather, staff-patient interactions following social-learning principles were more effective with both equal and fewer rates of interaction than in the milieu program. This was especially true for increasing adaptive behavior and teaching patient competencies. The specification of desired behavior, provision of opportunities to perform, and overall goal-directed staff-patient interactions resulted in substantial improvements within 1-2 months in both psychosocial programs; however, milieu patients' adaptive functioning then leveled off or declined whereas social-learning patients' adaptive functioning continued to show gradual and major improvements. Paul and Lentz suggest that the initial response reflected the activation of previously dormant skills, with SRIC data showing that adaptive functioning of milieu patients was maintained only when milieu procedures happened to result in the application of staff-patient interactions that also followed social-learning principles (i.e., contingent social reinforcement).

In contrast, the consistent and systematic application of individual prompts and modeling, response-contingent consequences with both social and material reinforcement, associative learning procedures, and careful breaking down of patient adaptive behavior into learnable components that successively approximated the "normal" functioning ultimately desired was remarkably effective in helping social-learning patients to acquire new self-care, interpersonal, and communications skills. These social-learning procedures, combined with brief time-out and token response costs for failures, were also exceptionally effective in increasing patients' attention span, attendance at classes, meetings, and vocational training, and other instrumental skills as well as increasing the complexity of performance in vocational areas. Overall, these social-learning procedures, which have shown documented effectiveness when applied independently in other studies with a wide range of functional psychotics (Wong et al. 1986), were uniquely powerful in developing new intrapersonal, interpersonal, and instrumental skills for even the most severely disabled patients when the procedures were integrated within the comprehensive program.

Integrated procedures to reduce or eliminate extreme bizarre and inappropriate behavior. In keeping with other research findings, the active participation in alternative adaptive activities did lead to significant reductions in bizarre motoric behavior and some aspects of cognitive dysfunction (e.g., delusions and hallucinations, incoherent speech, and smiling without a stimulus). The vast majority of social-learning patients reduced extreme maladaptive behavior as a result of staff ignoring inappropriate acts while prompting and differentially reinforcing incompatible adaptive behavior. The explicit, consistent, behavior-contingent application of time-out and token response-costs was also usually effective in reducing or eliminating extreme bizarre behavior, including dangerous and aggressive acts, when these procedures were applied within the context of the overall constructive focus of the social-learning program—even though the frequency of application and level of aversiveness were lower than procedures typically applied in most institutional programs. Overcorrection/restitution procedures were 100 percent effective in eliminating assaults and other dangerous behavior that did not respond to the overall programmatic procedures. Although the consistent application of such negative consequences was an important and effective component of the integrated social-learning program, its greatest value may be in increasing the effectiveness of purely verbal social-learning procedures, since verbal prompts were 94 percent effective in terminating ongoing bizarre behavior from the start of the program, increasing over time to 98 percent effectiveness. Thus, the consistency of such staff-patient interactions was critical in terminating bizarre and psychotic acts to allow positive training in alternative coping and problem-solving skills. Social-learning procedures involving graduated reinforced exposure in stressful circumstances, the provision of active solutions, information, and advice, and training in alternative adaptive skills were considerably more effective than milieu procedures in assisting patients to overcome negative emotional reactions to stress and maladaptive behavior based on conditioned anxiety reactions. Although these overall programmatic procedures were adequate in reducing such emotional reactions in the chronically mentally ill to a level allowing discharge and community tenure, the most effective social-learning procedures for treating conditioned anxiety reactions would require more professional staff for their inclusion (see Paul and Bernstein 1976), especially if large numbers of acutely admitted patients were being treated.

Token-economy structure. The overall token-economy motivational structure was a critical component of the social-learning program for effective treatment of the severely debilitated chronic psychiatric patient. However, Paul and Lentz caution that the "token economy" of the social-learning program may have little in common with others that do not include all of the unique integrated aspects of the specific program that was found to be the treatment of choice. For severely desocialized patients, tangible tokens within a "tight economy" (preventing inflation and deflation), which includes primary reinforcers as backups, are necessary to provide the immediate consequences required for motivational purposes and to facilitate new learning by providing immediate feedback (e.g., Skinner 1938; Reynolds 1968). Such undersocialized patients respond to few purely social stimuli without new learning and any time lag at all allows interference from poor attention and/or the inability to receive and process information (Wallace and Boone 1984). The consistent staff verbal and nonverbal interactions with patients, contingently paired with token disbursement and collection, not only make the immediate social communication salient but, over time, patients gradually learn to value and benefit from social contact, verbal instruction, and social reinforcement alone. The token-economy structure comes to function primarily as an efficient management system for patients as they develop internal controls and learn to value social stimuli, or for patients who are less severely disabled initially. The carefully developed and implemented token economy thus provides the immediacy of informational feedback and reinforcement with fine gradations for patients who need it and an overall management system for all patients until they function well enough to "buy out" of the use of tangible tokens at the highest step in the program.

Consistent pacing and generalization training. Considerable research on cognitive processes in schizophrenia indicates that either understimulating environments or hyperarousal can interfere with performance and/or the reception, processing, and action upon relevant versus irrelevant information. Many studies have reported deleterious effects from some intensive inpatient programs (Liberman, Falloon, and Wallace 1984). The careful structure and pacing of focused, positively reinforced concrete skills training in small steps over all waking hours, as included in the comprehensive inpatient social-learning program, in addition to the application of specific principles and procedures to particular problems, may be critical to effectiveness with the severely disabled. High-demand programs with less pacing in concrete problem solving and skills building may overstimulate patients who are hyperaroused or who are too deficient in cognitive skills to benefit from new information in time-limited training periods. Low-demand programs may fail to activate patients enough to attend to external stimuli or attempt to learn from the services offered. The careful pacing incorporated within the comprehensive social-learning program also ensures generalization of patient skills across staff and situations with gradual fading of dependence on immediate token reinforcement. As patients progress in the social-learning program, treatment and consultations are systematically shifted from staff-initiated problem identification, solution, and skills training through eliciting problem solving from patients to patient-initiated problem solving and action. Paul (1984) notes that these aspects of the social-learning inpatient and aftercare programs of the long-term comparative study are common to successful community or aftercare programs starting with less severely debilitated patients that have been reported by more than 10 clinical research groups in different locales.

Current Status of Social-Learning Treatment

Because of the experimental design of the long-term comparative study reported by Paul and Lentz, it is the only study to date in the institutional literature to provide cause-effect evidence for the absolute and relative effectiveness of total treatment programs. Consequently, the comprehensive social-learning program in that study is the clear treatment of choice for the severely debilitated chronic mental patient. The findings of the long-term comparative study also provide support and clarification for the effectiveness of a number of social-learning principles and procedures reported in a wide range of research and clinical studies (Liberman et al. 1974; Liberman, King, and DeRisi 1976; Wong et al. 1986). Given the results of the Paul and Lentz study, it is somewhat surprising that social-learning programs are still in the minority in psychiatric facilities. Nevertheless, the importance of social-learning interventions can be seen both in the growing emphasis on patient accountability for behavior and in specialized psychiatric treatment programs for especially problematic patients.

General Incorporation of Accountability and Awareness of Incentives. The principles of social-learning have had at least two major effects on the current psychiatric treatment of institutionalized patients. First, holding patients accountable for their actions appears to be a common component of many programs. Level systems and earned privileges are routinely found as part of the treatment plan in most psychiatric hospitals. Even severely disorganized patients are often expected to perform simple self-care and housekeeping skills before being given access to television, grounds, and other privileges. Second, the philosophy of positively reinforcing adaptive behavior and punishing negative behavior (behavior modification) is well known to most mental health professionals today. Behavioral programs providing access to reinforcers (e.g., cigarettes, coffee) for adaptive behavior and limiting access to such reinforcers (e.g., time-out, extinction) for maladaptive behavior are integral parts of many psychiatric treatment plans. Nevertheless, well-organized, behaviorally oriented treatment programs are the exception rather than the rule (Boudewyns and Fry 1985).

Camarillo Clinical Research Unit (CRU). The CRU at Camarillo State Hospital is a unique psychiatric unit that strives to adhere rigorously to the social-learning principles found effective in the study reported by Paul and Lentz. This 11-bed unit, under the joint sponsorship of

Camarillo State Hospital and the UCLA Department of Psychiatry, has been in continuous operation since 1970, longer than any other social-learning unit extant. The day-to-day unit programming and token economy are very similar to those reported by Paul and Lentz, with objective data on patients being collected almost continuously. All patients are expected to adhere to specified rules backed by contingencies of reinforcement. In addition, specific intervention programs are developed for problems not addressed through general unit programming (e.g., water intoxication patients earn ground privileges for 7 days of controlled liquid intake; a loud, verbally abusive patient accrues small token fines for each verbal outburst). The clinical research objectives of this unit require special equipment (e.g., computers) and higher staff/patient ratios than throughout the hospital, so additional funding is provided through National Institute of Mental Health grants, the University, and the State hospital.

Table 3. Impact of behavioral treatment on the Camarillo/UCLA Clinical Research Unit

Levels of improvement for different categories of behavioral problems targeted for the 199 patients treated on the CRU (1970-1985)

	No. of patients with problem behaviors	Average improvement level
Prader-Willi syndrome	9	1.0
Spitting and mucous smearing	3	1.0
Entering restricted areas	1	1.0
Inactivity, amotivation	1	1.0
Psychogenic polydipsia	1	1.0
Vomiting	1	1.0
Sexual deviance	7	1.57
Self-injury	20	1.65
Verbal aggression	35	1.66
Peskiness, demandingness	18	1.67
Work and leisure skills	48	1.84
Self-care, grooming	141	1.86
Depression	13	1.92
Social skills deficit	70	1.96
Stereotypic movements and posturing	32	1.97
Social isolation	13	2.0
Screaming, tantrums	2	2.0
Obsessive-compulsive behavior	1	2.0
Assault and property destruction	85	2.16
Delusional speech	21	2.19
Incontinence	3	2.3
Hallucinations, self-talk	5	2.60
Mutism	1	4.0
Incoherent speech	1	4.0
Total Number of patient behaviors treated	532	
Overall average improvement level		2.06

Pretreatment baseline rates of behavior were compared to the rate of the same behavior during the last 3 weeks of CRU treatment. For each subject, % change was calculated and "improvement level" was assigned using the following scale: marked improvement (1) = 75-100% change from baseline; moderate improvement (2) = 50-74% change from baseline; slight improvement (3) = 25-49% change from baseline; and no improvement (4) = less than 25% improvement.

The CRU serves three purposes at Camarillo State Hospital. First, it is an intensive treatment facility for some of the most intractable patients at the hospital. Patients referred to the CRU are usually long-term, multiple-admission patients with numerous behavioral excesses and deficits. A review of the data from 199 patients discharged from the CRU before June 1985 indicated that the most common patient problems included deficits in self-care and grooming, social skills, and work and leisure skills, as well as excesses in assaultiveness, property destruction, stereotyped movements and posturing, and verbal aggression. Seventy-two percent of the patients exhibited one or more of these difficulties when admitted to the CRU, but even with this chronic, refractory population, behavioral programming yielded substantial benefits. The typical patient improved by at least 50 percent from baseline levels on the spectrum of targeted clinical problems. Shown in table 3 is the improvement rate, measured through direct observation of patient's behavior, of clinical problems of these patients.

This positive effect of social-learning treatment was also maintained in long-term followup evaluation of discharged CRU patients. Banzett et al. (1984) found, in a 2-year followup of CRU patients discharged from 1975 to 1979, that 80 percent of respondents (patient relatives, conservators, or treatment staff) reported maintenance or further improvement both in the patients' overall functional and clinical status and in their original referring problems since discharge. Fifty-two percent of ex-patients were living in noninstitutionalized settings. Although this rate of discharge and community tenure is lower than that obtained by Paul and Lentz, the patient populations and goals are not directly comparable; this discharge rate is better than that of the chronic units at Camarillo State Hospital. It is especially impressive given the exceedingly low level of patient functioning resulting from the referral of intractable patients and the presence of State-mandated policies which prohibit use of more powerful primary reinforcers (e.g., food) for patients.

The CRU serves two other purposes at Camarillo State Hospital. It is a site for a variety of research projects directed toward improving functioning of institutionalized patients. Current research includes a study on the effects of innovative behavioral and pharmacotherapies on treatment-refractory schizophrenia and studies on social skills training (Wallace and Liberman 1985; Liberman et al. 1985) for severely withdrawn and thought-disordered schizophrenics. Finally, the CRU staff act as consultants on behavioral programming to other hospital staff who have identified patients for whom standard hospital treatment is ineffective. By acting as consultants, the CRU staff help to disseminate behavioral techniques and encourage the development of social-learning programs throughout the hospital. In recent years, members of the CRU staff, under the auspices of the UCLA Clinical Research Center for Schizophrenia and Psychiatric Rehabilitation, have provided technical assistance and professional training to over 3,000 mental health and rehabilitation practitioners in psychiatric agencies and hospitals throughout the world.

Dissemination of the Comprehensive Social-Learning Program. Although the consulting and dissemination functions of special units such as the Camarillo CRU can be expected to increase the rehabilitative efficacy of psychiatric programs as well as contributing new clinical-research findings, the full benefits of social-learning programs like the one reported by Paul and Lentz cannot be achieved by service providers without the complete implementation of comprehensive inpatient and aftercare elements, including integrated assessment systems. On the basis of their obtained rates of change, Paul and Lentz estimate that effective implementation of social-learning programs could improve the functioning of disabled chronic psychiatric patients to a level of acceptability for community placement in 26–30 weeks. In fact, given appropriate implementation mechanisms, social-learning programs could be conducted in community-based residential facilities as well as in hospitals and mental health centers.

The major impediment to widespread dissemination of the comprehensive social-learning program has been the unavailability of the assessment systems to provide the detailed information needed for the proper implementation and conduct of the program. Since Paul and his colleagues have completed the research and development for the TSBC/SRIC System to serve as the core assessment strategy for nearly any inpatient or residential program and population, the materials required to implement the TSBC/SRIC System (Paul, in press *d*) and the social-learning CFRS (Pau and Shelite, in preparation) will soon be available.

Recent Developments in Social-Learning Programs

Since the publication of the Paul and Lentz (1977) study, other researchers have continued to investigate social-learning approaches to the rehabilitation of the chronically mentally ill. In line with the work of Paul and Lentz, these authors typically find that social-learning programs result in dramatic improvements in behavior (Baker et al. 1977; Fullerton, Cayner, and McLaughlin-Reidel 1978; McCreadie, Main, and Dunlop 1978; Nelson and Cone 1979; Stoffelmayr, Faulkner, and Mitchell 1979; Stone and Nelson 1979; Turner and Luber 1980; Woods, Higson, and Tannahill 1984; Wong et al. 1985). Nevertheless, critical research and treatment issues remain.

One area of investigation concerns the identification of the remediative elements within the token economy program. For example, Baker et al. (1977) found that both a token economy patient group *and* a yoked control group improved in behavior, raising the possibility that improvement results from some other factor (e.g., enriched positive reinforcements) than contingent reinforcement. Studying a token economy program in a day hospital, researchers from the UCLA Clinical Research Center found that the specific behavioral goals and social reinforcement contingencies inherent in the economy accounted for therapeutic change rather than the tangible, backup rewards (Liberman et al. 1977). Similarly, Turner and Luber (1980) reported that patients judged *feedback* about performance (and not tokens) as being the most critical aspect of the program. Further research is needed to determine the relative importance of tokens in providing information and motivation. In addition, alternate explanations for the success of the token economy must be developed and tested.

A second important issue concerns generalization of improvements. Maintenance and generalization of treatment gains are, of course, critical tests of the utility of treatment interventions. For social-learning programs, the crucial question is: Are token economies primarily prosthetic, resulting in behavioral change only when it is supported by contingent token reinforcement; or are they therapeutic, resulting in long-term behavioral change across settings? The answer to this question has not been fully determined. Woods, Higson, and Tannahill (1984) found that there were extreme differences among patients' maintenance of behavioral improvements when contingent tokens were changed to noncontingent tokens. Some patients showed no decrements in performance while others deteriorated almost immediately. Interestingly, the authors' data suggested that rapid initial rates of change when the token economy was applied predicted maintenance. These investigators suggest that some patient improvements are maintained because they are "trapped" by positive social reinforcement. Certainly, identifying patient variables that are related to the development and maintenance of improvements in functioning is an important issue which merits attention.

Finally, the extreme deficits of treatment-refractory schizophrenics raise the possibility that a social-learning program may be a necessary but insufficient condition for successful community tenure of these patients. Two recent studies offered structured, intensive social skills training to treatment-resistant schizophrenics as an adjunct to their treatment on a token economy hospital unit with very encouraging results (Massel et al. 1985; Wallace and Liberman 1985). Patients' social skills increased dramatically in both studies, and Wallace and Liberman (1985) found that participation in social skills training actually decreased probability of relapse during a 2-year followup. This addition of specialized remediation of skill deficits to a strong social-learning program maximizes patients' likelihood of behavioral improvement and may augment the benefits of behavioral rehabilitation (Liberman and Evans 1985).

Because psychosocial interventions based on social-learning principles are very effective in reducing maladaptive behavior and increasing adaptive functioning of "hardcore" institutionalized psychiatric patients, reducing medications, facilitating discharge and maintaining community tenure, they merit continued attention, refinement, and dissemination. The comprehensive social-learning program, including the integrated assessment systems and inpatient and aftercare technologies, is the clear preferred treatment for chronically institutionalized mental patients on economic, clinical, and humanitarian grounds.

References

Baker, R.; Hall, J.K.; Hutchinson, K.; and Bridge, G. Symptom changes in chronic schizophrenic patients on a token economy: A controlled experiment. *British Journal of Psychiatry*, 131:381–393, 1977.

Banzett, L.K.; Liberman, R.P.; Moore, J.W.; and Marshall, B.D. Long term follow-up of the effects of behavior therapy. *Hospital & Community Psychiatry*, 35:277–279, 1984.

Bellack, A.S., and Mueser, K.T. A comprehensive treatment program for schizophrenia and chronic mental illness. *Community Mental Health Journal*, in press.

Boudewyns, P.A., and Fry, T.J. "Token Economy Programs in VA Medical Centers: Where Are They Today?" Presented at the Annual Meeting of the American Psychological Association, Los Angeles, CA, 1985.

Dickey, B.; Gudeman, J.; Hellman, S.; Donatella, A.; and Greenspoon, L. A follow-up of deinstitutionalized chronic clients four years after discharge. *Hospital & Community Psychiatry*, 32:326–329, 1981.

Friedman, I.; von Mering, O.; and Hinko, E.N. Intermittent patienthood: The hospital career of today's mental patient. *Archives of General Psychiatry*, 14:386–392, 1966.

Fullerton, D.I.; Cayner, J.J.; and McLaughlin-Reidel, T. Results of a token economy. *Archives of General Psychiatry*, 35:1451–1453, 1978.

Gardos, G., and Cole, J.O. Maintenance antipsychotic therapy: Is the cure worse than the disease? *American Journal of Psychiatry*, 133:32–36, 1976.

Goffman, E.C. *Asylums*. Garden City, NY: Doubleday, 1961.

Honigfeld, G. *Nurses Observation Scale for Inpatient Evaluation (NOSIE-30)*. Glen Oaks, NY: Honigfeld, 1966.

Kirk, S.Q., and Therrien, M.E. Community mental health myths and the fate of former hospitalized patients. *Psychiatry*, 38:209–217, 1975.

Kohen, W., and Paul, G.L. Current trends and recommended changes in extended-care placement of mental patients: The Illinois system as a case in point. *Schizophrenia Bulletin*, 2:575–594, 1976.

Lehman, A. The well-being of chronic mental patients. *Archives of General Psychiatry*, 40:369–373, 1983.

Lehman, A.F.; Ward, N.C.; and Linn, L.S. Chronic mental patients: The quality of life issue. *American Journal of Psychiatry*, 139:1271–1276, 1982.

Liberman, R.P. Social factors in schizophrenia. In: Grinspoon, L., ed. *Psychiatry 1982 Annual Review*. Washington, DC: American Psychiatric Press, 1982. pp 97–111.

Liberman, R.P., and Evans, C.C. Behavioral rehabilitation for chronic mental patients. *Journal of Clinical Psychopharmacology*, 5 (Supplement):8s–14s, 1985.

Liberman, R.P.; Falloon, I.R.H.; and Wallace, C.J. Drug-psychosocial interactions in the treatment of schizophrenia. In: Mirabi, M., ed. *The Chronically Mentally Ill: Research and Services*. New York: SP Medical & Scientific Books, 1984. pp. 175–212.

Liberman, R.P.; Fearn, C.H.; DiRisi, W.J.; Roberts, J.; and Carmona, M. The credit incentive system: Motivating the participation of patients in a day hospital. *British Journal of Social and Clinical Psychology*, 16:85–94, 1977.

Liberman, R.P.; King, L.; and DeRisi, W.J. Behavior analysis and modification in community mental health. In: Leitenberg, H., ed. *Handbook of Behavior Therapy and Behavior Modification*. Englewood Cliffs, NJ: Prentice Hall, 1976. pp. 566–603.

Liberman, R.P.; Massel, H.K.; Mosk, M.D.; and Wong, S.E. Social skills training for chronic mental patients. *Hospital & Community Psychiatry*, 36:396–403, 1985.

Liberman, R.P.; Wallace, C.J.; Teigen, J.; and Davis J. Interventions with psychotics. In: Calhoun, K.S.; Adams, H.E.; and Mitchell, E.M., eds. *Innovative Treatment Methods in Psychopathology*. New York: John Wiley & Sons, Inc., 1974. pp. 323–412.

Licht, M.H., and Paul, G.L. The Staff-Resident Interaction Chronograph (SRIC). In: Hersen, M., and Bellack, A.S., eds. *Dictionary of Behavioral Assessment Techniques*. New York: Pergamon Press, in press.

Massel, H.K.; Bowen, L.; Wong, S.; Mosk, M.; Zarate, R.; and Milan, M. "Social Skills Training: Acquisition and Generalization Effects." Presented at the Annual Meeting of the Association for the Advancement of Behavior Therapy, Houston, TX, 1985.

McCreadie, R.G.; Main, C.J.; and Dunlap, R.A. Token economy, pimozide and chronic schizophrenia. *British Journal of Psychiatry*, 133:179–181, 1978.

Mechanic, D. *Mental Health and Social Policy*. Englewood Cliffs, NJ: Prentice-Hall, 1969.

Nelson, G.L., and Cone, J.D. Multiple baseline analyses of a token economy for psychiatric inpatients. *Journal of Applied Behavior Analysis*, 12:255–271, 1979.

Paul, G.L. Residential treatment programs and aftercare for the chronically institutionalized. In: Mirabi, M., ed. *The Chronically Mentally Ill: Research and Services*. New York: S.P. Medical and Scientific Books, Spectrum Publications, 1984. pp. 239–262.

Paul, G.L. Net relative cost of the maximum potential utility paradigm. In: Paul, G.L., ed. *Principles and Methods to Support Cost-Effective Quality Operations. Assessment in Residential Treatment Settings, Part 1*. Champaign, IL: Research Press, 1986*a*. pp. 165–190.

Paul, G.L. Rational operations in residential treatment settings through ongoing assessment of client and staff functioning. In: Peterson, D.R., and Fishman, D.B., eds. *Assessment for Decision*. New Brunswick, NJ: Rutgers University Press, 1986*b*. pp. 1–35.

Paul, G.L., ed. *Observational Assessment Instrumentation for Service and Research—The Time-Sample Behavioral Checklist: Assessment in Residential Treatment Settings, Part 2*. Champaign, IL: Research Press, in press *a*.

Paul, G.L., ed. *Observational Assessment Instrumentation for Service and Research—The Staff-Resident Interaction Chronograph: Assessment in Residential Treatment Settings, Part 3*. Champaign, IL: Research Press, in press *b*.

Paul, G.L., ed. *Observational Assessment Instrumentation for Service and Research—The Computerized TSBC/SRIC Planned-Access Observational Information System: Assessment in Residential Treatment Settings, Part 4*. Champaign, IL: Research Press, in press *c*.

Paul, G.L., ed. *Observational Assessment Instrumentation for Service and Research—The TSBC/SRIC System Implementation Package: Assessment in Residential*

Treatment Settings, Part 5. Champaign, IL: Research Press, in press *d.*

Paul, G.L., and Bernstein, D.A. Anxiety and clinical problems. In: Spence, J.T.; Carson, R.C.; and Thibaut, J.W., eds. *Behavioral Approaches to Therapy.* Morristown, NJ: General Learning Press, 1976. pp. 47-87.

Paul, G.L., and Lentz, R.J. *Psychosocial Treatment of Chronic Mental Patients: Milieu Versus Social-Learning Programs.* Cambridge, MA: Harvard University Press, 1977.

Paul, G.L., and Lentz, R.J. *Psychosocial Treatment of Chronic Mental Patients: Milieu Versus Social-Learning Programs.* 2nd ed. Champaign, IL: Research Press, in preparation.

Paul, G.L., and Licht, M.H. The Clinical Frequencies Recording System (CFRS). In: Hersen, M., and Bellack, A.S., eds. *Dictionary of Behavioral Assessment Techniques.* New York: Pergamon Press, in press *a.*

Paul, G.L., and Licht, M.H. The Time-Sample Behavioral Checklist (TSBC). In: Hersen, M., and Bellack, A.S., eds. *Dictionary of Behavioral Assessment Techniques.* New York: Pergamon Press, in press *b.*

Paul, G.L., and Mariotto, M.J. Potential utility of the sources and methods: A comprehensive paradigm. In: Paul, G.L., ed. *Principles and Methods to Support Cost-Effective Quality Operations: Assessment in Residential Settings, Part 1.* Champaign, IL: Research Press, 1986. pp. 113-164.

Paul, G.L., and Shelite, I. The Clinical Frequency Recording System (CFRS): Social-Learning Program Forms. Supplement to: Paul, G.L., and Lentz, R.J. *Psychosocial Treatment of Chronic Mental Patients.* 2nd ed. Champaign, IL: Research Press, in preparation.

Paul, G.L.; Tobias, L.L.; and Holly, B.L. Maintenance psychotropic drugs in the presence of active treatment programs: A "triple-blind" withdrawal study with long-term mental patients. *Archives of General Psychiatry,* 27:106-115, 1972.

Reynolds, G.S. *A Primer of Operant Conditioning.* Glenview, IL: Scott, Foresman, 1968.

Rhoades, L.J. *Treating and Assessing the Chronically Mentally Ill: The Pioneering Research of Gordon L. Paul.* (DHHS Publication No. ADM 81-1100) Rockville, MD: Science Reports Branch, Division of Scientific and Public Information, National Institute of Mental Health, 1981.

Rosen, A.J.; Sussman, S.; Mueser, K.T.; Lyons, J.S.; and Davis, J.M. Behavioral assessment of psychiatric inpatients and normal controls across different environmental contexts. *Journal of Behavioral Assessment,* 3:25-36, 1981.

Skinner, B.F. *The Behavior of Organisms.* New York: Appleton-Century-Crofts, 1938.

Spivack, G.; Siegel, J.; Sklaver, D.; Deuschle, L.; and Garrett, L. The long-term client in the community: Lifestyle patterns and treatment implications. *Hospital & Community Psychiatry,* 33:291-295, 1983.

Stoffelmayr, B.E.; Faulkner, G.E.; and Mitchell, W.S. The comparison of token economy and social therapy in the treatment of hard-core schizophrenic patients. *Behavior Analysis and Modification,* 3:3-17, 1979.

Stone, M.E., and Nelson, G.L. Coordinated treatment for long-term psychiatric inpatients. *Social Work,* 15:406-410, 1979.

Turner, S.M., and Luber, R.F. The token economy in day hospital settings: Contingency management or information feedback. *Journal of Behavior Therapy and Experimental Psychiatry,* 11:89-94, 1980.

Wallace, C.J., and Boone, S.G. Cognitive factors in the social skills of schizophrenic patients: Implications for treatment. In: Spaulding, W.D., and Cole, J.K., eds. *Nebraska Symposium on Motivation, 1983.* Lincoln, NE: University of Nebraska Press, 1984. pp. 283-318.

Wallace, C.J., and Liberman, R.P. Social skills training for patients with schizophrenia: A controlled clinical trial. *Psychiatry Research,* 15: 239-247, 1985.

Wing, J.K., and Freudenberg, R.K. The response of severely ill chronic schizophrenic patients to social stimulation. *American Journal of Psychiatry,* 118:311-322, 1961.

Wolkon, G.H.; Karman, M.; and Tanaka, H.T. Evaluation of a social rehabilitation program for recently released psychiatric patients. *Community Mental Health Journal,* 7:312-322, 1971.

Wong, S.E.; Terranova, M.D.; Bowen, L.; Zarate, R.; Massel, H.K.; and Liberman, R.P. "Reducing Bizarre Stereotypic Behavior in Chronic Psychiatric Patients: Effects of Supervised and Independent Recreational Activities." Unpublished manuscript, 1985.

Wong, S.E.; Massel, H.K.; Mosk, M.D.; and Liberman, R.P. Behavioral approaches to the treatment of schizophrenia. In: Burrows, G.D.; Norman, T.R.; and Rubenstein, G., eds. *Handbook of Studies on Schizophrenia.* Amsterdam: Elsevier Science Publishers, 1986. pp. 79-100.

Woods, P.A.; Higson, P.J.; and Tannahill, M.M. Token economy programmes with chronic psychiatric patients: The importance of direct measurement and objective evaluation for long-term maintenance. *Behaviour Research and Therapy*, 22:41-51, 1984.

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Audio Tapes on Schizophrenia Available

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Sex Education and Rehabilitation With Schizophrenic Male Outpatients

by David Lukoff, Debbie Gioia-Hasick, Greer Sullivan, Joshua S. Golden, and Keith H. Nuechterlein

Abstract

Research indicates that schizophrenic patients lack intimate relationships and show a high rate of sexual dysfunction. Despite increasing awareness of the rights of handicapped persons to sexual expression, the treatment of schizophrenic patients rarely addresses their sexuality. A sex education program for recent-onset male schizophrenic patients attending an outpatient clinic was developed in response to several incidents involving patients' inappropriate sexual behaviors. To enhance our understanding of the current sexual functioning and needs of these patients, sex histories were taken. Almost all of the 16 patients interviewed were sexually active, with autoerotic activity predominating. Sixty-three percent of the patients reported orgasmic and/or erectile dysfunctions. Other studies have linked sexual dysfunction to the side effects of antipsychotic medications. The objectives of the sex education program were: (1) to provide information; (2) to clarify values; (3) to overcome sexual dysfunction; and (4) to enhance intimacy skills. The authors used role playing, modeling, group exercises, and explicit sex therapy audiovisual material to improve patients' intimacy skills. Patients participated actively and used the group to explore sexual issues. No exacerbations of symptoms were observed among patients participating in the program.

Sexual functioning is a legitimate and important concern in a comprehensive rehabilitation program for schizophrenic patients. While many rehabilitation programs for physically disabled (Madorsky and Dixon 1983) and ill (Golden 1986) people now include sexual rehabilitation as part of comprehensive care, the sexual functioning of mentally ill people continues to be ignored (Wasow 1980; Friedman and Harrison 1984). The Sex Information and Education Council of the U.S. (1986), which operates a clearinghouse for sex education materials, recently noted:

> Unfortunately . . . there are no sex education resources, such as curricula or booklets, at this time for either the mentally ill themselves or educators working with them. [p. 4]

No studies of the outcome of sex education with schizophrenic patients have been published, and there are only a handful of anecdotal accounts of sex education programs with the mentally ill reported in the literature.

Several factors contribute to this situation. Psychiatric staff members report discomfort when having to deal personally with patients about sexual concerns (Wolfe and Menninger 1973; Withersty 1976). This personal discomfort is compounded by the fear that psychiatric patients are not capable of responsibly handling their sexuality and that recognition of their sexuality might trigger inappropriate behavior (Sadow and Corman 1983).

Since at least the early 1900s, psychiatry has linked sexual activity and psychiatric illness. Von Krafft-Ebing in 1904 maintained that sexual excesses, particularly masturbation, caused insanity. Even as recently as 1972, a survey of 18 hospital-based psychiatrists in Boston found that "consultant and staff psychiatrists believed sexual activity might have been a contributory factor for all listings under anxiety and depression

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and for two-thirds of those under schizophrenia" (Pinderhughes, Grace, and Reyna 1972, p. 98). Furthermore, those surveyed thought that sexual activity could retard recovery in one- to two-thirds of their schizophrenic patients. Although there is no empirical evidence to support these beliefs, they continue to influence the expectations and behavior of mental health professionals. In the same survey, for example, psychiatrists stated that they were much less likely to discuss sexual functioning with schizophrenic patients than with depressed or anxious patients.

Fears that sex education programs lead to increases in sexual acting out have been shown to be false in studies with high school students (Kirby 1985). With developmentally disabled patients, Demetral (1981) found that sex education did not worsen but improved patients' behaviors relating to sexuality, including appropriateness and hygiene. With a population of psychiatric inpatients, Wolfe and Menninger (1973) noted that after a sex education program, "no tendencies were seen toward eroticism, nor were there any additional major sexual experiences, such as intercourse or homosexuality noted on the ward" (p. 150). Patients in this and other programs were found to be eager for sex education and receptive to it (Axelrod, O'Brien, and Hines 1973; Mindek 1974; Wasow 1980).

Sexual Dysfunctions in Schizophrenic Patients

Investigators have studied the nature of sexual functioning in the normal population (Levine and Yost 1976) and a variety of acute and chronic illnesses (LoPiccolo and LoPiccolo 1980). The situation in psychiatric disorders is different:

> Surprisingly, psychiatry, in contrast to other medical specialties, has generated little data about the sexual behavior of patients suffering from the various mental disorders. [Friedman and Harrison 1984, p. 555]

As a result, little is known about the "natural history" of sexual functioning in people with schizophrenia. A few studies of hospitalized chronic schizophrenic patients have been reported (Rozan, Tuchin, and Kurland 1971; Akhtar et al. 1977; Nestros, Lehman, and Ban 1981). These studies suggest that schizophrenic patients' sexual functioning is both qualitatively and quantitatively different from normals. Nestros, Lehman, and Ban (1981) found that schizophrenic patients were more likely, even premorbidly, to have autoerotic behavior as their primary sexual activity. With one exception (Lukianowicz 1963), these studies have reported that schizophrenic patients have less overall sexual activity of any type than do normals. Furthermore, Nestros, Lehman, and Ban (1981) found that while normal sexual activity peaked at ages 30–39 and then gradually dropped off, the schizophrenic patients' sexual activity dropped progressively after adolescence.

For a better understanding of the sexual functioning of our patients, we administered a 15-minute interview covering sexual history and current sexual behavior to 16 Caucasian male patients with schizophrenia at the UCLA Aftercare Clinic. One other patient in the clinic declined to answer the questions and another was too conceptually disorganized to provide useful information. The average age of the patients in the survey was 25.6 years and most came from social classes III–V. Since the Aftercare Clinic is linked to a longitudinal follow-through study of recent-onset *DSM–III* schizophrenic and schizophreniform patients, all patients in the group had been ill less than 2 years at entry into the clinic. These are the first published data on the sexual functioning of recent-onset male schizophrenic patients living in the community.

The data in table 1 show that recent-onset male schizophrenic patients are sexually active, with most of their sexual activity being autoerotic. Only 19 percent reported having a current sexual partner and the median number of sexual partners during their lifetime was three, considerably below average for a group of males in their twenties (Kinsey, Pomeroy, and Martin 1948). Nineteen percent reported never having had a sexual partner. Current homosexual contacts were reported by only one patient, and one other patient reported a homosexual experience in the past.

Sexual dysfunctions were reported by 63 percent of the patients. Five said they had difficulty achieving or maintaining erections when masturbating. During sexual intercourse, one reported difficulty with erections 25 percent of the time, two with premature ejaculation 25 percent of the time, and two with retarded ejaculations 25 percent of the time. Two patients reported more than one sexual dysfunction. Four did not answer these questions (including the three who had never had sexual intercourse). Thus, 3 of the 12 patients who responded to questions about sexual intercourse reported sexual dysfunction 25 percent of the time. Several of the patients who reported no dysfunction admitted during the interview that

Table 1. Frequency of sexual activity of recent-onset male schizophrenic patients (n = 16)

Sexual activity		Every-day	Several times a week	Once a week	Once a month	Once every several months	Once a year	Never
Mastur-bation	%	19	38	31	6	0	6	0
	n	3	6	5	1	0	1	0
Sexual inter-course	%	6	6	0	6	19	13	50
	n	1	1	0	1	3	2	8

they had few experiences on which to base their report.

These rates are consistent with the high incidence of sexual dysfunction reported in the literature in medicated schizophrenic patients (Ghadirian, Chouinard, and Annable 1982). Studies in the general population have found the prevalence of orgasmic dysfunction to be somewhere between 15 and 25 percent and the prevalence of impotence, about 10 percent (Frank, Anderson, and Rubinstein 1978). Thus, the rates for schizophrenic patients are clearly elevated.

Many of these sexual dysfunctions are known to be related to antipsychotic medications including: total inhibition of ejaculation and retarded ejaculation (Blair and Simpson 1966); spontaneous ejaculation (Keitner and Selub 1983); difficulties in achieving and maintaining erections (Kotin et al. 1976); decreased sexual desire (Witton 1962); painful orgasms (Berger 1979); and priapism, a prolonged painful erection which frequently requires immediate surgical intervention (Moloney, Elliot, and Johnson 1976).

Sex Education for Schizophrenic Patients

There are ample reasons for including sexual functioning in rehabilitation programs:

1. Schizophrenic patients, like others, are sexual beings and the recognition and validation of their sexuality is an important "quality of life" issue. In our culture, sexual competence is a major component of self-esteem and well-being (Golden and Golden 1980).

2. The incidence of sexual dysfunction in schizophrenic patients is high. Studies, including our own, have found that 30–60 percent of patients have major dysfunctions (Nestros, Lehman, and Ban 1981). Worry and concern over having a sexual dysfunction can be a stressor. Within the vulnerability-stress-coping-competence model of the course of schizophrenia (Liberman et al. 1986), sexual dysfunction could be a contributing stress factor leading to the exacerbation of symptoms or relapse, as well as impairing coping and competence.

3. Noncompliance with medication among some patients has been attributed to complaints about medication-induced side effects of sexual dysfunction (Carlson and Sadoff 1971). Many studies on the causes of noncompliance have not considered this source, and it may be greatly underreported in the literature.

4. Training schizophrenic patients in intimacy skills and motivating them to develop sexual relationships could help them to overcome social isolation. In addition to being considered a negative symptom of schizophrenia, social isolation is also a risk factor in relapse (Sokolovsky et al. 1978). Both social and sexual functioning appear to deteriorate with time in the absence of intervention.

5. Inappropriate sexual behaviors, sexual fears, and delusions of some patients may be major obstacles to their integration into the community (Akhtar and Thompson 1980).

6. Reproductive rates of both male and female schizophrenic patients appear to be on the increase (Erlenmeyer-Kimling et al. 1969). Many of these pregnancies are unwanted and unplanned, suggesting that awareness and use of birth control are lacking (Abernethy et al. 1976). In addition, schizophrenic women seem to be at high risk for sexual abuse (Friedman and Harrison 1984).

Sex Education Program

At the UCLA Aftercare Clinic, an outpatients clinic for schizophrenic and schizophreniform patients, problems of sexual dysfunction, as well as problems linked to inappropriate sexual behaviors, led to our decision to introduce a sex education program. These incidents included: (1) two female receptionists complained to our staff that two male patients from the Aftercare Clinic were constantly asking them for dates and making inappropriate sexual remarks to them; (2) one male patient who had his first experience of sexual intercourse caused a disruption at his board-and-care residence by claiming that his girlfriend's baby was his and they should get married; (3) one patient had the delusion that little children were being placed in front of him to tempt him to molest them; (4) one female patient underwent a pregnancy scare which caused her great distress; and (5) one male patient was asked not to return to his health club because several of the women complained that he touched them and made sexual remarks to them in the jacuzzi. In addition, several patients complained during therapy sessions about their lack of an active sex life and guilt over masturbation.

Aftercare Clinic patients were invited to participate in a series of group sex education sessions. Since some of them came to the clinic on Mondays and others on Thursdays, two sections of the group were offered. Not only the patients specially nominated for the sex education group by the Aftercare Clinic team, but all 18 male patients coming to the clinic on these 2 days were offered the sessions. Although we would have preferred a coed group, our clinic is disproportionately composed of male patients. Since only one female patient was attending the clinic on either of these 2 days, we limited the group to male patients.

Objectives. Since no curricular materials for schizophrenic patients were available, the first two authors designed a program which incorporated the standard goals that have been established for sex education programs in general: (1) to increase participants' knowledge and comfort about their sexuality and the sexuality of others; (2) to help participants identify and clarify their values and attitudes about sexuality and; (3) to help participants acquire decision-making skills (George and Behrendt 1985).

However, schizophrenic patients have additional specific clinical needs such as overcoming sexual dysfunction, preventing deterioration of sexual functioning, and learning intimacy skills. The sex education materials available for students (McCary 1975) and developmentally disabled patients (de la Cruz and La Veck 1973) do not incorporate these goals. Programs for persons with major physical disabilities who are at risk for loss of sexual functioning do deal with sexual functioning graphically and explicitly (Madorsky and Dixon 1983).

To help us develop a sex education program, we consulted with the staff of the UCLA Human Sexuality Program who have experience working with physically ill and disabled patients. The result was a program that incorporated sexually explicit material usually used during sex therapy sessions—for example, the *Sensate Focus* films (Bozanian 1972) which show a couple engaging in and then discussing mutually pleasurable exercises.

After reviewing the results from our survey, we also included more rudimentary intimacy skills training than is typical for sex education programs—for example, role plays and discussions focused on finding appropriate sexual partners, initiating sexual encounters and communicating about sex with partners. This curriculum would probably be considered too explicit for most sex education programs conducted in public educational settings.

Of the 16 patients who completed the sex history and functioning interview, 10 attended the sex education program regularly. Two groups of six and four patients met for eight biweekly 1-hour sessions. The groups were co-led by David Lukoff, Ph.D., and Debbie Gioia-Hasick, L.C.S.W. Joshua Golden, M.D., Director of the UCLA Human Sexuality Program, provided supervision for the groups. (See table 2 for an outline of the lesson plans used in the group education program.)

Results of the Program

Participation in the group started with an interview on sexual history and functioning. When the interview schedule was first presented at the Aftercare Clinic team meeting, there was a collective "gulp" at the explicitness of the questions. The mental health professionals described some discomfort at having patients respond to questions about age at first masturbation and first sexual intercourse, number of partners, and occurrence of sexual dysfunction. In contrast, the interviewers did not find that the schizophrenic patients showed signs of discomfort at being asked these questions.

Although we did not inquire into the reasons six patients chose not to participate in the group, it is possible that the interview served to forewarn and screen out patients who did not wish to explore further issues of their sexuality. Unfortunately, this included the patient who had delusions that children were being placed in front of him to tempt him sexually. However, he was being seen in individual therapy which addressed this symptom.

Table 2. Content of sex education sessions held with schizophrenic patients

Session 1. Your sexual identity and self-esteem

Brief discussion on the importance of sex in everyone's life.

Exercise: Make a collage of your sexual identity. Magazines (*Playboy, Time, Sports Illustrated*, etc.), scissors, glue, crayons, and string were available for this exercise. Presentation and discussion by each person of the meaning of his drawing.

Session 2. Thinking of your sexual partner as a person

Exercise: List and then share three characteristics of someone you would like to meet and go on a date with. Group brainstorming session of places where one might meet potential dating partners. Role plays of introducing yourself to someone at a party.

Session 3. A. Male reproductive anatomy
B. Your strengths as a partner

Slide show of male reproductive anatomy including information on the size and functioning of the penis.

Exercise: Write a classified ad about yourself for a singles magazine. Discussion of each person's assets as a potential partner in a relationship.

Session 4. A. Female reproductive anatomy
B. Dating behavior

Slide show of female reproductive anatomy including the relationship of menstruation and lubrication to sexual intercourse.

Exercise: Share personal experiences of dates, e.g., who initiated the date and how; what did you do on the date; how did the date move into sexual intimacy; how did you feel during the different sexual phases?

Session 5. Pleasure not performance as the focus in sex

Viewing of ***Sensate Focus***, which shows a couple engaging in the Masters and Johnson sensate focus exercises. Discussion of the importance of communication between partners about what is pleasurable during sexual activities.

Exercise: Each person was given a navel orange and asked to explore and report on the sensory processes of seeing, feeling, smelling, peeling, and eating the orange.

Session 6. Birth control and the prevention of sexually transmitted diseases

Presentation from a Planned Parenthood educator on the alternative forms of birth control, their proper use, and rates of effectiveness. Discussion of which birth control techniques the members of the group had employed in the past, what their personal preferences were, and what plans they had for use of birth control in the future.

Session 7. Open communication with sexual partners

Viewing of the movie ***Active Partners*** (Silverman and Lenz 1979) concerning a spinal injured male's relationship with his girlfriend and their sexual activities. Discussion of the obstacles to living full and rewarding sexual lives experienced by the patients as the result of their schizophrenic illness, ways of overcoming them, and the diversity of possible sexual activities for achieving pleasure.

Session 8. Human sexual response and sexual dysfunction

Presentation on the four phases of the sexual response cycle, the effects of neuroleptic medication on sexual functioning, types of dysfunction, how to tell whether the dysfunction might be medication-related, and steps to take if you suspect that the dysfunction is medication-related. Patients encouraged to ask questions.

During the group sessions, active verbal participation had to be solicited. As is typical of schizophrenic patients, most did not initiate interactions. To compensate for the passivity of the patients, the coleaders introduced exercises to facilitate sharing of experiences and exploration of attitudes. In response to questions frequently asked of the participants to elicit their views and experiences, none showed reluctance to participate. Over the course of the program, the atmosphere in the group became lighter with appropriate joking, sharing of personal sexual frustrations, and asking of questions. One patient wanted advice on how to deal with parental disapproval of his sexual behavior. Another asked whether masturbation could cause mental illness, and another whether all orgasms were the same. A patient who expressed the fear that his medication might cause birth defects was reassured to be told that this issue had been investigated and no evidence found to link neuroleptic medication with an increase in birth defects.

The patients who attended were enthusiastic about the program. Since all patients in the Aftercare Clinic are assessed every 2 weeks for level of symptomatology on the Brief Psychiatric Rating Scale (Overall and Gorham 1962), it was possible to evaluate whether the sex education program might have stimulated exacerbations or relapses. None occurred among the participants during the 4 months of the program. Data on changes in sexual knowledge and self-esteem are being analyzed for a future publication.

Conclusion

The answer to the question, "Is there sex after schizophrenia?" may depend on the availability of timely and effective intervention. To prevent deterioration of sexual functioning, some which is iatrogenically caused by medication regimens, sex education needs to be incorporated in comprehensive rehabilitation programs. While this article has discussed the negative effects of antipsychotic medication on sexual functioning, the overall effect of medication is more complex:

> the possibility that certain schizophrenic patients may function sexually better while taking neuroleptics than when they are drug-free cannot be ignored. In these patients, the facilitation of sexual functioning produced by the alleviation of psychotic anxiety, thought disorganization, fear of physical intimacy with potential sexual partners, and other symptoms that can be expected to interfere with sexual functioning may be more significant and outweigh the detrimental effects of the drugs on sexual functioning. [Nestros, Lehman, and Ban 1981, p. 436]

However, many of the negative side effects of neuroleptics on sexual functioning are dose related (Ghadirian, Chouinard, and Annable 1982). Recent findings indicate that most schizophrenic patients can be maintained on doses that are a fraction of the previously recommended levels (Marder et al. 1984). This offers hope that side effects, including sexual dysfunction, can be reduced. In addition, patients who experience sexual dysfunction on one of the neuroleptic medications may not on a different type. Thus, issues of medication-related sexual dysfunction can be addressed in rehabilitation programs without the fear that patients will discontinue their medications as a consequence of becoming better informed of their possible side effects.

Even patients who engage in autoerotic activity as their primary or sole sexual activity might be very disturbed by sexual dysfunctions during masturbation. Yet studies have found that patients usually do not bring up the subject of sexual dysfunction, even with their physicians. Mental health professionals need to become more aware of the potential side effects of antipsychotic medications on sexual functioning and routinely inquire about sexual dysfunction even in patients who are not interpersonally sexually active.

In view of the large percentage of schizophrenic patients who suffer from global deficits in sexual functioning, lectures on reproduction and birth control will not constitute effective intervention. To produce improvements in sexual functioning, many patients will require explicit information about sexual dysfunction as well as training in intimacy skills. The most effective methods of achieving these ends have yet to be determined.

For physically handicapped persons who do not have sexual partners, sexual surrogates offer specialized training in sexual functioning (Tapley 1985). Many magazines for people with physical disabilities routinely publish advertisements for sexual surrogates (e.g., the *Westchester Advocate*) who view their services in a rehabilitation context. While our program did not make use of sexual surrogates, there may be some schizophrenic patients who could benefit from the training services available from surrogates.

The question remains open as to which patients might be most likely to benefit from sex education and rehabilitation programs. Some mental health professionals have expressed the fear that the intensity of intimate sexual relationships—or orgasm itself—might precipitate exacerbation of symptoms (see Knoepfler 1982). Indeed, there are case reports of decompensation during sex therapy with schizophrenic patients (Kaplan 1983). However, exacerbation of symptoms is a risk in all types of rehabilitation with schizophrenic patients. For example, Goldberg et al. (1977) found that their intensive vocational and sociotherapy rehabilitation program precipitated relapses among some patients.

Little is known about contraindications for participation in sexual education and therapy by schizophrenic patients. While better information will become available as more attention is paid to this area of functioning, perhaps a good recommendation would be to conduct screening interviews to exclude patients who are highly symptomatic, particularly patients who are highly anxious. These were the patients who suffered the highest incidence of relapses in other rehabilitation programs (Goldberg et al. 1977). Patients who are recuperating from a recent decompensation should be excluded until their clinical condition stabilizes. Good premorbid patients who enjoyed relatively "normal" social and sexual functioning and had good intimacy skills before becoming ill would be particularly appropriate candidates for programs incorporating sexual education.

Of course, patients who show an exacerbation of symptoms during a sex rehabilitation program should also be evaluated immediately to determine whether the treatment should be postponed. Patients should be free to choose whether to participate. Some may have religious values that preclude open exploration of sexuality, and some may simply prefer an asexual existence.

Although this sex education program did not include women, studies have found that the sexual functioning of female schizophrenic patients is also frequently dysfunctional and can be impaired by antipsychotic medications (Ghadirian, Chouinard, and Annable 1982; Friedman and Harrison 1984; Raboch 1984). The most effective sex education might be achieved in groups where men and women jointly practice communication skills and explore sexual myths that interfere with their sexual functioning.

Improving the sexual functioning of schizophrenic patients is an important objective that requires sex education programs for both patients and staff members. To the detriment of their patients' well-being, mental health professionals have generally ignored the area of sexual functioning. Yet we know from work with the developmentally and physically disabled that when professionals can communicate openly and provide information about sexual issues, their patients benefit.

References

Abernethy, V.; Grunebaum, H.; Clough, L.; Hunt, B.; and Groover, B. Family planning during psychiatric hospitalization. *American Journal of Orthopsychiatry*, 46:154–162, 1976.

Akhtar, S.; Crocker, E.; Dickey, N.; Helfrich, J.; and Theuban, W. Overt sexual behavior among psychiatric inpatients. *Diseases of the Nervous System*, 38:359–361, 1977.

Akhtar, S., and Thomson, J. Schizophrenia and sexuality: A review and a report of twelve unusual cases—Part II. *Journal of Clinical Psychiatry*, 41:166–174, 1980.

Axelrod, T.; O'Brien, E.; and Hines, P. Agencies, hospital staff cooperate to develop sex education seminars. *Hospital & Community Psychiatry*, 24:137–146, 1973.

Berger, S. Trifluoperazine and haloperidol: Sources of painful ejaculation. *American Journal of Psychiatry*, 136:350, 1979.

Blair, J., and Simpson, G. Effect of antipsychotic drugs on reproductive functions. *Diseases of the Nervous System*, 27:645–647, 1966.

Bozanian, R. *Sensate Focus*. San Francisco: EDCOA Series on Sexual Counseling, 1972.

Carlson, B., and Sadoff, R. Thioridazine in schizophrenia. *Journal of the American Medical Association*, 217:1705, 1971.

de la Cruz, F., and La Veck, R., eds. *Human Sexuality and the Mentally Retarded*. New York: Brunner/Mazel, 1973.

Demetral, G. Does ignorance really produce irresponsible behavior? *Sexuality and Disability*, 4:151–160, 1981.

Erlenmeyer-Kimling, L.; Nicol, S.; Rainer, J.; and Deming, Q. Changes in fertility rates of schizophrenic patients in New York State. *American Journal of Psychiatry*, 125:916–927, 1969.

Frank, E.; Anderson, C.; and Rubinstein, D. Frequency of sexual dysfunction in "normal" couples. *New England Journal of Medicine*, 299:111–115, 1978.

Friedman, S., and Harrison, G. Sexual histories, attitudes, and behavior of schizophrenic and "normal" women. *Archives of Sexual Behavior*, 13:555–567, 1984.

George, K., and Behrendt, A. Research priorities in sex education. *Journal of Sex Education and Therapy*, 11:56–60, 1985.

Ghadirian, A.; Chouinard, G.; and Annable, L. Sexual dysfunction and plasma prolactin levels in neuroleptic-treated schizophrenic outpatients. *Journal of Nervous and Mental Disease*, 170:463–467, 1982.

Goldberg, S.; Schooler, N.; Hogarty, G.; and Roper, M. Prediction of relapse in schizophrenic outpatients treated by drug and sociotherapy. *Archives of General Psychiatry*, 34:171–184, 1977.

Golden, J. Sexual problems of cancer patients. *Medical Aspects of Human Sexuality*, 20:70–76, 1986.

Golden, J., and Golden, M. Cancer and sex. In: Vaeth, J., ed. *Frontiers of Radiation Therapy and Oncology: Body Image, Self-Esteem and Sexuality in Cancer Patients.* Vol. 14. New York: Karger, 1980. pp. 68–76.

Kaplan, H.S. *The Evaluation of Sexual Disorders*. New York: Brunner/Mazel, 1983.

Keitner, G., and Selub, S. Spontaneous ejaculation and neuroleptics. *Journal of Clinical Psychopharmacology*, 3:1, 1983.

Kinsey, A.; Pomeroy, W.; and Martin, C. *Sexual Behavior in the Human Male*. Philadelphia: W.B. Saunders, 1948.

Kirby, D. The effects of selected sexuality education programs. *Journal of Sex Education and Therapy*, 11:28–37, 1985.

Knoepfler, P. Sexuality and psychiatric disability. *Sexuality and Disability*, 5:14–27, 1982.

Kotin, J.; Wilbert, D.; Verburg, D.; and Soldinger, S. Thioridiazine and sexual dysfunction. *American Journal of Psychiatry*, 133:82–85, 1976.

Levine, S., and Yost, M. Frequency of sexual dysfunction in a general gynecological clinic: An epidemiological approach. *Archives of Sexual Behavior*, 5:229–238, 1976.

Liberman, R.; Jacobs, H.; Boone, S.; Foy, D.; Donahoe, P.; Falloon, I.; Blackwell, G.; and Wallace, C. New methods for rehabilitating chronic mental patients. In: Talbott, J., ed. *Our Patients' Future in a Changing World*. Washington, DC: American Psychiatric Press, 1986. pp. 99–130.

LoPiccolo, J., and LoPiccolo, L., eds. *Handbook of Sex Therapy*. New York: Plenum Press, 1980.

Lukianowicz, N. Sexual drive and its gratification in schizophrenia. *International Journal of Social Psychiatry*, 9:250–258, 1963.

McCary, J. Teaching the topic of human sexuality. *Teaching Psychology*, 2:16–21, 1975.

Madorsky, J., and Dixon, T. Rehabilitation aspects of human sexuality. *Western Journal of Medicine*, 139:174–176, 1983.

Marder, S.; Van Putten, T.; Mintz, J.; Labell, M.; Mckenzie, J.; and Faltico, G. Maintenance therapy in schizophrenia: New findings. In: Kane, J., ed. *Drug Maintenance Strategies in Schizophrenia.* Washington, DC: American Psychiatric Press, 1984. pp. 31–49.

Moloney, P.; Elliot, G.; and Johnson, H. Experiences with priapism. *Journal of Urology*, 116:51–53, 1976.

Mindek, L. Sex education on a psychiatric unit. *American Journal of Nursing*, 74:1865–1868, 1974.

Nestros, J.; Lehman, H.; and Ban, T. Sexual behavior of the male schizophrenic: The impact of illness and medications. *Archives of Sexual Behavior*, 10:421–442, 1981.

Overall, J., and Gorham, D. The Brief Psychiatric Rating Scale. *Psychological Reports*, 10:799–812, 1962.

Pinderhughes, C.; Grace, B.; and Reyna, L. Psychiatric disorders and sexual functioning. *American Journal of Psychiatry*, 128:96–103, 1972.

Raboch, J. The sexual development and life of female schizophrenic patients. *Archives of Sexual Behavior*, 13:341–349, 1984.

Rozan, G.; Tuchin, T.; and Kurland, M. Some implications of sexual activity for mental illness. *Mental Hygiene*, 55:318–323, 1971.

Sadow, D., and Corman, A. Teaching a human sexuality course to psychiatric patients: The process, pitfalls, and rewards. *Sexuality and Disability*, 6:47–53, 1983.

Sex Information and Education Council of the U.S. *The SIECUS Notebook*. New York: SIECUS, 1986.

Silverman, M., and Lenz, R. *Active Partners*. New York: MultiFocus, 1979.

Sokolovsky, J.; Cohen, C.; Berger, D.; and Geiger, J. Personal networks of ex-mental patients in a Manhattan S.R.O. hotel. *Human Organization*, 37:5–15, 1978.

Tapley, L. Sex surrogates and handicapped people. *Disabled USA*, 4:18–20, 1985.

Von Krafft-Ebing, R. *Textbook of Insanity*. Philadelphia: F.A. Davis, 1904.

Wasow, M. Sexuality and the institutionalized mentally ill. *Sexuality and Disability*, 3:3–15, 1980.

Withersty, D. Sexual attitudes of hospital personnel: A model for continuing education. *American Journal of Psychiatry*, 133:573–575, 1976.

Witton, K. Sexual dysfunction secondary to Mellaril. *Diseases of the Nervous System*, 23:175, 1962.

Wolfe, S., and Menninger, W. Fostering open communication about sexual concerns in a mental hospital. *Hospital & Community Psychiatry*, 24:147–150, 1973.

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Family Intervention for the Rehabilitation of Schizophrenia: Toward Protection and Coping

by Angus M. Strachan

Abstract

Recent family aftercare intervention programs for schizophrenia have been reported by groups of investigators led by Falloon, Goldstein, Hogarty and Anderson, and Leff. Despite varied methods of intervention with families, all of these programs, when combined with neuroleptic medication, have demonstrated very significant decreases in relapse rates; some have also demonstrated improvements in social and role functioning. These programs share a number of common features: Family interventions are concrete and practical rather than analytical and interpretive; education about schizophrenia is delivered in a straightforward manner; and family members are treated with respect and helped to develop coping strategies. These demonstration programs are all related to vulnerability-stress-coping-competence model of schizophrenia. It is argued that future research programs should aim at gaining a better understanding of the crucial family mediating mechanisms which protect the individual from episodes of schizophrenia.

Interest in the aftercare and rehabilitation of schizophrenics has shifted over the last decade toward a greater focus on providing family support and treatment for the families of schizophrenics. As a consequence of the movement to deinstitutionalize the mentally ill, and the sparseness of financial support for community residential and outpatient programs, the burden of dealing with schizophrenia has increasingly fallen on the shoulders of their families. Goldman and Gatozzi (1981) estimate that 60 percent of psychiatrically disabled people now live at home with other family members.

With these sociopolitical changes as background, rehabilitation workers have become increasingly interested in helping the family. In the last 10 years, there has been a tremendous change in public advocacy for the mentally ill and their families. Voluntary self-help groups and organizations have spread rapidly, foremost among them being the Alliance for the Mentally Ill, which has had a meteoric growth in membership. During the Carter years, Rosalyn Carter championed the cause of the mentally ill and brought important rehabilitation issues into the open; at the same time, the Presidential Commission on Mental Illness issued its report (Parloff et al. 1978). All these developments led to an increase in the public's awareness of the plight of the mentally ill and their families.

Meanwhile, there have been important scientific developments. The diagnosis of schizophrenia has been made more systematic and reliable with the introduction of *DSM-III* (American Psychiatric Association 1980). Surveys of the relatives of the mentally ill have provided more information about what family members want from mental health professionals (e.g., Hatfield 1978; Spaniol et al., in press). Finally, important research on the relationship between family affective climate and the course of schizophrenia (Leff and Vaughn 1985) has stimulated a series of systematic outcome studies of interventions with families (Goldstein et al. 1978; Falloon et al. 1982, 1985; Leff et al. 1982, 1983*b*, 1985; Hogarty et al. 1986). These in turn have provided more practical information on how family members can

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cope and help their mentally disabled relative. All of these scientific developments mean that, now more than ever, there is the possibility of helping family members and patients with psychoeducational family intervention programs.

Relatives' Coping and Needs

A number of investigators have surveyed relatives of the mentally ill to determine their coping patterns and their needs from mental health professionals. In some early research, Freeman and Simmons (1963) argued that relatives were most upset by severe psychiatric symptoms, because the emergence of symptoms was the best predictor of rehospitalization, which suggested that the symptoms overwhelmed relatives. Later, Creer and Wing (1974) and Doll (1976) found that family members can tolerate a surprisingly high amount of psychiatric symptomatology, but this tolerance is at the expense of a great deal of internal distress and family burden—physical, financial, and emotional.

Another focus has been on the patterns of coping used by relatives. For example, Hatfield (1978) surveyed the emotional reactions of relatives of the mentally ill and found a variety of patterns varying from resentment and frustration to sadness and denial. In a later study, Hatfield (1981) explored the coping styles of relatives. Because the sample volunteered from a local family support organization, they were of higher socioeconomic status, which may limit the generalizability of the findings. She distinguished between "effective" and "ineffective" copers. Effective copers actively sought out information, were realistic in their expectations of the patient, and were able to enjoy activities separate from the patient. Ineffective copers, by contrast, did not plan ahead but reacted only to crisis, felt unable to handle the patient, and had fewer interests that were separate from the patient. This survey approach to the study of coping has some conceptual problems (see below), but the surveys do illustrate the range of coping mechanisms used—emotional, cognitive, and behavioral—which have an impact on the patient.

Another area of interest has been what family members want from mental health professionals and what they actually receive. Creer and Wing (1974) reported that relatives wanted information and practical advice from psychiatrists and social workers but were often ignored or given very vague information. Hatfield (1981) further found that relatives perceived that the treatment offered, such as individual therapy, was not useful. Holden and Lewine (1982) reported that relatives wanted more specific information about symptoms and the effect of drugs, practical help from the treatment team about accommodation and support for the patient, and emotional support for themselves. Instead, many relatives felt that they were met with vagueness and even implied criticism. Another more recent survey of the needs and coping behaviors of the relatives of the mentally ill has been conducted by Spaniol et al. (in press) who surveyed members of the Alliance of the Mentally Ill. They found that nearly half of the relatives were dissatisfied with the mental health services they received. They wanted more support, more information and input to medication management, and more help with their own stress and coping abilities. Spaniol et al. also surveyed mental health professionals. Most believed that families are satisfied with the services provided. Spaniol et al. suggest that this gap between the views of professionals and family members indicates a lack of empathy for family members' viewpoints.

The recent family intervention programs have attempted to change this by altering professionals' responses to family members. Instead of ignoring or blaming relatives, these programs have given relatives the best information about schizophrenia available and have enlisted their aid in helping their disabled relative.

Toward an Integrative Conceptual Framework

Many of the problems in the way relatives have been treated by mental health professionals have been caused by overly simplistic models of the cause and treatment of schizophrenia. Too often, professionals adopt a one-sided view of the etiology of schizophrenia: either they assume schizophrenia is caused by biological factors that are passed on genetically and can only be cured by medication; or they assume schizophrenia is caused by psychological factors rooted in defects in the family that can only be cured by individual therapy away from the noxious family environment, or family therapy to change the interactional patterns assumed to cause or maintain schizophrenic symptomatology.

Thus, for professionals with a biological viewpoint, relatives are perceived as useful for describing symptoms and history but not useful in treatment; as a result, relatives may feel ignored. Professionals with an individual therapy viewpoint assume that it is important to keep family members away from the patient while he or she undergoes individual therapy; as a result, relatives may feel shunned. Professionals with a family therapy viewpoint assume it is important to involve family members and to change their interaction with the patient; as a result, family members may feel responsible and blamed (Terkelson 1983). Thus, any simplistic model of the cause and treatment of schizophrenia can lead to family members feeling left out or blamed.

There are other reasons why mental health professionals may be reluctant to talk with relatives about diagnosis and treatment. First, diagnoses are not always clear, and staff may fear giving relatives incorrect information. Second, professionals may fear the impact of labeling someone as "schizophrenic" (Berkowitz et al. 1984). Unfortunately, giving no information to relatives or remaining vague about diagnosis seems to compound the problem: relatives become increasingly frustrated, fearful, or guilty. Indeed, withholding diagnostic information may even reinforce the stigma staff wish to avoid. When there is doubt about the diagnosis, professionals could be clear and direct about the lack of decisiveness, educate the patient and relative about the reasons for the ambiguity, and suggest how the diagnostic decision might be clarified (e.g., more interviews, letting time pass).

Accumulated scientific developments in schizophrenia are now leading to a more sophisticated, integrated conceptual framework than these either/or models: Genetic predispositions lead to varying degrees of biological vulnerability which interact with the stresses and strains of life in causing a schizophrenic episode (Nuechterlein and Dawson 1984). Evidence for this comes from more sophisticated genetic research (Kessler 1980) as well as research sparked by the finding that antipsychotic medication was not a cure-all: While phenothiazines significantly reduced the length of hospitalization of most patients, 35 percent of patients relapsed within 2 years even though they were maintained on medication, and the psychotic symptoms of 7 percent of patients remained untouched (Leff and Wing 1971). Similarly, Hogarty, Goldberg, and Schooler (1974) found that over a 2-year period following discharge from the hospital, 30 percent of schizophrenic patients who continued on oral medication relapsed, in comparison with 68 percent of those who received a placebo. To investigate whether the 30 percent relapse rate was due to noncompliance with medication, a further study was mounted to compare long-acting injectible medication (in which compliance is assured) with oral medication. Hogarty et al. (1979) found the 2-year relapse rates to be very similar: 50 percent for the long-acting injectible; 55 percent for the oral medication. Thus, relapse on medication is not due to noncompliance.

Findings such as these led researchers to investigate stress factors that led to relapse. One body of research has suggested that for patients on medication, stressful life events play a triggering role in the onset of a schizophrenic episode (e.g., Brown and Birley 1968; Leff et al. 1983*a*). Another body of research has examined the impact of stressful family emotional environments and has found that patients who return to live with family members who show high levels of "expressed emotion" are significantly more likely than patients who return to low expressed emotion environments to have relapses of their symptoms within 9 months and at 2-year followup (Brown, Birley, and Wing 1972; Vaughn and Leff 1976; Leff and Vaughn 1981; Vaughn et al. 1984). Thus, short-term life events and more long-term family pressures are both stressors that interact with biological predispositions and the protective effect of medication in precipitating the recurrence of schizophrenic symptomatology.

Since the body of research on expressed emotion (EE) has had such an impact on the family programs that are the focus of this article, I shall make a few observations about the construct.

1. **EE is rated from an interview.** EE is measured from a semistructured interview with the relative about the patient's behavior over the previous 3 months. The interview is usually conducted shortly after the patient is hospitalized. The critical comments made about the patient are counted, and the level of emotional overinvolvement displayed toward the patient is rated from an audiotape of this interview. If the relative rates high on either or both of these measures, the relative is said to be high on expressed emotion.

2. **EE fluctuates.** The level of EE changes with the course of the illness. Thus, Brown, Birley, and Wing (1972) showed that 30 percent of relatives in a British sample were high EE at admission, and this fell to 14 percent at 9-month followup. Similarly, Kottgen et al. (1984*a*) showed that 52 percent of relatives in a German sample were high EE at admission, but only 27 percent at 6 months and 15 percent at 18 months.

3. **Relatives express EE.** Several studies in the United States and England have shown that relatives do express emotional attitudes congruently in direct interaction with the patient (Valone et al. 1983;

Miklowitz et al. 1986; Strachan, Goldstein, and Miklowitz 1986). Critical attitudes correlate with critical interaction; supportive attitudes correlate with supportive behavior.

4. **EE measured at admission predicts relapse.** Several studies have shown that EE measured at admission predicts relapse. Thus, in a British sample, schizophrenic patients who returned to homes with one or more high-EE relatives were found to have 9-month relapse rates of 51 percent whereas those who returned to homes with low EE relatives were found to have relapse rates of only 13 percent (Vaughn and Leff 1976). These results have been replicated at 2-year followup (Leff and Vaughn 1981) and in a U.S. sample (Vaughn et al. 1984). Thus, patients who return to households with low EE attitudes expressed at the time of admission are protected from relapse. That the admission rating is predictive suggests that relatives' reactions during the acute episode are good predictors of the tension level in the household after discharge.

5. **Patient disturbance correlates with EE.** Brown, Birley, and Wing (1972) did a careful analysis of the relationship between patient disturbance and EE levels. They found that EE was not correlated with symptoms, but it was highly correlated with impairment at work and disturbed behavior (.84). In a further study of 205 American and British relatives, Miklowitz et al. (1986) showed no relation between EE and chronicity.

6. **But EE predicts over and above patient disturbance.** Brown, Birley, and Wing (1972) showed that EE relates strongly to relapse (.75), and patient impairment does so but less strongly (.47). When patient impairment is partialed out, however, EE still strongly predicts relapse (.63). In other words, patient factors have little effect on relapse in comparison with the effect of EE. Thus, these data support a causal effect of family attitudes on the course of schizophrenia.

While the EE construct has been successful in its impact on the scientific and professional community, it has been something of a public relations disaster in its impact on the relatives of the mentally ill and is in danger of creating a backlash if not properly understood. It is unfortunate that the name of the construct stresses the negative instead of emphasizing positive qualities of relatives. As a result, some relatives feel that, yet again, scientists and professionals are blaming them. It is important to note, however, that many relatives have low EE ratings and therefore have a positive effect on the course of the illness. The EE research does not indict all relatives but rather suggests that some emotional environments provide better protection of the patient than others. Further, it is implied that family members can control their attitudes and behavior, and can change the way they express themselves, thus providing an emotional environment that could be conducive to the protection of the patient. The conceptual framework can be expanded to assume that the family environment can be a potential stressor, but it can also provide protection and "buffer" the schizophrenic against outside stressors.

The final aspect of the framework is that many schizophrenic patients are deficient in the coping skills required to deal with situations brought on by life events or stressful family environments. They may therefore experience greater distress than other people because they lack the coping abilities to solve problems and enlist social supports (Lukoff et al. 1984). It is important to make a conceptual distinction between coping—the process of reacting to events—and the outcome of that coping; that is, how successful the coping is in affecting the patient's actual social competence (Folkman et al. 1986). As noted above, some previous investigators have confounded these two aspects of coping when examining coping by relatives. Current work is investigating the relationship between relatives' EE and their coping styles (Strachan and Dunkel-Schetter 1985).

In summary, a more integrative framework than the simplistic either/or model is one which assumes that a genetic *vulnerability* interacts with short-term life events and long-term family *stressors* in causing schizophrenic episodes. Further, families can create an emotional atmosphere in which conflict is resolved; this atmosphere reduces the impact of any family stress, *protects* patients against external life events, and enables the patient to improve *coping* skills and achieve a higher level of social *competence*.

Early Family Intervention With Schizophrenia

Before systematic studies of family intervention with schizophrenia, psychoanalysts such as Federn (1952) thought that psychodynamic individual psychotherapy could cure schizophrenia. Later, after the discovery of phenothiazines, other psychoanalysts thought that psychoanalytic psychotherapy in combination with neuroleptics could improve the outcome of patients with schizophrenia. However, a major study reported by May and Tuma (1976) has not supported this contention. Random assignment of patients to drug treatment, psychotherapy, drug treatment plus psychotherapy, milieu therapy, or electroconvulsive therapy showed that an ego-supportive, reality-oriented psychodynamic therapy was neither as effective as medication, nor added to its effectiveness. More recently, a major study by John Gunderson and the Boston group (Gunderson et al. 1984; Stanton et al. 1984) has shown that there were minimal outcome differences between exploratory insight-oriented therapy and reality-adaptive, supportive therapy, both given to individuals in addition to maintenance pharmacotherapy.

Subsequently, family systems theory emerged, principally from observations of the families of schizophrenics viewed through lenses tinted by the historical zeitgeist of interest in the social, economic, or ecological *context* of a phenomenon. Thus, for example, Laing and Esterson (1964) suggested that people became schizophrenic because their families "mystified" them with their confusing communication. Esterson, Cooper, and Laing (1965) reported the results of therapy aimed at families of schizophrenics. They emphasized the importance of taking psychotic symptoms seriously and of understanding the meaning of hallucinations and delusions in relation to family communication. They took an existential approach with the patient and a demystifying approach with the family, trying to unravel the disguised family communication. Although this was not a randomized study, the authors compared 1-year rehospitalization rates in their sample of 42 families with rehospitalization rates in a matched sample of 374 provided by the MRC Social Psychiatry Unit in London. The rehospitalization rate in the treatment group was 17 percent compared with 52 percent in the comparison group, a highly significant difference.

Another early family therapist, Bowen (1978) described work in which he hospitalized whole families of schizophrenic patients for observation and treatment. Bowen worked intensively with such families attempting to "differentiate" family members from "the undifferentiated family ego mass." This work was "intensive" in its use of interpretation and insight.

In the 1960s and 1970s, the work of Weakland et al. (1974) and of Palazzoli et al. (1978) focused on the communication of families in "schizophrenic transition." They saw the psychotic behavior of the identified patient as a symptom of a disturbed pattern of transactions in the family. Weakland et al. (1974) described the results of 96 cases (only some containing schizophrenics) who were seen for an average of seven sessions and reported that 72 percent were significantly improved. Palazzoli et al. (1978) also claimed success for their methods in which they saw families of schizophrenics about once a month and focused on "positive connotation" of the psychotic behavior and the prescription of therapeutic paradoxes.

At about the same time, Liberman and his colleagues were developing behavioral approaches to the families of schizophrenics. After early case studies (Liberman 1970, 1972; Liberman, DeRisi, and King 1973; Falloon and Liberman 1983), Falloon, Liberman, and colleagues developed a comprehensive behavioral approach to a skills-based intervention focused on changing the inappropriate communication of feelings (Falloon et al. 1981). These interventions also included social skills training for the patient. This was followed by a randomized study of social skills training and behavioral family therapy in comparison with "holistic therapy" and supportive family therapy (Snyder and Liberman 1981; Wallace and Liberman 1985). Significant differences were found in the social functioning of patients, favoring the skills-based therapies.

Unfortunately, while all of the above approaches represent intriguing methods of inducing change in the family system, none employed randomized assignment designs to compare outcomes of family therapy in aftercare. The more recent studies to be described here differ from most of these in that their major focus is on improving the aftercare of patients discharged from the hospital by involving the family in treatment. This new focus reflects two trends, one theoretical and one pragmatic. Theoretically, these programs rely on a diathesis-stress model of schizophrenia. Pragmatically, the focus of therapy has moved from "curing" the patient of their psychotic symptoms to seeing what impact family therapy can have on their social functioning and resistance to stress once the major tranquilizers have reduced acute psychotic symptoms.

Systematic Family Treatment Studies of Schizophrenia

This review focuses on four recent systematic studies of the impact of family treatment on schizophrenia (see table 1). Each study had a randomized experimental design with at least one comparison condition that included little or no family contact. Each study also had rigorous procedures for selecting participants according to research diagnostic criteria. The timing and pacing of treatment varied from study to study, but all provided family treatment in the months immediately following hospital discharge. Most patients had had previous admissions, except in Goldstein's study, where most were first admissions.

Table 2 shows relapse rates for the family therapy condition versus one of the comparison groups. All four studies had a strong positive impact in decreasing the relapse rates at 6 or 9 months (see figure 1).

Crisis-Oriented Family Therapy. Goldstein et al. (1978) reported the first random assignment study of family therapy as used in aftercare. They examined the impact of a short-term, crisis-oriented family therapy aftercare program in combination with various levels of drug dosage.

The sample consisted of 96 first and second admissions (mean age 23 years) who received independent diagnoses of schizophrenia from ratings on the New Haven Schizophrenia Index. Of these, 69 percent were first admissions and 31 percent were second admissions who had all been admitted within the year.

Patients were assigned to one of four conditions by a stratified random method. Their premorbid sociosexual adjustment was measured on the UCLA Social Adjustment Scale, and median splits were used to stratify males and females separately into good and poor premorbid groups. Then patients were randomly assigned to cells in a 2 × 2 factorial design. One factor was the presence or absence of family therapy. The other factor was the level of medication. All patients were given long-acting injections of phenothiazines. The low-dose group received what was judged to be a

Table 1. Recent family therapy programs with schizophrenia

Study	Comparison groups	Timing of treatment[1]	Place of meetings	Patients (*n*)	Mean age	% 1st admissions
Goldstein et al. (1978)	2 x 2 factorial: low and medium drug dose + family crisis therapy or not	6 weekly family meetings	Clinic	96	23	69
Leff et al. (1982, 1985)	Relative education + group vs. standard aftercare	18 biweekly-relatives' groups + 6 family meetings (average)	Group at clinic; family at home	24	35	30
Falloon et al. (1982, 1985)	Family education + behavioral family therapy vs. individual case management	Weekly for 3 months, biweekly for 3 months monthly for 3 months	Home	36	25	36
Hogarty et al. (1986)	2 x 2 factorial: relative education and structural family therapy or not; + social skills training or not	Twice a week during hospitalization, then every 2 or 3 weeks	Clinic	103	27	23

[1] In the first 9 months after discharge; family treatment was continued after 9 months for the 3 later programs.

Table 2. Relapse rates for 4 randomized studies of family therapy with schizophrenics

Study	1st followup	% Relapse in family therapy group	% Relapse in comparison group	Comparison group
Goldstein et al. (1978)	6 months	0	48	Low dose, no family therapy
Leff et al. (1982)[1]	9 months	8	50	Normal outpatient care
Falloon et al. (1982)[2]	9 months	6	44	Individual education + case management
Hogarty et al. (1986)	9 months	9	28	Individual treatment

[1] At 2 years, relapse rates were 33% for family cases compared with 75% for individual cases.

[2] At 2 years, relapse rates were 17% for family cases compared with 83% for individual cases.

minimally therapeutic dose of fluphenazine (0.25 ml); the moderate dose group received 1 ml.

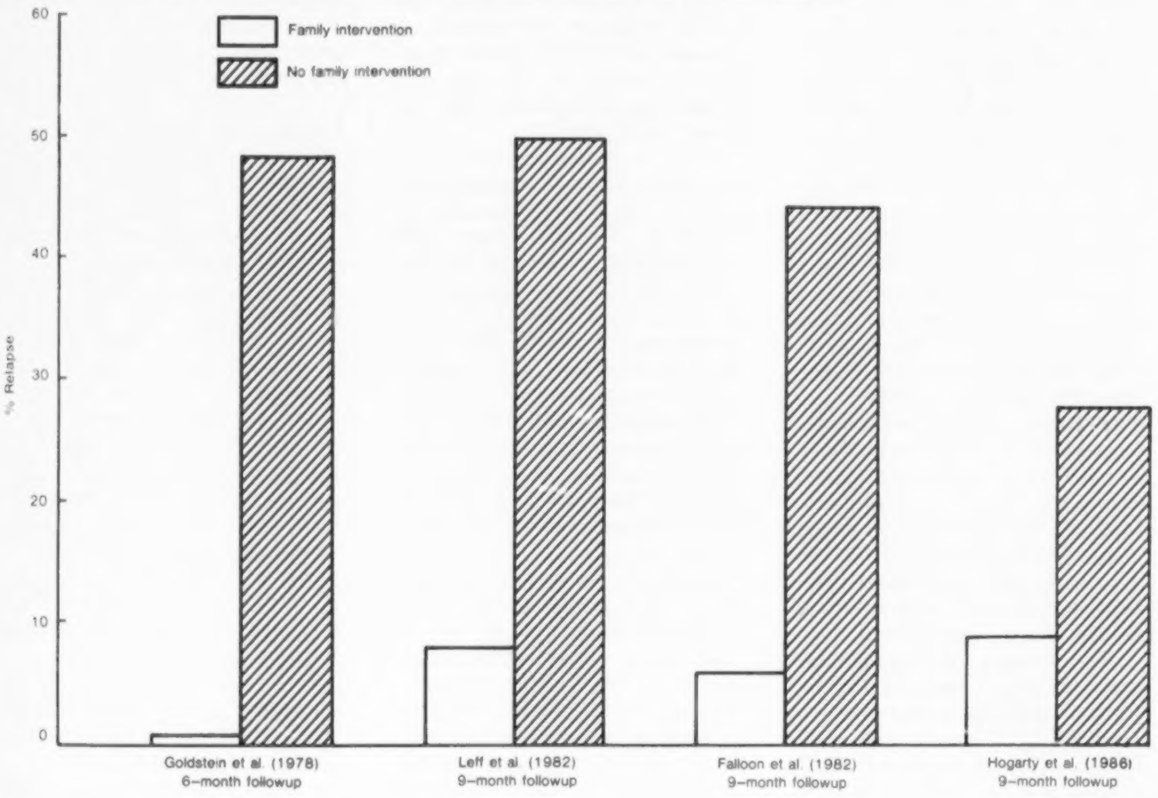
The family therapy was of short duration and very concrete. Based on crisis intervention theory, the therapy was designed to help the family understand the events around the psychosis and to improve future coping. Family therapy sessions with two co-therapists were held weekly in the outpatient clinic for the 6 weeks following discharge. In the first stage of treatment, each family member, including the patient, was asked about the events before and during the patient's psychotic break. Many mental health professionals avoid dealing with this event, letting patient and relatives "seal over" emotions or feel hidden shame about the psychotic break. Goldstein and Kopeikin (1981) report that both patient and relatives experience a great deal of relief in talking about the breakdown, and they also become aware of its serious nature. During this initial discussion, the therapists help the family identify stressful events that preceded the breakdown and make links between these precipitating events and the breakdown.

The second stage of therapy identified the two or three situations that were currently most stressful to the patient. The third stage developed strategies for avoiding or coping with these stressful situations. During the week, the family carried out these methods of coping and reported back at the next meeting. In the final stage, plans were made to anticipate future crises and develop stress prevention and coping mechanisms in advance of such crises. Of major importance was the discussion of the possible emergence of psychiatric symptoms. Other topics might be resuming employment, making friends, or future living arrangements. In summary, the family meetings revolved around identifying current potential stressors and planning coping mechanisms for the patient and relatives to use.

Results. At the end of 6 weeks of treatment, 10 of the 96 patients had deteriorated clinically so that they had to be rehospitalized or have their medication increased dramatically. This was an overall relapse rate of only 10 percent, considerably less than the 31 percent found previously. At 6-month followup, the rate of relapse was 0 percent for the moderate dose-family therapy group but 48 percent for the low dose-no therapy group. The other two groups were in between: Moderate dose-no therapy had a relapse rate of 14 percent; low dose-no therapy was 21 percent. There was a significant effect of drug dosage ($p < .01$), and family therapy ($p < .05$), and the extreme groups differed very significantly ($p < .001$).

The symptomatic status of the patient was examined using behavior ratings on Overall and Gorham's (1962) Brief Psychiatric Rating Scale (BPRS). The most significant effects were for the family therapy: At 6 weeks, family therapy had a significant overall effect on symptoms, particularly on social withdrawal and

Figure 1. Relapse rates from 4 studies of family therapy for schizophrenia[1,2]



[1] All schizophrenic patients were on continuous medication.

[2] Comparison groups are specified in table 2.

blunted affect. Thus, the family therapy seemed to prevent the patient from withdrawing socially and becoming less emotionally responsive. At 6 months, only withdrawal was significantly related to treatment group: The effect of therapy at this point was only found in the group with the higher drug dosage.

Overall, it can be concluded that the 6 weeks of moderate drug dosage and short-term family therapy had significant effects on relapse rates and social withdrawal up to the 6-month followup. A long-term followup was conducted on those patients still in the area after 3–6 years. There were no longer any effects. In other words, the effect of the 6-week intervention ceased to be significant as its impact diminished in comparison with that of later events.

Since the publication of this brief family intervention project, the results of two projects with smaller samples but longer-term interventions have been published.

Education, Relatives' Support Group, and Family Therapy. First there is the work of Leff et al. (1982, 1983*b*, 1985), which grew out of the research relating expressed emotion (EE) and face-to-face (FF) contact to relapse. To examine whether this correlation was causal, they decided to "manipulate experimentally" relatives' EE and FF and examine the effect on relapse.

Consecutive admissions to several London hospitals were screened for the presence of the following: (1) continuous residence with relatives for at least 3 months before admission; (2) a diagnosis of schizophrenia according to the Present State Examination (PSE) criteria; and

(3) a high level of FF, more than 35 hours per week. Subsequently, each relative with high FF was given the Camberwell Family Interview (CFI) and their EE level assessed. Twenty-four high EE families were randomly assigned to an experimental treatment group or a comparison treatment-as-usual group. Both groups received identical assessment at intake and 9-month followup: a PSE with the patient; a CFI and a structured interview about their knowledge of schizophrenia with each relative. Most patients were on long-acting injections of phenothiazines. Thirty percent were first admissions, and the sample had a mean age of 35. This was therefore an older and more chronic group than in the Goldstein study. Sixteen low EE families were also randomly assigned to treatment or comparison groups, but data were not reported because of dropouts from these groups.

The intervention package had three components (Berkowitz et al. 1981). First, an education program was given to relatives in their homes. This consisted of four short lectures covering possible causes, symptoms, likely course, and treatment. Relatives were encouraged to ask questions. The patient's condition was explicitly described as "schizophrenia."

The second component was a relatives' group composed of both high and low EE relatives. The group was led by two professionals and met every 2 weeks. It had been observed previously that low EE relatives had more effective coping mechanisms for dealing with their schizophrenic relative which had the dual effect of decreasing pressure on the patient and eliciting more support for themselves. Thus, the initial hope was that low EE coping mechanisms would rub off on the high EE relatives. This turned out to be overly optimistic because most of the low EE relatives simply stopped attending. In response, the group leaders became more active in helping the relatives find better coping mechanisms.

The third component, family meetings in the home, was developed to include the patient and to deal with family dynamics. The approach was pragmatic, varied, and flexible.

Most relatives in the treatment group received the educational component and attended the group about once a month. Every family had at least one family meeting at home with an average of five. Nine months after discharge, the PSE and CFIs were repeated. A further followup PSE on those who had not already relapsed was repeated at 2 years.

Results. Relapse was defined as the reappearance of schizophrenic symptoms that did not exist at discharge, or the exacerbation of symptoms that had stabilized at discharge (Leff et al. 1982, 1983*b*). At 9-month followup, only 1 out of 12 patients in the treatment group (8 percent) had relapsed compared with 6 out of 12 in the treatment-as-usual comparison group (50 percent), a significant difference (Fisher's exact test, $p = .03$). During this time, two patients stopped taking medication, and one of the two relapsed. At 2-year followup, (Leff et al. 1985), 4 out of the 12 in the treatment group (33 percent) had relapsed compared with 9 out of 12 in the comparison group (75 percent), again a significant difference (Fisher's exact test, $p = .04$).

However, five patients stopped taking medication during the 2 years, two in the treatment group and three in the comparison group. To analyze the effect of treatment without the confounding effect of medication compliance, the analysis was repeated without these subjects. Only 20 percent relapsed in the treatment group compared with 78 percent in the comparison group ($p = .02$).

Further analyses examined the impact of the social interventions on EE, the focus of treatment and an indicator of the family emotional environment. The CFI was administered at admission and at 9-month followup. A reanalysis of the data (Strachan, personal communication), revealed that for the 11 treatment families who were initially very critical and who had both initial and followup data, the mean number of critical comments decreased from 17.6 to 6.9; for the six comparison patients with both sets of data, the mean number of critical comments decreased from 13.3 to 10.7. The ranked change scores were highly significantly different ($F = 9.6$, $df = 3/12$, $p < .001$). Changes in ratings of emotional overinvolvement were not significant, however.

Berkowitz et al. (1984) examined the impact of the education sessions on knowledge about schizophrenia. They found that high EE attitudes were associated with disbelief that an illness caused the problems, and reluctance to change from a blaming stance, whereas relatives with low EE attitudes were less blaming and accepted the notion of an illness more easily.

In overview, these data suggest that the combination of medication and social interventions with the families of schizophrenics has a significant effect on the likelihood of relapse at both 9-month and 2-year followups. Further, the analysis of EE (and knowledge) data suggests that this success is associated with relatives developing less critical attitudes toward the patient, which is probably related to their changing attributions about the causes of the patient's behavior, the group support, the direct education, and the clear focus in family meetings on changing the way emotion is expressed.

Behavioral Problem-Solving Family Management. The program of Falloon et al. (1982, 1985) differed from the previous two in several respects. First, medication was given orally and flexibly, at the lowest level possible. This design followed findings that long-acting intramuscular medication may impair social functioning more than oral medication (Falloon, Watt, and Shepherd 1978), despite similar effects on relapse rates. Furthermore, it was assumed that flexible, minimal dosages would mean better responses to psychosocial intervention.

Second, a short educational program was given to each family. In contrast to the Leff program, however, the patient was included and made an expert on his or her own symptoms by the clinician, thus unbalancing the expected status hierarchy in the family!

Thirdly, the main focus of intervention was behavioral family management with the whole family. On the basis of previous work by Falloon and Liberman (Liberman 1970; Falloon et al. 1981; Falloon and Liberman 1983), the model assumes that the family is the most effective resource for community rehabilitation. Further, the goal of family management is to improve the family's ability to shield the patient from stress by enhancing family problem-solving and communication skills through structured problem-solving tasks. All family sessions were conducted in the home for the first 9 months of treatment. The first two sessions were spent educating the patient and family about the nature of schizophrenia and the rationale for combining drug and psychosocial interventions. Thereafter, family meetings were held weekly for the first 3 months, biweekly for the next 3 months, and monthly for the next 3 months. After that, families were invited to multifamily groups held monthly at the hospital.

The family meetings focused on problem solving, and thus the family was trained to pinpoint a problem, generate solutions, evaluate potential consequences, agree on a best strategy, implement the strategy, and then review results. The therapist taught the model and then coached the family, expecting them to take responsibility for problem solving between meetings. The therapist also focused on communication training, encouraging the family to communicate clearly by active listening, being specific about requests, and expressing positive and negative feelings clearly. Falloon et al. point out that their aim was not to stifle emotion but to change its expression.

Behavioral family management was compared with individual case management of the patient. Therapists saw an equal number of patients in family and individual treatment. The individual program consisted of education about schizophrenia followed by supportive therapy focused on the problems of everyday living. Thirty-six schizophrenic patients were randomly assigned to individual or family management. As in the Leff program, all patients were living with families with high EE attitudes or significant family tension. The sample had a mean age of 25 years, and 36 percent were first admissions.

Results. The assessment of relapse was done systematically with a variety of measures of exacerbation: PSE interviews were given at 0, 9, and 24 months; and monthly ratings were made of key schizophrenic symptoms and global BPRS scores. At 9-month followup, only 1 out of 18 (6 percent) in the family management group had had a major exacerbation of schizophrenia compared with 8 out of 18 (44 percent) in the individual treatment group, a highly significant difference (Fisher's exact test, $p < .01$). At the 2-year followup, only 3 out of 18 (17 percent) in the family management group had had a major exacerbation compared with 15 out of 18 (83 percent) in the individual management group, again a highly significant difference (Fisher's exact test, $p < .0001$). This higher rate of exacerbations was also associated with longer and more numerous hospital admissions.

A feature of Falloon's study was the comprehensive assessment of change, particularly in social and family functioning. Whereas the previous studies focused on psychopathology, Falloon, Boyd, and McGill (1984) report outcome data on the social and economic benefits of their program. Multiple outcome measures were used, including the Social Behavior Assessment Schedule (Platt et al. 1980), which assesses both subjective and objective measures of social functioning. Data showed enhanced functioning in the treatment group both at 9 and 24 months, as indicated by fewer hospitalizations, less behavioral disturbance, better reported family relationships, and more friends. Informants reported greater satisfaction with the patients' social behavior and less family burden. Over the 24 months, family cases were involved in full- or part-time work or education for an average of 12.6 months compared with 7.2 months for individual cases.

Examining direct interactions between family members, analyses were also performed to investigate changes in family communication and problem solving. Doane et al. (1985) showed that the total number of critical and intrusive remarks made by parents was significantly lower after 3 months of family therapy compared with individual treatment. Further, parental criticism and intrusiveness was associated with relapse in the individual treatment. Using a coding scheme for recording instances in which family members adopted recommended problem-solving strategies, Doane et al. (1985) showed that decreases in critical or intrusive remarks were associated with increases in problem-solving statements. It was concluded that problem-solving behavior had replaced negative affective interaction, although it could not be concluded what happened first.

A cost-benefit and cost-effectiveness analysis was performed to assess whether the home-based family management was more or less expensive (Cardin, McGill, and Falloon 1986). All direct and indirect costs of community management to the patients, families, health, welfare, and community agencies were recorded throughout the 2 years. Cost-benefit analysis of the 12-month data showed that total costs for family management were 19 percent less than those of individual management, despite the added expense of time to travel to the patients' homes for the family sessions. The greater cost-effectiveness of family management was mainly due to the increased need for crisis intervention and hospitalization for the individually managed patients. The cost-benefit analysis weighed the effectiveness of treatment on clinical, social, and family functioning against the total cost of services. Family management showed double the effectiveness at 58 percent less cost.

Thus, this program seems to have been extremely effective in reducing exacerbations of schizophrenia, and in improving social and role functioning. Further, these gains were made at lower cost to society than traditional individual case management. There was some evidence that individually treated patients complied with medication less, but this effect was not strong enough to explain the large differences in the rates of exacerbation. Interestingly, there was a (nonsignificant) trend for family cases to have lower doses of neuroleptics prescribed and ingested than individual cases. In conclusion, behavioral family management may allow lower drug dosages and thus reduce side effects and improve social functioning.

Family Psychoeducation and Survival Skills. The results of the final study to be reviewed here are just emerging. Hogarty et al. (1986) have reported the 1-year effects of a large study of family treatment and social skills training in conjunction with pharmacotherapy. After hospital admission, 103 patients living with high-EE relatives who met Research Diagnostic Criteria for schizophrenia or schizoaffective disorder were randomly assigned to four conditions of a 2-year aftercare study. Twenty-three percent of the patients were first admissions. Their mean age was 27 years.

The study examined the additive effect of two different treatment methods to maintenance antipsychotic medication: psychoeducational family treatment and individually oriented social skills training. The four conditions of the study were thus (1) medication only; (2) family treatment and medication; (3) social skills training and medication; and (4) family treatment, social skills training, and medication.

The family treatment (Anderson, Hogarty, and Reiss 1980; Anderson, Reiss, and Hogarty 1986) in this study was predicated on two assumptions: that the emotionally laden atmosphere in the home should be reduced and that reasonable expectations of the patient's performance should be maintained. The family treatment provided family members with education about schizophrenia and strategies for managing the patient, with the immediate goal of forming an alliance with the family.

The family treatment has four phases: In phase 1, the emphasis is on forming a good working alliance with the family and involves two sessions per week during the brief hospitalization or acute phase. During these sessions with the relatives, the therapist connects with the family members and enlists their cooperation with the program. The therapist is empathic and, through discussion of the recent episode, aims to decrease the guilt, frustration, and anxiety often experienced by relatives. Additionally, the family therapist acts as a mediator with the hospital and treatment system.

Phase 2 of treatment is unique to this program. Psychoeducation about schizophrenia is provided in a 1-day "survival skills" workshop in which four or five families with a recently hospitalized member meet to receive information about schizophrenia, concrete management suggestions, and encouragement to have lives of their own. An important aspect of this workshop is the informal interaction with the other people in similar predicaments which promotes togetherness and the beginning of a support network.

The next stage of the program involves the whole family including the patient meeting every 2 weeks for

6 months. The basic goal of this phase is to apply the general strategies from the survival skills workshop to the particular family. This involves reinforcing the boundaries between the patient and other family members and building connections with friends and the community. The patient is encouraged to resume responsibility gradually. Often relatives expect patients to return to work quickly and to live on their own. The family therapist encourages relatives to lower their expectations initially, to be patient, and to help the patient accomplish small steps toward independence.

In the final phase of treatment, the family members make one of two choices. Either they can continue in family therapy to improve family communication and to deal with unresolved conflict, or they can move to decreased involvement with the treatment team. Sessions become decreasingly frequent and serve to reinforce the lessons learned earlier. The clinical team remains available to families for maintenance sessions or crisis management. Thus, the family treatment is most intensive for the first year after the acute episode, becoming less frequent thereafter (Anderson, Reiss, and Hogarty 1986).

The social skills training used with the patients was similar to previous efforts with schizophrenics (Wallace et al. 1980; Liberman et al. 1985) but focused in particular on improving the patient's social skills in dealing with family members. Initially, patients were taught (through a process of instruction, modeling, role play, feedback, and assigned homework) to avoid conflict and criticism of family members, the goal being to prevent the patient from eliciting high EE behaviors and instead to elicit favorable responses from the family. Thus, this approach attempted to change the family emotional environment by working with the patient only. The comparison "no treatment" condition was a combination of medication and biweekly individual supportive therapy.

For inclusion in the study, patients had to live with at least one high EE relative. Families were rapidly inducted into treatment within a week or two of hospitalization. Patients were said to have relapsed if there was a return of schizophrenic symptoms that were not present at discharge, or if there was an exacerbation of symptoms persisting at discharge. Of the 103 patients, 90 stayed in treatment, 11 relapsed during the early process of engagement, and 2 received only minimal treatment. These latter 13 cases were designated as "partial treatment takers." Relapse was monitored for 12 months, at the end of which a repeat CFI was obtained.

Results. At 1-year followup, the medication comparison group had a relapse rate of 41 percent (consistent with previous findings) whereas the group that received both family and social skills treatments had a relapse rate of 0 percent. The group that received only family treatment had a relapse rate of 1 percent, and the group that received only social skills training had a relapse rate of 20 percent. The main effects of family treatment and social skills training were both statistically significant, but the interaction was not, suggesting that the effects were additive. (Comparable figures at 9 months were 28, 0, 9, and 10 percent, respectively.) Similar effects were found when partial takers were included. When only those patients who were consistently compliant in taking medication were considered, family treatment had a significant effect, and the effect of social skills training was only marginally significant. This appeared to be due to a high rate of noncompliance in the drug comparison group (24 percent) compared with 5–10 percent in the three treatment groups. Sixty-six percent of these noncompliers relapsed, reducing the effect sizes.

A feature of this study was the careful measurement and analysis of EE at intake and 1-year followup. All patients at intake lived within high-EE households. The most interesting findings were (1) that there were *no* patient relapses in any household that changed from high to low EE, independent of treatment condition; and (2) that in households that stayed high in EE only the combination of family treatment and social skills training prevented relapse. In fact, there were no relapses in this group. These striking results suggest that reductions in family tensions are associated with improvements in patient outcome, however the reductions are achieved. Further, family treatment may be the most effective in this regard because changes in EE level were most pronounced in the two family therapy conditions.

This study also supports data from an earlier design by Liberman, Falloon, and Aitchison (1984). They used a brief multifamily behavioral family treatment in conjunction with social skills training with 14 patients from high EE families. In comparison with a contrast group from a similar population that received no treatment, 9-month relapse rates were 21 percent versus 56 percent, with a concomitant drop in EE.

In conclusion, the Hogarty study provides important information on combining medication, family treatment, and individual social skills training. Note that the combination of these three treatments leads to the best outcome. Of particular pertinence is the fact that the individual treatment has an effect additive to the other forms of treatment.

Discussion

The four programs described above have different emphases: Goldstein et al. stressed family crisis intervention but did not offer psychoeducation; Leff et al. stressed multifamily group support and skill-sharing; Falloon et al. stressed problem-solving and communication training; and Hogarty et al. stressed survival skills and management of the patient.

Nevertheless, these programs share a number of common features that may explain their greater effectiveness than previous psychosocial programs. Some of these common features are so-called "nonspecific" therapy factors that transcend particular techniques (Gurman and Razin 1977). Others are specific techniques.

Family Therapy Techniques. In general, the therapists in all four programs were warm, empathic, and concrete in their focus on helpful management strategies. None of the programs used traditional psychodynamic (intensive, exploratory, uncovering, insight-oriented) therapy with families (e.g., Boszormenyi-Nagy 1965). Such intensive family therapy can be overstimulating for patients and can lead to psychotic regression. For example, depth interpretations of internal motivations can lead to patients' thinking the therapist can read their minds and can play into delusional thinking. Thus, Kottgen et al. (1984*b*) describe a program in which patients and relatives met separately in analytically oriented psychotherapy groups. These groups, which used an uncovering, insight-oriented approach, did not reduce the likelihood of patient relapse, presumably because they were overstimulating.

In contrast, the four successful programs used some or all of the following processes:

Connecting. Each program had ways of making the patient and family members feel that their point of view was valued and important.

Further, the CFI and the PSE are research interviews with a model of respectful inquiry: Relatives are asked to describe in detail the problems that *they* have experienced in relation to the patient's behavior. The goal of the CFI is to obtain information about the relatives' emotional reactions, how they coped, and how they reacted. Relatives often report that the interview itself was helpful.

Investigators from three psychoeducational programs report that the educational sessions in particular help with the connection process. At last, the relatives are getting some answers to their questions! Cozolino and Nuechterlein (1986) report that even a single, limited educational session had salutary effects on the therapeutic relationship and alliance between relatives and professionals.

Finally, because of the legal requirement for obtaining informed consent, clear contracts were negotiated at the outset with each family. The aims, structure, and length of the program were explained fully, including randomization to treatment conditions and followup procedures.

Reframing. Relatives often feel that their contribution to helping the patient is not appreciated by hospital staff. They may also feel blamed for the patient's problems (especially if they hear the phrase "family therapy"). Thus, they may react defensively and reject any attempt at "treatment." Further, their guilt and anxiety can lead them to behave toward the patient in counterproductive ways.

Therefore, it is often useful to talk not of "family therapy" but of "family meetings," which does not imply there is something wrong with family members. Hogarty et al. use the phrase "family treatment." Furthermore, relatives can often be engaged in treatment by appealing to their altruism and asking them to come "so we can help you help [the patient]."

Once the joining process has taken place, the intentions of relatives and patient can be positively reframed. Depth interpretations that can imply criticism and induce guilt are avoided. Rather, every effort is made to see the problem from each person's point of view and to reframe intentions positively. Thus, parents can be described as making their best efforts to be helpful, as being "protective" rather than "emotionally overinvolved," as "encouraging independence" rather than being "hostile and critical."

Focus on current transactions. The focus of treatment in all these programs was on the current transactions between family members. Therapists focused neither on the historical development of the family nor on the development of personality, but on present-day coping, on how the patient and relatives deal with stress, and how they react to each other. Very quickly, the focus was moved away from either "What is wrong with the patient and how can he or she be cured?" at one extreme, or "What is wrong with the relatives and how can they be tamed?" at the other extreme, to an examination of family interaction and coping to see if there are alternative methods of coping that can be used to deal more effectively with the inevitable stresses of life.

Focus on communication. All four programs focused on having clear communication between family members and improving the family's emotional atmosphere. However, there were different emphases. Goldstein et al., for example, focused on the patient and relatives' communication about what happened during the acute episode, what experiences the patient had (often never revealed to family members), the reactions of relatives, and the meaning of the precipitant to the family. The other three programs focused more explicitly on reducing the emotional temperature in these families, which were selected as high-risk environments because relatives were rated as being high on EE. Although EE is measured as an indirect response to the patient, it has been shown to relate to the quality of emotional interaction in the family (Strachan, Goldstein, and Miklowitz 1986). The programs of Leff and Hogarty focused explicitly on reducing high EE in the relatives. In contrast, Falloon et al. did not focus explicitly on the goal of reducing EE. Instead, they assumed that high EE was an index of emotional distress in a relative, which was a reaction to lack of conflict resolution. Relatives were not expected to reduce the intensity of their emotional expression but rather to express their feelings more clearly, differentiating their personal reactions from other people's internal motivations. In fact, even though there was not a focus on reducing emotional expression, the redirection of expression led to a reduction in the number of criticisms and intrusions directed at the patient in direct interaction with treatment (Doane et al. 1985; Goldstein and Strachan 1986).

Focus on concrete problem solving. Falloon et al. and Goldstein et al. focused most explicitly on structured techniques for problem solving. However, the other two programs also focused on practical issues of management and concrete coping strategies. Of all the family programs, Falloon et al. concentrated most on specific and directive skill-building, both in communication and problem solving. It could be that such a behavioral skill-building focus may be particularly beneficial to schizophrenic patients who have deficits in social skills and result in the more favorable social and occupational rehabilitation outcomes reported by Falloon, Boyd, and McGill (1985).

Psychoeducation. Each program stressed the importance of facing the psychotic experience directly, although each treated this slightly differently: Goldstein focused on understanding the precipitants of the psychotic episode without talking about schizophrenia explicitly; Falloon, Anderson, and Leff all had structured education programs about schizophrenia that were given to families; Falloon and Goldstein included the patient, whereas Anderson and Leff did not.

Interestingly, the education, although short, seems to be very powerful and helpful, particularly for relatives of people who have had several hospitalizations over the years (Cozolino and Nuechterlein 1986). They may have been asking what was wrong over the years and received only vague answers from mental health professionals. They may have recognized that medication helped but have had no idea what it did. They may have felt guilty, particularly if ward staff had told them to stay away because their visits upset the patient. And, perhaps most important, the fantasy may have developed that there was something terribly wrong, fueled by the vague answers of mental health professionals, just as people guess they have cancer from the growing wall of silence around them: "Maybe my relative is mad and will only get worse and worse. Maybe he [or she] has even got the dread disease 'schizophrenia.' "

The educational program seems to burst this bubble and leads to relief instead of despair. Knowing at last that their relative has schizophrenia and hearing explanations of its likely course and response to treatment (which most relatives experience as better than their worst fantasies), they tend to be much more optimistic (Cozolino and Nuechterlein 1986). Paradoxically, they feel more in control. They are more realistic and now have a better idea of what they can expect and what is and isn't possible for them to do to help.

Drug Compliance. It has been argued that family therapy may have a beneficial effect on relapse by increasing compliance with medication. Falloon et al. (1982) noted that more reliable tablet-taking was found in their family treatment group and suggested that even if family treatment only leads to greater compliance with medication and hence less symptom exacerbation, the family treatment approach is indeed a step forward. However, in both the Leff and Goldstein studies, medication compliance was excellent in both the treatment and comparison groups, and the family approach still reduced relapse rates. In the study of Hogarty et al., the effects of family treatment were apparent whether all patients were considered or just those who were compliant with medication. Thus, family treatment, while improving medication compliance, has an effect beyond that of increased compliance.

Implication for a Conceptual Framework for Schizophrenia. It is assumed that schizophrenics have a biopsychological *vulnerability* that makes them particularly susceptible to *stress* and that reductions in external stress and increases in personal *coping* can lead to increased *competence*. The vulnerability of schizophrenic patients seems to be manifest as a sensitivity to their social environment (even when they are not experiencing symptoms) such that either an overstimulating or an understimulating social environment may lead to a return of symptoms and increased arousal (Wing and Brown 1970).

The focus of these family programs has been on reducing overstimulation from the natural environment, by buffering the patient against the stress of external, independent life events (Brown and Birley 1968), by improving family coping, and by decreasing stress from the family (Doane et al. 1981; Leff and Vaughn 1981; Goldstein 1985, in press).

However, as Hogarty et al. (1986) point out, overstimulation can also occur in the therapeutic environment. Thus, overenthusiastic attempts to rehabilitate long-stay patients led to the return of symptoms that had been absent for years (Brown, Birley, and Wing 1972). Hogarty, Goldberg, and Schooler's (1974) program of major role therapy (MRT) seems to have had expectations of role performance that were too high and thus too stimulating for some patients. Thus, Goldberg et al. (1977) showed that MRT helped patients who were free of symptoms but increased relapse in more symptomatic patients. Similarly, the disappointing results of the Kottgen et al. (1984*b*) program appear to have been due to the overstimulating nature of their interpretive interventions.

Somewhat forgotten in the push to reduce overstimulation has been the deleterious effect of *understimulation*. Thus, as Wing and Brown (1970) observe, an understimulating "back ward" may lead to withdrawal and a lack of external social stimulation and support. Internal stimuli may therefore come into the foreground and lead to relapse. This understimulation can occur in natural environments as well as therapeutic environments. Thus, some overprotective family environments may neglect or shelter the patient from social stimulation to the extent that internal stimuli come to the fore, thereby precipitating a relapse or interfering with social functioning. In other words, the level of expectation of the patient can be reduced too far below that of the very high expectations of the highly critical relative.

These programs suggest that increasing the problem-solving and communicational abilities of families that include a schizophrenic protects and buffers patients from external life stresses and reduces stress from family conflict. Further, it is argued that both therapeutic and natural environments can be overstimulating and can increase stress, or understimulating and thus provoke social withdrawal.

Models that focus directly on the social skills of the patient as well as family members may have additive effects on the long-term social functioning of the patient. Falloon et al. had directive skill-building for the patient in the family context. Anderson et al. used "enactment" and "reenactment" in their family program. These direct interventions with the patients may lead to better social functioning at outcome than a purely psychoeducational approach.

Only one of these programs, that by Hogarty et al., showed an additive effect of individually focused treatment. The individual treatment of Hogarty et al. differed from that of Falloon et al. in that the social skills training focused strongly on the patient's social skills with the family and was carried out in small groups of patients. The patient was trained to be more positive and less critical with the family members in particular rather than people in general. One implication of this finding could be that this improvement in patient coping with the associated improvements in social competence decreases the impact of an underlying vulnerability and decreases the number of life events caused by the patient's lack of competence. The success of the Hogarty social skills training appears due to its focus on the patient's family interactions.

Conclusions

1. *Family psychoeducational treatment programs in conjunction with antipsychotic medication substantially improve the prognosis of schizophrenic patients.*

This review has illustrated the consistent effectiveness of four programs that, despite differences in where the family is seen, which family members are seen, and the format and timing of sessions, share the common features of respectful inquiry, the provision of information, and concrete management strategies. These characteristics differentiate them from more interpretive, insight-oriented family programs. Further, the improvements in social and psychological functioning are particularly associated with reductions in the intensity of negative emotional family interaction.

2. *Such family treatment programs reduce costs.*

As the Falloon study showed, even though a family approach requires additional time and money in the short-term, it is cost-effective and saves money in the long-term because of fewer rehospitalizations and less use of crisis facilities, as well as improved role functioning in the community.

3. *All kinds of families may benefit from a psychoeducational family treatment approach.*

The three later psychoeducational programs selected patients living in highly charged emotional atmospheres (high EE families) as being most at risk for relapse. However, it is now being recognized that patients who live in neutral and/or supportive emotional atmospheres (low EE families) are also at risk for relapse (albeit less so) due to the stress of life events or noncompliance with medication (Leff et al. 1985; Hogarty et al. 1986). Furthermore, the selection of patients living with high EE relatives has led to a preponderance of unmarried males living with parents in these studies. There is little doubt that married and unmarried female schizophrenics could also benefit from such programs. Thus, low EE families could benefit from education about schizophrenia and improved family coping.

4. *Psychoeducation cannot stand alone.*

The information provided to relatives about the symptoms, likely course, causes, and treatment of schizophrenia reduce guilt feelings and blame, factors that can exacerbate the patient's problem. However, the psychoeducational family programs have been successful in the context of the provision of long-term support to families, and the integration of medical and community services with the family approach. The family approach is neither a panacea nor a substitute for community facilities such as residential rehabilitation programs, crisis residential facilities, and group homes. Family programs are one link in a comprehensive system of care.

5. *Family programs should be tailored to the family.*

Probably the most important variables affecting the family's response to treatment are the number of previous admissions and the length of the illness. Anderson (1983) noted that whereas relatives of long-term patients usually respond with relief to the survival-skills workshop, relatives of recent-onset patients can be shocked by information about schizophrenia and its likely course; they find the prognosis to be very pessimistic and grim. In fact, the prognosis for a onetime admission is much better than for a repeat admission; it is entirely possible that the patient will never experience another psychotic break.

Thus, it may be most appropriate in the case of recent-onset patients to talk with the family about the "psychotic experiences" of the patient, possible precipitants, and future ways of coping in the brief crisis-oriented approach of Goldstein et al. If further admissions occur, it may then be appropriate to talk directly about schizophrenia and its implications, and to involve the family in a long-term program of aftercare.

6. *Family treatment may allow reduced or intermittent medication.*

Falloon et al. showed that medication compliance was better with family treatment, but also that patients were prescribed and ingested lower dosages. This lowers the risk of long-term side effects and allows greater improvement in social functioning.

Carpenter and Heinrichs (1983) point out the drawbacks of continuous long-term medication: Many patients develop tardive dyskinesia or an exacerbation of the "deficit" syndrome. Carpenter and Heinrichs are experimenting with "targeted" medication in which patients are followed drug-free until prodromal signs of an impending relapse appear. Medication is then started to prevent an exacerbation and stopped when the patient restabilizes. However, this approach depends on the cooperation of people who interact with the patient to respond to prodromal signs. Because of the nature of their illness, patients are less likely to be able to monitor their own prodromal signs. Carpenter and Heinrichs' program was applied in the context of a psychoeducational program for families. They suggested that medication could be reduced significantly without increasing psychopathology.

7. *Supportive, skill-oriented treatment can help too.*

Hogarty et al. show that a supportive program of social skills training, when focused on improving communicational skills with the family, significantly improves patients' prognoses, particularly when given in conjunction with family treatment. This suggests that there is a role for individual or group treatment of schizophrenic patients, if it is practical, skills-oriented, and proceeds at an appropriate pace.

Docherty (1984) suggests that the sequencing of treatment is important. A family approach is most critical during the first year posthospitalization; social skills training may have an effect in the beginning of the second year; and a subgroup of patients may benefit from a supportive form of psychotherapy thereafter. Future research should determine whether behaviorally oriented skills training is necessary for patients to attain higher levels of social and occupational outcomes.

Treatment of patients without their relatives may be the only choice available for certain patients because of the large number of chronic patients who have become estranged from their families. Platman (1983) cites a New York State study which showed that of patients who had been hospitalized more than 6 months, only 23 percent returned to private residences, compared with 63 percent hospitalized less than 6 months. For these patients, a family approach cannot be used and work can only be done with the individual or in groups of peers. However, psychoeducational programs could still be mounted in nonfamilial settings such as foster and board-and-care homes.

8. *Family psychoeducational programs may generalize to other populations.*

It has now been shown that the family emotional atmosphere predicts the outcome of disorders other than schizophrenia. Hooley, Orley, and Teasdale (1986) showed that depressed patients have poorer outcomes if they live with high-EE critical spouses. Similarly, Miklowitz et al. (in press) showed that turbulent family atmospheres indexed by EE and affective style predict relapse of bipolar-manic patients living with parents. These naturalistic studies suggest that family intervention may be applicable to the treatment of other disorders as well.

An example of such a program is a program by Glick et al. (1985) in which family intervention was given to both schizophrenic and affectively disordered inpatients. Preliminary results show that global outcomes at discharge were better for good premorbid schizophrenics, marginally better for patients with major affective disorders, but not better for poor premorbid schizophrenics, when compared with customary hospital care. That effects were not more pronounced may have been due to the brevity of the family intervention, and in particular that it did not extend after discharge. Nevertheless, such a program provides some optimism about the generalization of family psychoeducational approaches to other disorders.

10. *Further research is required to distinguish the active ingredients of the family treatment and crucial mediating family variables.*

It is unclear what the "active ingredients" of these family treatments are. Some active ingredients may be specific treatment approaches, while others may be nonspecific therapist factors. I have outlined presumed ingredients above, but these remain speculative. Additionally, although decreases in relapse are particularly associated with decreases in the intensity of emotional atmospheres in the home, it has not been proved that decreasing emotional temperature is the cause of a better outcome. Doane et al. (1985) showed that a negative parental affective style is associated with poor problem solving. It is as plausible to argue that Falloon's family program improved problem solving which displaced negative emotion as it is to argue that decreases in conflict allowed improved problem solving. It is important therefore in future research to study both stressful and protective aspects of family process.

11. *Good science can be poor politics.*

The social scientists who innocently coined the term "expressed emotion" to describe the expression of strong feeling (Brown et al. 1962) knew little of the storm of reactions from relatives of the mentally ill that would emerge two decades later. Relatives of the mentally ill have felt unfairly blamed for precipitating relapses, that their contributions to helping remain unacknowledged, and that the burden of coping remains on their shoulders: The scientists are out to get the family again.

There is more than a grain of truth to the charge that mental health professionals focus on the negative side of people and look for what is wrong. A major part of our training as diagnosticians is to find out what is wrong. However, this does not invalidate the scientific research that has been completed. An important body of research has accumulated that shows the prognostic importance of EE. That low EE may index useful coping strategies such as self-control and emotional distancing rather than confrontation has not been investigated, and certainly not publicized to relatives.

There has been a public relations gap. Managers in the private sector know well about "staying close to the customer" and "management by wandering around" (Peters and Austin 1985). They study how to market their products. It seems that in the case of EE, social scientists have been poor politicians, communicating with their peers but forgetting the real "customers"—patients and their relatives. Nevertheless, there are still opportunities for research scientists to recast their findings on EE more positively, for family members to understand the EE research better, and for administrators to recognize that the family intervention programs spawned by this research provide the possibility of adding one important cost-effective component to the long-term care of people with schizophrenia.

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Anderson, C.M. "Psychoeducational Family Interventions: Suggested Modifications for Specific Subgroups of Schizophrenic Patients." Presented at the Max Planck Institute International Conference on Young Schizophrenic Patients, Bavaria, West Germany, 1983.

Anderson, C.M.; Hogarty, G.E.; and Reiss, D.J. Family treatment of adult schizophrenic patients: A psychoeducational approach. *Schizophrenia Bulletin,* 6:490–505, 1980.

Anderson, C.M.; Reiss, D.J.; and Hogarty, G.E. *Schizophrenia in the Family: A Practitioner's Guide to Psychoeducation and Management.* New York: Guilford Press, 1986.

Berkowitz, R.; Eberlein-Fries, R.; Kuipers, L.; and Leff, J.P. Educating relatives about schizophrenia. *Schizophrenia Bulletin,* 10:418–429, 1984.

Berkowitz, R.; Kuipers, L.; Eberlein-Fries, R.; and Leff, J. Lowering expressed emotion in relatives of schizophrenics. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics.* London: Jossey-Bass, Publishers, 1981. pp. 27–48.

Boszormenyi-Nagy, I. A theory of relationships: Experience and transaction. In: Boszormenyi-Nagy, I., and Framo, J.L., eds. *Intensive Family Therapy.* New York: Harper and Row, 1965. pp. 33–66.

Bowen, M. *Family Therapy in Clinical Practice.* New York: Jason Aronson, 1978.

Brown, G.W., and Birley, J.L.T. Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behavior,* 9:203–214, 1968.

Brown, G.W.; Birley, J.L.T.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry,* 121:241–258, 1972.

Brown, G.W.; Monck, E.M.; Carstairs, G.M.; and Wing, J.K. Influence of family life on the course of schizophrenic illness. *British Journal of Preventative and Social Medicine,* 16:55–68, 1962.

Cardin, V.A.; McGill, C.W.; and Falloon, I.R.H. An economic analysis: Costs, benefits, and effectiveness. In: Falloon, I.R.H., ed. *Family Management of Schizophrenia.* Baltimore: Johns Hopkins University Press, 1965. p. 115–123.

Carpenter, W.T., Jr., and Heinrichs, D.W. Early intervention, time-limited, targeted pharmacotherapy of schizophrenia. *Schizophrenia Bulletin,* 9:533–542, 1983.

Cozolino, L., and Nuechterlein, K. A pilot study of the impact of a family education program of relatives of recent onset schizophrenic patients. In: Goldstein, M.J.; Hand, I.; and Hahlweg, K., eds. *Treatment of Schizophrenia: Family Assessment and Intervention.* Heidelberg: Springer-Verlag, 1986. pp. 129–144.

Creer, C., and Wing, J.K. *Schizophrenia at Home.* London: Institute of Psychiatry, 1974.

Doane, J.A.; Falloon, I.R.H.; Goldstein, M.J.; and Mintz, J. Parental affective style and the treatment of schizophrenia. *Archives of General Psychiatry,* 42:34–42, 1985.

Doane, J.A.; West, K.L.; Goldstein, M.J.; Rodnick, E.H.; and Jones, J.E. Parental communication and affective style: Predictors of subsequent schizophrenia-spectrum disorders in vulnerable adolescents. *Archives of General Psychiatry,* 38:679–695, 1981.

Docherty, J.P. O Tempora, O Mores: Directions in research on the psychotherapeutic treatment of schizophrenia. *Schizophrenia Bulletin,* 10:621–623, 1984.

Doll, W. Family coping with the mentally ill: An unanticipated problem of deinstitutionalization. *Hospital & Community Psychiatry,* 27:183–185, 1976.

Esterson, A.; Cooper, D.G.; and Laing, R.D. Results of family-oriented therapy with hospitalized schizophrenics. *British Medical Journal,* 2:1462–1465, 1965.

Falloon, I.R.H.; Boyd, J.L.; and McGill, C.W. *Family Care of Schizophrenia: A Problem-Solving Approach to the Treatment of Mental Illness.* New York: Guilford Press, 1984.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Razani, J.; Moss, H.B.; and Gilderman, A.M. Family management in the prevention of exacerbations of schizophrenia. *New England Journal of Medicine,* 306:1437–1440, 1982.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Williamson, M.; Razani, J.; Moss, H.B.; Gilderman, A.M.; and Simpson, G.M. Family management in the prevention of morbidity of schizophrenia: Clinical outcome of a two-year longitudinal study. *Archives of General Psychiatry,* 42:887–896, 1985.

Falloon, I.R.H., and Liberman, R.P. Behavioral family interventions in the management of chronic schizophrenia. In: McFarlane, W.R., ed. *Family Therapy in Schizophrenia.* New York: Guilford Press, 1983. pp. 117–137.

Falloon, I.R.H.; Liberman, R.P.; Lillie, F.J.; and Vaughn, C.E. Family therapy for relapsing schizophrenics and their families: A pilot study. *Family Process,* 20:211–221, 1981.

Falloon, I.R.H.; Watt, D.C.; and Shepherd, M. The social outcome of patients in a trial of long-term continuation therapy in schizophrenia: Pimozide vs. fluphenazine. *Psychological Medicine,* 8:265–274, 1978.

Federn, P. *Ego Psychology and the Psychoses.* New York: Basic Books, 1952.

Folkman, S.; Dunkel-Schetter, C.; DeLongis, A.; and Gruen, R.J. The dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *Journal of Personality and Social Psychology,* 50:992–1003, 1986.

Freeman, H.E., and Simmons, O.G. *The Mental Patient Comes Home.* New York: John Wiley & Sons, Inc., 1963.

Glick, I.D.; Clarkin, J.F.; Spencer, J.H.; Haas, G.L.; Lewis, A.B.; Peyser, J.; DeMane, N.; Good-Ellis, M.; Harris, E.; and Lestalle, V. A controlled evaluation of inpatient family intervention: I. Preliminary results of the six-month follow-up. *Archives of General Psychiatry,* 42:882–886, 1985.

Goldberg, S.C.; Schooler, N.R.; Hogarty, G.E.; and Roper, M. Prediction of relapse in schizophrenic outpatients treated by drug and social therapy. *Archives of General Psychiatry,* 34:171–184, 1977.

Goldman, H.H., and Gatozzi, A.A. Defining and counting the chronically mentally ill. *Hospital & Community Psychiatry,* 32:21–27, 1981.

Goldstein, M.J. Family factors that antedate the onset of schizophrenia and related disorders: The results of a fifteen year prospective longitudinal study. *Acta Psychiatrica Scandinavica,* 71:7–18, 1985.

Goldstein, M.J. Family interaction patterns that antedate the onset of schizophrenia and related disorders: A further analysis of data from a longitudinal prospective study. The results of a fifteen year prospective longitudinal study. In: Hahlweg, K., and Goldstein, M.J., eds. *The Impact of Family Research on Our Understanding of Family Psychopathology.* Heidelberg: Springer-Verlag, in press.

Goldstein, M.J., and Kopeikin, H.S. Short- and long-term effects of combining drug and family therapy. In: Goldstein, M.J., ed. *New Developments in Interventions With Families of Schizophrenics.* London: Jossey-Bass, Publishers, 1981. pp. 5–26.

Goldstein, M.J.; Rodnick, E.H.; Evans, J.R.; May, P.R.A.; and Steinberg, M.R. Drug and family therapy in the aftercare treatment of acute schizophrenia. *Archives of General Psychiatry,* 35:169–177, 1978.

Goldstein, M.J., and Strachan, A.M. The impact of family intervention programs on family communication and the short-term course of schizophrenia. In: Goldstein, M.J.; Hand, I.; and Hahlweg, K., eds. *Treatment of Schizophrenia: Family Assessment and Intervention.* Heidelberg: Springer-Verlag, 1986. pp. 185–192.

Gunderson, J.G.; Frank, A.F.; Katz, H.M.; Vannicelli, M.L.; Frosch, J.P.; and Knapp, P.H. Effects of psychotherapy in schizophrenia: II. Comparative outcome of two forms of treatment. *Schizophrenia Bulletin,* 10:564–598, 1984.

Gurman, A.S., and Razin, A.M. *Effective Psychotherapy: A Handbook of Research.* New York: Pergamon Press, 1977.

Hatfield, A.B. Psychological costs of schizophrenia to the family. *Social Casework,* 23:355–359, 1978.

Hatfield, A.B. Coping effectiveness in families of the mentally ill: An exploratory study. *Journal of Psychiatric Treatment and Evaluation,* 3:11–19, 1981.

Hogarty, G.E.; Anderson, C.M.; Reiss, D.J.; Kornblith, S.J.; Greenwald, D.P.; Javna, C.D.; Madonia, M.J.; and the EPICS Schizophrenia Research Group. Family psycho-education, social skills training and maintenance chemotherapy in the aftercare treatment of schizophrenia: I. One year effects of a controlled study on relapse and expressed emotion. *Archives of General Psychiatry,* 43:633–642, 1986.

Hogarty, G.E.; Goldberg, S.C.; and Schooler, N.R. Drug and sociotherapy in the aftercare of schizophrenic patients: III. Adjustment of non-relapsed patients. *Archives of General Psychiatry,* 31:609–618, 1974.

Hogarty, G.E.; Schooler, N.R.; Ulrich, R.; Mussare, F.; Ferro, P.; and Herron, E. Fluphenazine and social therapy in the aftercare of schizophrenic patients. *Archives of General Psychiatry,* 36:1283–1294, 1979.

Holden, D.F., and Lewine, R.R.J. How families evaluate mental health professionals, resources, and effects of illness. *Schizophrenia Bulletin,* 8:626–633, 1982.

Hooley, J.M.; Orley, J.; and Teasdale, J.D. Levels of expressed emotion and relapse in depressed patients. *British Journal of Psychiatry,* 148:642–647, 1986.

Kessler, S. The genetics of schizophrenia: A review. *Schizophrenia Bulletin,* 6:404–416, 1980.

Kottgen, C.; Sonnichsen, I.; Mollenhauer, K.; and Jurth, R. The family relations of young schizophrenic patients: Results of the Hamburg Camberwell Family Interview Study I. *International Journal of Family Psychiatry,* 5:61–70, 1984*a*.

Kottgen, C.; Sonnichsen, I.; Mollenhauer, K.; and Jurth, R. Group therapy with the families of schizophrenic patients: Results of the Hamburg Camberwell Family Interview Study III. *International Journal of Family Psychiatry,* 5:83–94, 1984*b*.

Laing, R.D., and Esterson, A. *Sanity, Madness and the Family.* London: Tavistock, 1964.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; Eberlein-Fries, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry,* 141:121–134, 1982.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; Eberlein-Fries, R.; and Sturgeon, D. Social intervention in the families of schizophrenics: Addendum. *British Journal of Psychiatry,* 142:313, 1983*b*.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; and Sturgeon, D. A controlled trial of social intervention in the families of schizophrenic patients: Two year follow-up. *British Journal of Psychiatry,* 146:594–600, 1985.

Leff, J.P.; Kuipers, L.; Berkowitz, R.; Vaughn, C.E.; and Sturgeon, D. Life events, relatives' expressed emotion and maintenance neuroleptics in schizophrenic relapse. *Psychological Medicine,* 13:799–806, 1983*a*.

Leff, J.P., and Vaughn, C.E. The role of maintenance therapy and relatives' expressed emotion in relapse of schizophrenia: A two year follow-up. *British Journal of Psychiatry,* 139:102–104, 1981.

Leff, J.P., and Vaughn, C.E. *Expressed Emotion in Families.* New York: Guilford Press, Inc., 1985.

Leff, J.P., and Wing, J.K. Trial of maintenance therapy in schizophrenia. *British Medical Journal,* 3:599–604, 1971.

Liberman, R.P. Behavioral approaches to couple and family therapy. *American Journal of Orthopsychiatry,* 40:106–118, 1970.

Liberman, R.P. Behavioral methods in group and family therapy. *Seminars in Psychiatry,* 4:145–156, 1972.

Liberman, R.P.; DeRisi, W.J.; and King, L.W. Behavioral interventions with families. In: Masserman, J., ed. *Current Psychiatric Therapies.* New York: Grune & Stratton, 1973. pp. 175–182.

Liberman, R.P.; Falloon, I.R.H.; and Aitchison, R.A. Multiple family therapy for schizophrenics: A behavioral approach. *Psychosocial Rehabilitation Journal,* 4:60–77, 1984.

Liberman, R.P.; Massel, H.K.; Mosk, M.; and Wong, S.E. Social skills training for chronic mental patients. *Hospital & Community Psychiatry,* 36:396–403, 1985.

Lukoff, D.; Snyder, K.; Ventura, J.; and Nuechterlein, K.H. Life events, familial stress, and coping in the developmental course of schizophrenia. *Schizophrenia Bulletin,* 10:258–292, 1984.

May, P.R.A., and Tuma, A.H. The Paul H. Hoch Award Lecture: A follow-up study of the results of treatment of schizophrenia. In: Spitzer, R.L., and Klein, D.F., eds. *Evaluation of Psychological Therapies.* Baltimore and London: John Hopkins University Press, 1976. pp. 256–284.

Miklowitz, D.J.; Goldstein M.J.; Falloon, I.R.H.; and Doane, J.A. Interactional correlates of expressed emotion in the families of schizophrenics. *British Journal of Psychiatry,* 144:482–487, 1965.

Miklowitz, D.J.; Goldstein, M.J.; Nuechterlein, K.H.; Snyder, K.S.; and Doane, J.A. Expressed emotion, affective style, lithium compliance, and relapse in recent-onset mania. *Psychopharmacology Bulletin,* 22:628–632, 1986*a*.

Miklowitz, D.J.; Strachan, A.M.; Goldstein, M.J.; Doane, J.A.; Snyder, K.S.; Hogarty, G.E.; and Falloon, I.R.H. Expressed emotion and communication deviance in the families of schizophrenics. *Journal of Abnormal Psychology,* 95:60–66, 1986*b*.

Nuechterlein, K.H., and Dawson, M.E. A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin,* 10:300–312, 1984.

Overall, J.E., and Gorham, D.R. The Brief Psychiatric Rating Scale. *Psychological Reports,* 10:799–812, 1962.

Palazzoli, M.S.; Boscolo, L.; Cecchin, G.; and Prata, G. *Paradox* *and Counterparadox.* New York: Jason Aronson, 1978.

Parloff, M.B.; Wolfe, B.; Hadley, S.; and Waskow, I.E. *Assessment of Psychosocial Treatment of Mental Disorders: Current Status and Prospects.* (Report to the Presidential Commission on Mental Illness) Washington, DC: National Institute of Mental Health, 1978.

Peters, T., and Austin, N. *A Passion for Excellence.* New York: Random House, 1985.

Platman, S.R. Family caretaking and expressed emotion: An evaluation. *Hospital & Community Psychiatry,* 34:921–925, 1983.

Platt, S.; Weyman, A.; Hirsch, S.; and Hewett, S. The Social Behaviour Assessment Schedule (SBAS): Rationale, contents, scoring, and reliability of a new interview schedule. *Social Psychiatry,* 15:43–55, 1980.

Snyder, K.S., and Liberman, R.P. Family assessment and intervention with schizophrenics at risk for relapse. In: Goldstein, M.J., ed. *New Developments in Intervention With Families of Schizophrenics.* San Francisco: Jossey-Bass, Publishers, 1981. pp. 49–60.

Spaniol, L.; Jung, H.; Zipple, A.M.; and Fitzgerald, S. The needs and coping strengths of families of the mentally ill: Report of a national survey. *Psychosocial Rehabilitation Journal,* in press.

Stanton, A.H.; Gunderson, J.G.; Knapp, P.H.; Frank, A.F.; Vannicelli, M.L.; Schnitzer, R.; and Rosenthal, R. Effects of psychotherapy in schizophrenia: I. Design and implementation of a controlled study. *Schizophrenia Bulletin,* 10:520–563, 1984.

Strachan, A.M., and Dunkel-Schetter, C. "Coping and Expressed Emotion in the Relatives of Schizophrenics." Unpublished manuscript, University of California, Los Angeles, 1985.

Strachan, A.M.; Goldstein, M.J.; and Miklowitz, D.J. Do relatives express expressed emotion? In: Goldstein, M.J.; Hand, I.; and Hahlweg, K., eds. *Treatment of Schizophrenia: Family Assessment and Intervention.* Heidelberg: Springer-Verlag, 1986. pp. 51–58.

Strachan, A.M.; Leff, J.P.; Goldstein, M.J.; Doane, J.A.; and Burtt, C. Emotional attitudes and direct communication in the families of schizophrenics: A cross-national replication. *British Journal of Psychiatry,* 149:279–287, 1986.

Terkelson, K.G. Schizophrenia and the family: II. Adverse effects of family therapy. *Family Process,* 22:191–200, 1983.

Valone, K.; Norton, J.P.; Goldstein, M.J.; and Doane, J.A. Parental expressed emotion and affective style in an adolescent sample at risk for schizophrenia spectrum disorders. *Journal of Abnormal Psychology,* 92:399–407, 1983.

Vaughn, C.E., and Leff, J.P. The influence of family and social factors on the course of psychiatric illness. A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry,* 129:125–137, 1976.

Vaughn, C.E.; Snyder, K.S.; Jones, S.; Freeman, W.B.; and Falloon, I.R.H. Family factors in schizophrenic relapse: A California replication of the British research on expressed emotion. *Archives of General Psychiatry,* 41:1169–1177, 1984.

Wallace, C.J., and Liberman, R.P. Social skills training for patients with schizophrenia: A controlled clinical trial. *Psychiatry Research,* 15:239–247, 1985.

Wallace, C.J.; Nelson, C.J.; Liberman, R.P.; Aitchison, R.A.; Lukoff, D.; Elder, J.P.; and Ferris, C. A review and critique of social skills training with schizophrenic patients. *Schizophrenia Bulletin,* 6:42–63, 1980.

Weakland, J.H.; Fisch, R.; Watzlawick, P.; and Bodin, A.M. Brief therapy: Focused problem resolution. *Family Process,* 13:141–168, 1974.

Wing, J.K., and Brown, G.W. *Institutionalism and Schizophrenia.* Cambridge: Cambridge University Press, 1970.

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Role of the Family in Case Management of the Mentally Ill

by James Intagliata, Barry Willer, and Gladys Egri

Abstract

This article provides a systematic discussion and assessment of the actual and potential contributions that families of the chronic mentally ill can make to the case management of their patient-relatives. It examines family members' involvement with six major case management functions including assessment, linkage, monitoring, assistance with daily problems, crisis intervention, and advocacy. For each function, the authors consider the potential of family members to make a contribution, specific activities that are more appropriately assumed by professional staff, and any limitations or caveats about family members' involvement with the given case management activity. The article points out that family members are clearly quite capable of making significant contributions in almost all aspects of managing their patient-relatives and that their potential contributions are too frequently ignored or underused by professionals. The authors recommend that professionals give greater acknowledgment to the case management efforts of family members, work to integrate the contributions of these natural helpers with formal case management systems, and provide family members with the encouragement and support they need to enhance their caring contributions.

Over the past 20 years, the views of many mental health professionals toward families of the mentally ill have been a significant obstacle to the development of a potentially powerful helping alliance between the two groups (Appleton 1974; Lamb and Oliphant 1978; Beels and McFarlane 1982). As noted by Beels and McFarlane (1982), scientific debate about causal factors in schizophrenia, in which the family itself has been proposed as a contributing factor, has raised the tension level between professionals and families and has added to the burden of family guilt. Further, based on their views of the family role in mental illness, many clinicians have excluded family members from the treatment process altogether or invited them to participate only on the condition that they themselves be treated as part of their relative's problem (Moroney 1980).

Fortunately, this situation has begun to improve significantly in recent years. One major reason has been the growing acceptance of a diathesis-stress model for explaining the cause and course of schizophrenia (Zubin and Spring 1977; Liberman 1982; Liberman, Falloon, and Wallace 1982). In such a multidimensional model, the development and manifestation of severe mental disorders are seen to be influenced by a host of interacting factors including personal variables, such as biological vulnerability and social competence level, and environmental variables, such as the level of stress and the availability of social and emotional support. One important implication of this model is that family members of mentally ill persons can play a positive role in the treatment process. Specifically, they can provide the protection and support needed to counteract the potentially debilitating effects of their relative's biological vulnerability and stressful environment.

A second major reason for the changing views of mental health professionals and their improving relationships with families of the

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mentally ill is the significant progress made in developing practical and effective models of family treatment. A number of excellent programs have been developed by mental health researchers and clinicians in just the past few years (Anderson, Hogarty, and Reiss 1981; Falloon et al. 1981; Liberman et al. 1981; Leff et al. 1982). The research evidence gathered on such programs thus far has demonstrated that when schizophrenic patients and their families participate, rates of relapse can be reduced significantly. These programs have been well received by families because their approach avoids blaming, provides much needed information, teaches coping skills, and communicates the message that families have the power to influence the course of their relative's illness for the better. The demonstrated success of these programs has also made it quite clear to mental health professionals that the families of their patients are a crucial resource that they can enlist in the treatment process.

A third major reason for the increasingly more positive regard of mental health professionals for families of the mentally ill is the growing acknowledgment of the crucial role that family members play in providing primary care and aftercare as a result of deinstitutionalization efforts. Without the willingness of family members to assume the significant responsibility of caring for their relatives, deinstitutionalization of the mentally ill could never have taken place in the magnitude that it has. Nationwide data collected by the Division of Biometry and Epidemiology of the National Institute of Mental Health (Goldman 1982) indicate that there are an estimated 800,000 to 1.5 million chronically mentally ill persons in this country. According to various reports (Lamb and Goertzel 1972, 1977; Minkoff 1978; Tessler et al. 1982; Baker and Intagliata 1984), one-quarter to one-third of these chronically mentally ill persons live with family members, most often with their parents. Further, the data indicate that if one considers not just the chronically mentally ill, but all individuals discharged from inpatient psychiatric facilities, perhaps as many as 65 percent return to live with relatives.

Many family members also remain quite involved with their mentally ill relative even when they do not live together. They continue to show concern for their family member who may live in a group home or apartment by assisting the relative in dealings with the community (e.g., service providers), by providing direct material assistance (e.g., food, money, clothing), or by providing temporary housing if their relative's alternative living arrangement becomes untenable.

The fact that so many families are actively assisting their patient-relative on a day-to-day basis has led some authors (Minkoff 1978; Hatfield 1981*a*) to suggest that it is the family, and not the traditional formal treatment system, that has assumed primary responsibility for case management of the mentally ill in this country. In view of the importance of case management for successfully maintaining the chronic mentally ill in the community (Test 1979; Bachrach 1980; Intagliata 1982; Tessler et al. 1982; Miller 1986), the role that families can and do play in providing such a supportive service merits closer examination.

The present article is intended to assist professionals in their consideration of this issue by presenting a systematic discussion of family members' actual and potential contributions to each of six specific aspects of the case management described by Intagliata (1982). These aspects include (1) being aware of the comprehensive needs of patients, (2) assisting patients in getting linked to and remaining engaged with the services they need, (3) monitoring the quality of the services being provided, (4) assisting patients with the management of simple life activities and practical daily problems, (5) assisting the patient in times of crisis, and (6) engaging in advocacy efforts at an individual or systems level to enhance the quality of clients' services and lives. In addition, while the "natural helping" capabilities (Bachrach 1983) of the family are highlighted, attention is also given to the limitations and caveats related to family members' involvement in case management activities.

Families and Case Management

Table 1 summarizes much of the discussion that follows. It operationalizes each of the six major case management functions and delineates how various case management activities might be shared between family members and professional staff.

Assessment. The first and perhaps most basic role that must be played by case managers in any system is to be aware of the comprehensive needs of their patients. When professionals or paraprofessionals assume the case-management role, they generally either take primary responsibility for or are actively involved in some systematic process to assess their patients' needs. This process usually provides the basis for the subsequent treatment plan. For the treatment plan to be most useful, information gathered in the needs assessment should be as complete and current as possible.

Family members clearly have a major contribution to make in identifying the needs of their mentally ill relative. A number of authors (Appleton 1974; Ryglewicz 1981; Yess 1981; Smets 1982) have acknowledged the family as an extremely important source of information about their relative's history of illness, treatment, and current functioning. Further, there is empirical evidence (Ellsworth et al. 1968) that family members' ratings of their ill relative's behavior are generally as valid as those of professional staff in predicting length of hospital stay, patients' self-report of symptoms, and future community tenure.

The fact that many family members are quite knowledgeable about their mentally ill relative seems natural when one considers that they alone have the firsthand knowledge that comes from living together daily in a nonhospital setting. Further, unlike the views of mental health professionals, which are typically based on cross-sectional assessments of their patients, family members have a longitudinal perspective of their relative's needs and functioning. It is their close, ongoing, long-term contact with their relative that frequently enables them to be the first to recognize the early signs of decompensation and relapse and to help to intervene to avoid it (Leavitt 1975; Herz and Melville 1980; Hansell 1981; Miller 1986). Finally, because of their extensive daily contact with their relative, family members are in a better position to be sensitive to many simple personal needs that may not come to the attention of the mental health professionals who interact with patients less frequently and generally only in treatment settings (Sullivan 1981).

Despite the fact that family members have considerable knowledge of their relative's needs, mental health professionals have not routinely invited them to discharge planning meetings where their relative's needs are to be discussed. In cases where the family is contacted, treatment staff often do little more than call the family in to inform them of what has already been decided without their input (Appleton 1974; Leavitt 1975; Ryglewicz 1981). By not inviting the input of the family as part of the assessment and treatment planning process, mental health professionals cut themselves off from a valuable information resource. In addition, they run the risk of alienating the family members whose understanding of and involvement in the rehabilitation process can be powerful determinants of their relative's successful adjustment in the community (Goldberg, Schooler, and Hogarty 1977; Krauss 1980; Falloon et al. 1982; Leff et al. 1982).

What seems advisable is that mental health professionals more consistently invite the active input of family members in the assessment and treatment planning process. As noted in table 1, however, certain tasks related to assessment must continue to be the responsibility of the professional case manager. These include assessing the family itself regarding such issues as communication and affective styles as well as taking responsibility for integrating the wide variety of information on a patient's needs into a comprehensive systematic plan of treatment. As noted throughout the case management literature, case management is a process that involves a variety of functions that need not all be performed exclusively by a single formal case manager. Thus, although it would be inappropriate for family members to take sole or even primary responsibility for assessing their relative's treatment needs, they can nevertheless provide considerable valuable assistance to the professional who has assumed ultimate case-management responsibility.

Linking. A second major function of the case manager is to link clients to services that will meet their needs. Many mentally ill persons lack the capacity to orchestrate the complex combinations of services that may be required. Thus, a case manager who is knowledgeable about the variety of available resources and who takes personal responsibility for ensuring that patients get the services and other supports (e.g., foodstamps, SSI payments) that they need can play a central role in making the system work as intended.

Assuring that clients get linked to needed services is not an easy task, especially when the clients are chronically mentally ill. In addition to the barriers that are inherent in the structure of the service system itself (e.g., eligibility criteria), many mentally ill persons are unable to become or stay engaged with the support services to which they are referred (Evans and Stellman 1977; Anthony, Cohen, and Vitalo 1978; Turner and Shiffren 1979; Krauss 1980). One way to increase the likelihood that the mentally ill will get and stay linked to the various services and supports that they need is to provide them with assertive case management (Beard, Malamud, and Rossman 1978; Stein and Test 1980). Another alternative is to follow the approach developed by Liberman and Falloon (Liberman et al. 1981) and focus on teaching patients themselves the social skills necessary to conduct the problem solving and self-advocacy needed to locate adequate

Table 1. Case-management functions and activities

Functions	Activities	
	Family members	**Professionals**
Assessment	1. Provide accurate historical information & longitudinal perspective on patient's functioning & symptoms	1. Seek out information on patient's functioning & needs from all relevant sources, including family
	2. Provide accurate information on clients' current level of functioning at home & in community	2. Invite & encourage family members to be part of the treatment planning & process
	3. Help to identify patient's needs not apparent to treatment team	3. Assess the family unit itself to determine its needs and resources
		4. Integrate all relevant information in a comprehensive treatment plan for the patient
Linking	1. Be aware of what the patient's treatment plan entails (medications and/or treatment program activities)	1. Be aware of the wide variety of potential programs & resources to which patients & families could be linked
	2. Provide encouragement & support that patient may need to comply with treatment plan (e.g., reminders about medication)	2. Assist patient to overcome barriers that might exist in becoming engaged with prescribed services or taking advantage of available supports (e.g., follow up on referrals, assist client with SSI paperwork)
	3. Support patient in taking as much responsibility as possible for carrying out treatment plan	3. Support patient in taking as much responsibility as possible for carrying out treatment plan
Monitoring	1. Try to get a sense for how patient is responding to treatments being received	1. Maintain regular contact with patient & family to monitor progress
	2. Notice changes in patient's community behavior & share them with staff	2. Maintain regular contact with agencies from which patient is receiving services, including making occasional visits
	3. Keep historical perspective on what does or does not seem to help patient	3. Regularly assess whether patient's participation in programs or use of medications produces intended changes
Assistance in daily living	1. Encourage patient to be as independent as he/she can manage	1. Encourage patient to be as independent as he/she can manage
	2. Within appropriate limits, provide patient with direct assistance & help in managing practical problems when necessary	2. Help family members set realistic limits on their level of involvement with helping patient meet needs

Table 1. Case-management functions and activities—*Continued*

Functions	Activities: Family members	Activities: Professionals
	3. Make patient's unmet needs for such assistance known to treatment staff	3. Identify or develop resources that will provide the patient with assistance or do so directly if necessary
Crisis intervention	1. Learn the early warning signs of crisis or relapse	1. Assist family members in identifying early signs of crisis or relapse in patient
	2. Inform case manager or other appropriate treatment staff of impending crisis/relapse in a timely fashion	2. Identify or develop resources that patient or family can turn to for immediate help in crisis
	3. Be available to patient to provide basic life supports (e.g., money, housing) in times of crisis	3. Be available to provide crisis intervention directly if necessary
Advocacy	1. Make case manager aware of any need for formal advocacy efforts on behalf of the patient	1. Use formal channels to advocate for individual patients who are being denied needed services
	2. Become actively involved in consumer advocacy groups that strive to improve care for the mentally ill	2. Identify gaps & needs not met by the current service system; make this information known to those responsible for planning and administering the system

treatment. Clearly, it is important to strike a balance between these two approaches such that the patient is permitted to function as independently as possible but still receives the case-management support necessary to be linked to needed services.

Note that for those patients who do need assistance in getting linked to services, the nature of this assistance can vary greatly in formality and intensity. Examples of the kinds of assistance that might be provided include: reminding patients about an appointment or the medication they need to take, providing them with transportation to a needed service if they cannot get there on their own, accompanying them to a service appointment not only to make sure that they get there but also to ensure that the provider understands and is responsive to their needs, and discussing with them their fears of or misgivings about treatment efforts so that they might maintain their motivation for treatment.

As suggested in table 1, some aspects of the linkage functions are probably best handled by a professional case manager. These include being aware of the wide range of service and support alternatives available and assisting patients in getting through the often considerable bureaucratic red tape that is required for them to get started with entitlement programs as well as some supportive services. However, there are many types of assistance and encouragement that can help facilitate patients' linkage to services and that do not require any complex or technical knowledge about the service system. These may make the difference between a patient continuing with or dropping out of a needed treatment. Such assistance can be provided effectively only by someone who is actively concerned about the patient, has the time and motivation needed to manifest this concern through action, and can develop a trusting, supportive relationship with the patient.

Family members clearly have an important contribution to make in this regard. Their understanding of and attitude toward treatment can be a very strong influence on whether or not their patient-relative uses it as prescribed (Schwartz and Schwartz 1964; Stevens 1972; Goldberg, Schooler, and Hogarty 1977). For mental health professionals to ignore this reality and fail to encourage and assist family members in taking an active and positive role in linking their relative to services would be an unfortunate underutilization of their capabilities.

Monitoring. The third essential case management function is to monitor the services being provided to patients. This requires the case manager to assess in some ongoing fashion whether the services to which a patient has been linked seem to be appropriate and effective. To do this, the case manager must take into account the patients' own satisfaction with their service involvement as well as be intimately familiar with the goals and objectives that members of the treatment team have set for patients. Monitoring may also entail the case manager visiting occasionally with the patient at the program where he or she is being served. This kind of firsthand contact may provide valuable information about the degree of fit between the client and the service that would otherwise be overlooked. Monitoring provides the system with valuable feedback about the extent to which the prescribed interventions are actually meeting the patients' needs and thus informs staff members about important needed modifications in treatment plans.

As is indicated in table 1, the monitoring activities of family members and professional staff are generally similar. They differ only in that each brings a somewhat different perspective to the monitoring responsibility. While treatment staff may be highly skilled in their abilities to observe and understand patients' response to treatment, their role in monitoring is severely limited by the fact that the majority of their time in contact with the patient is restricted to the clinical program setting. Although it is important to follow how patients respond in treatment settings, it is perhaps even more essential to know whether patients are successfully using these skills in their natural environments.

Family members have a key role to play in supplementing the monitoring efforts of professional program staff. They are good observers of the behavioral adjustment of their patient-relative and have the crucial firsthand knowledge that comes from living with them in the community. As such, they are natural monitors of their relative. If their input is invited, family members can provide clinical staff, including their relative's case manager, with important information about such issues as medication side effects and the degree to which their relative's behavior at home and in other community settings shows evidence of progress related to treatment goals. Further, to the extent that they are invited by professionals to understand their relative's treatment goals, family members can not only monitor their relative's progress but help to facilitate it by encouraging and supporting the same behaviors that are the focus of treatment. Finally, because they are not themselves a part of the formal service system, family members do not have the conflict of interest problems that can compromise the monitoring effects of case managers who work for the same system that provides the clients with treatment.

Assistance in Daily Living. In some mental health systems, case managers for the chronically mentally ill are expected to assist their patients with the management of simple life activities and practical daily problems that they are unable to deal with independently. In this way, they provide the crucial missing personal support that many patients require to succeed. In New York State, for example, case managers of the chronically mentally ill often provide patients with the assistance or encouragement they need for maintaining their personal hygiene or individual households. Other supporting activities might include helping patients prepare a grocery list, accompanying them on a shopping trip, or transporting them to a needed service if no other arrangements can be made (Intagliata 1982).

As with other case-management functions already discussed, providing patients with assistance in dealing with practical daily problems is a task that need not be managed exclusively by the professional case manager but that can be shared with other helpers. Weinman and Kleiner (1978), for example, employed lay persons to act as enablers for chronic mental patients living in the community. These persons spent several hours each day with their individual clients teaching them basic community living skills, escorting and introducing them to services and resources in the community, and acting as a liaison to gain the understanding and support of neighbors and local merchants for their patients. This program was highly successful in facilitating patients' adjustment to the community.

Perhaps much more so than with any of the other previously discussed case management functions, family members are already extremely familiar with assisting their patient-relative to manage practical, everyday problems. In fact, in many mental health service systems in which case managers either do not exist or are not sanctioned to provide patients with this kind of assistance, there is no one but the family to assume such responsibility. This may be true regardless of whether patients actually live with their families. As noted by numerous authors (Stevens

1972; Horowitz 1978; Ryglewicz 1981; Holden and Lewine 1982), the family is often not only the strongest and most enduring but the *only* support system available to the mentally ill individual.

It is obvious that there is a natural convenience about family members assuming at least part of the responsibilities for assisting the patient with daily problems in living. Although family members are often willing to take on this responsibility, however, it is easy for this assistance to become overinvolvement. Such overinvolvement can lead to higher relapse rates for the patient (Anderson, Hogarty, and Reiss 1981; Leff et al. 1982) and to the physical and emotional exhaustion of family members (Doll 1976; Hatfield 1978).

While it is important for mental health professionals to acknowledge the helpful contribution that family members can make in meeting the daily needs of their patient-relative, they should also help family members avoid the problems that can result from overinvolvement. As suggested in table 1, this can be done by providing family members with advice on how to set limits on their involvement and by ensuring that there are trained support staff (e.g., case managers, enablers) available to assist their patient-relatives in managing at least some of their demanding daily problems. The needs of family members for assistance in caring for their relatives cannot be overemphasized. Family members often have no alternative to becoming "overinvolved" because they are the only ones available or willing to assist their relative.

Crisis Intervention. A fifth important case-management function is crisis intervention. The adjustment to community living made by many formerly institutionalized psychiatric patients is quite tenuous. Significant crises may be precipitated by unexpected changes in the environment, even events that seem trivial to persons with a "normal" level of coping ability. If such crises are not managed in a timely fashion, they can easily lead to significant deterioration in the patients' levels of functioning and perhaps to their rehospitalization. It is the role of the case manager to be available to both the patients and their family in times of crisis and to be capable of either providing or linking them to needed crisis intervention supports.

Perhaps the most important aspect in crisis intervention is timing. For crisis intervention to be successful, the developing crisis must be identified and appropriate action taken quickly to prevent the situation from deteriorating further. Family members, especially those who live with their patient-relative, have the advantage of being able to spot many developing crises in a timely manner because of their ongoing contact. If they have learned what kinds of signs to look for, they may be the first to recognize the early stages of decompensation and can call this to the attention of their relative's case manager or other treatment staff (Leavitt 1975; Herz and Melville 1980). With other types of crises, they may be able to serve as a key resource to alleviate their relative's stress. They may, for example, intervene to get them through particular crisis periods by providing needed food, money, or housing. These are resources that the mental health system may simply not have to offer, or which it may be unable to provide in a timely manner.

Advocacy. A final case-management function to consider is that of advocacy. Two types of advocacy are important: that which takes place at the individual patient level and that at the systems level (Intagliata 1982). Perhaps the most frequently occurring situation requiring individual level advocacy involves situations where a patient is having difficulty getting linked to a service initially as well as those where a patient is being dropped from a program.

When such situations can be managed simply through informal interpersonal negotiations involving the case manager and the given service provider, this activity is probably subsumed under the linking function described earlier. When such informal negotiation is ineffective, however, successful linkage requires the case manager to become involved in formal advocacy activities. For example, the case manager may need to enlist the support of supervisory or administrative level staff within his or her own agency or to make use of even more formal channels such as seeking the assistance of legal aid services or of agencies that traditionally provide advocacy services for patients in need (e.g., Mental Health Associations).

Systems-level advocacy is a somewhat different case-management function. In general, it involves someone taking responsibility for calling attention to some deficiency in the service system that adversely affects a large number or class of patients. Systems-level advocacy often requires political action and is especially appropriate for citizen groups to get involved in. In the mental retardation field, parents established the Association for Retarded Citizens for advocacy purposes in the 1950s. Since then, they have been particularly effective in calling national attention to the plight and needs of the mentally retarded. Similar advocacy groups for the mentally ill have at last also begun to emerge—for example, the National Schizophrenia Fellowship in England and the National Alliance for the Mentally Ill in the United States (Hatfield 1981*b*). In addition to their goals of providing families with needed information and emotional support, they are advocating increased public awareness about mental illness, improved services for the mentally ill, and a greater commitment to research into the causes and treatments of mental illness.

Because of their considerable personal commitment and concern about mental illness, parents and other family members of the mentally ill can make extremely effective advocates for systems-level change. In this regard, their voices may be as, if not more, powerful than those of the mental health professionals. The mental health establishment thus has gained a potentially strong and influential ally in the family. What professionals must understand, however, is that the objectives of such advocacy groups may not always be entirely consistent with their own. Family members are beginning to demand change not only for the system as a whole but from mental health professionals as well. For example, relatives of the mentally ill are demanding not just what mental health professionals themselves would like (e.g., larger service and research budgets) but also more responsiveness and more respect for the family from mental health professionals.

Discussion

In this article we have discussed the contribution that family members can and do make to the case management of chronically mentally ill persons. While we are not suggesting that family members assume primary case-management responsibility for their relatives, it is clear that they are powerful natural helpers who can and do provide crucial supplemental assistance to case managers and other mental health professionals. If mental health professionals are to forge a stronger helping alliance with family members, one way of doing it is to acknowledge the contribution that families are already making to the case management of the mentally ill and to consider including families in the treatment process to an even greater degree if they so wish.

While family members can and should be encouraged to contribute actively to the case management of their patient-relatives, there are some limitations and issues that must be considered about their functioning in such a capacity. Some aspects of the case-management role cannot be relegated to family members because their effective performance requires special professional training or credentials. Further, while there are many ways in which family members can make important contributions to their relative's case management, it is important to be sensitive to the significant level of burden that family members' overall caretaking responsibilities may entail. Recently, a great deal of concern has been expressed in the professional literature for the problem of "burnout" among professionals who work with chronic patients (Lamb 1979). If professionals who work with such individuals 8 hours a day are at risk for burnout, what about family members who are involved with their relatives 24 hours a day? Finally, there are some family members who, because of their particular personal situation, are not capable of or willing to be significantly involved in assisting their patient-relative.

In response to these limitations, mental health professionals must (1) take responsibility for assuming those aspects of the case-management function that only trained professionals can carry out effectively, (2) conduct systematic research to identify those complementary aspects of case-management responsibilities that may be beneficial for willing family members to assume, (3) provide families with the encouragement and/or training they need to make a case-management contribution, and (4) acknowledge that some families do not want and should not be encouraged to be significantly involved in their relative's treatment.

Regarding the notion of training family members to assume case management responsibilities, note that this idea has, in fact, been implemented successfully in at least one regional developmental disabilities service system in California (Franck 1982). In this program, interested family members attend a formal training course and may eventually work as their relative's certified case manager under professional supervision. While we are not advocating that families of the mentally ill become so integrally involved with their relative's treatment, the success of this pilot program demonstrates the desire and capability of many family members to carry out important case-management functions.

The case-management process involves a complex set of tasks and responsibilities that must be dealt with for all the available personal and system resources to be brought to bear for the ultimate benefit of the client. While professionals have a major role to play in the case-management process, so do family members. There is more than enough work to share. It is time for professionals to acknowledge the important contribution already being made by family members and to do whatever they can to enhance and support this contribution.

References

Anderson, C.M.; Hogarty, G.; and Reiss, D.J. The psychoeducational family treatment of schizophrenia. In: Goldstein, M., ed. *New Directions for Mental Health Services: New Developments in Interventions With Families of Schizophrenics.* No. 12. San Francisco: Jossey-Bass, 1981. pp. 79–94.

Anthony, W.A.; Cohen, M.R.; and Vitalo, R. The measurement of rehabilitation outcome. *Schizophrenia Bulletin,* 4:365–383, 1978.

Appleton, W.S. Mistreatment of patients' families by psychiatrists. *American Journal of Psychiatry,* 131:655–657, 1974.

Bachrach, L.L. Overview: Model programs for chronic mental patients. *American Journal of Psychiatry,* 137:1023–1031, 1980.

Bachrach, L.L. New directions in deinstitutionalization planning. In: Bachrach, L.L., ed. *New Directions for Mental Health Services: Deinstitutionalization.* No. 17. San Francisco: Jossey-Bass, 1983. pp. 93–106.

Baker, F., and Intagliata, J. The chronically mentally ill in New York State's community support system program. *Hospital & Community Psychiatry,* 35:39–44, 1984.

Beard, J.H.; Malamud, T.J.; and Rossman, E. Psychiatric rehabilitation and long-term rehospitalization rates: The findings of two research studies. *Schizophrenia Bulletin,* 4:622–635, 1978.

Beels, C.C., and McFarlane, W.R. Family treatment of schizophrenia: Background and state of the art. *Hospital & Community Psychiatry,* 33:541–550, 1982.

Doll, W. Family coping with the mentally ill: An unanticipated problem of deinstitutionalization. *Hospital & Community Psychiatry,* 27:183–185, 1976.

Ellsworth, R.B.; Foster, L.; Childers, B.; Arthur, G.; and Kroeker, D. Hospital and community adjustment as perceived by psychiatric patients, their families, and staff. *Journal of Consulting and Clinical Psychology,* 32:1–41, 1968.

Evans, D.A., and Stellman, V.P. Community leaders assess quality of life. *Journal of Existentialism,* 4:7–12, 1977.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Rayani, J.; Moss, H.B.; and Gilderman, A.M. Family management in the prevention of exacerbations of schizophrenics. *New England Journal of Medicine,* 306:1437–1440, 1982.

Falloon, I.R.H.; Boyd, J.L.; McGill, C.W.; Strang, J.S.; and Moss, H.B. Family management training in the community care of schizophrenics. In: Goldstein, M., ed. *New Directions for Mental Health Services: New Developments in Interventions With Families of Schizophrenics.* No. 12. San Francisco: Jossey-Bass, 1981. pp. 61–77.

Franck, L. *Parent Program Coordination Training Seminar.* San Gabriel, CA: San Gabriel Valley Regional Center, 1982.

Goldberg, S.C.; Schooler, N.R.; and Hogarty, G.E. Prediction of relapse in schizophrenic outpatients treated by drug and sociotherapy. *Archives of General Psychiatry,* 34:171–184, 1977.

Goldman, H.H. Mental illness and family burden: A public health perspective. *Hospital & Community Psychiatry,* 33:557–560, 1962.

Hansell, N. Medication. In: Talbott, J., ed. *The Chronically Mentally Ill.* New York: Human Sciences Press, 1981. pp. 29–46.

Hatfield, A.B. Psychological costs of schizophrenia to the family. *Social Work,* 35:355–359, 1978.

Hatfield, A.B. Coping effectiveness in families of the mentally ill: An exploratory study. *Journal of Psychiatric Treatment and Evaluation,* 3:11–19, 1981*a*.

Hatfield, A.B. Families as advocates for the mentally ill: A growing movement. *Hospital & Community Psychiatry,* 32:53–55, 641–642, 1981*b*.

Herz, M.I., and Melville, C. Relapse in schizophrenia. *American Journal of Psychiatry,* 137:801–805, 1980.

Holden, D.F., and Lewine, R.R.J. How families evaluate mental health professionals, resources, and effects of illness. *Schizophrenia Bulletin,* 4:626–633, 1982.

Horowitz, A. Family, kin, and friend networks in psychiatric help-seeking. *Social Science and Medicine,* 12:297–304, 1978.

Intagliata, J. Improving the quality of community care for the chronically mentally disabled: The role of case management. *Schizophrenia Bulletin,* 8:655–674, 1982.

Krauss, J.B. The chronic psychiatric patient in the community: A model of care. *Nursing Outlook,* 5:308–314, 1980.

Lamb, H.R. Staff burnout in work with long-term patients. *Hospital & Community Psychiatry,* 30:396–398, 1979.

Lamb, H.R., and Goertzel, V. High expectations of long-term ex-State hospital patients. *American Journal of Psychiatry*, 129:471–475, 1972.

Lamb, H.R., and Goertzel, V. The long-term patient in the era of community treatment. *Archives of General Psychiatry*, 34:679–682, 1977.

Lamb, H.R., and Oliphant, E. Schizophrenia through the eyes of families. *Hospital & Community Psychiatry*, 29:803–806, 1978.

Leavitt, M. The discharge crisis: The experience of families of psychiatric patients. *Nursing Research*, 24:33–40, 1975.

Leff, J.; Kuipers, L.; Berkowitz, R.; Eberlein-Vries, R.; and Sturgeon, D. A controlled trial of social intervention in families of schizophrenic patients. *British Journal of Psychiatry*, 141:121–134, 1982.

Liberman, R.P. What is schizophrenia? *Schizophrenia Bulletin*, 8:435–437, 1982.

Liberman, R.P.; Falloon, I.H.; and Wallace, C. *Drug-Psychosocial Treatment Interactions in Schizophrenia*. Unpublished manuscript, Brentwood VA Medical Center, Los Angeles, CA, 1982.

Liberman, R.P.; Wallace, C.J.; Falloon, I.R.H.; and Vaughn, C.E. Interpersonal problem-solving therapy for schizophrenics and their families. *Comprehensive Psychiatry*, 22:627–630, 1981.

Miller, G. *The Future of the Chronically Mentally Ill: Proceedings of the Sixteenth Annual Texas Research Institute of Mental Sciences Symposium*. New York: S.P. Medical and Scientific Books, 1986.

Minkoff, K. A map of the chronic mental patients. In: Talbott, J., ed. *The Chronic Mental Patient*. Washington, DC: American Psychiatric Association, 1978. pp. 11–38.

Moroney, R.M. *Families, Social Services, and Social Policy*. Rockville, MD: National Institute of Mental Health, 1980.

Ryglewicz, H. "Working With the Family of the Psychiatrically Disabled Young Adult." Presented at The Young Adult Chronic Patient II Conference, Albany, NY, June 3–5, 1981.

Schwartz, M., and Schwartz, C.G. *Social Approach to Mental Patient Care*. New York: Columbia University Press, 1964.

Smets, A.C. Family and staff attitudes toward family involvement in the treatment of hospitalized chronic patients. *Hospital & Community Psychiatry*, 33:573–575, 1982.

Stein, L.I., and Test, M.A. Alternative to mental hospital treatment: I. Conceptual model, treatment program, and clinical evaluation. *Archives of General Psychiatry*, 37:392–397, 1980.

Stevens, B.C. Dependence of schizophrenic patients on elderly relatives. *Psychological Medicine*, 2:17–32, 1972.

Sullivan, J.P. Case management. In: Talbott, J., ed. *The Chronically Mentally Ill*. New York: Human Sciences Press, 1981. pp. 119–131.

Tessler, R.C.; Bernstein, A.G.; Rosen, B.M.; and Goldman, H.H. The chronically mentally ill in community support systems. *Hospital & Community Psychiatry*, 33:208–211, 1982.

Test, M.A. Continuity of care in community treatment. *New Directions in Mental Health Services*, 2:15–23, 1979.

Turner, J., and Shiffren, I. Community support system: How comprehensive? In: Stein, L., ed. *Community Support Systems for the Long-term Patient*. San Francisco: Jossey-Bass, 1979. pp. 1–13.

Weinman, B., and Kleiner, R.J. The impact of community living and community member intervention on the adjustment of the chronic psychiatric patient. In Stein, L., and Test, M.A., eds. *Alternatives to Mental Hospital Treatment*. New York: Plenum Press, Inc., 1978. pp. 139–162.

Yess, J.P. What families of the mentally ill want. *Community Support Services Journal*, 1:1–3, 1981.

Zubin, J., and Spring, B. Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, 86:103–126, 1977.

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Training Mental Health Workers in Psychiatric Rehabilitation

by Erna S. Rogers, Barry F. Cohen, Karen S. Danley, Dorothy Hutchinson, and William A. Anthony

Abstract

Staff members from nine mental health agencies were trained in psychiatric rehabilitation technology. The trainers then received intensive followup to assist them in implementing the technology in their own agencies. Evaluation of the impact of the "training of trainers" strategy suggested that mental health workers were able to acquire and apply the skills of psychiatric rehabilitation. The training strategy also appeared to be effective in helping the agencies adopt the philosophy, principles, and skills of psychiatric rehabilitation.

Promoting the use of psychiatric rehabilitation technology is a challenge because it requires major changes in the structure of a mental health agency. Not only does the pattern of service delivery need modification, but changes are also required in the values, roles, and functions of the human service professional (Stern and Minkoff 1979). Psychiatric rehabilitation technology requires change at both the individual (Glaser and Backer 1979; Jansson 1980; Carolyn and Stoffelmayer 1981; Marlowe, Spector and Bedell 1983) and organizational levels (White 1974; Goldman 1976; Passmore and King 1978; Weinman et al. 1979; Astrachan and Tischler 1981; Boje, Fedor, and Rowland 1982; Glaser 1982).

An empirically based set of principles and practitioner skills, and a number of model service programs have been identified for the field of psychiatric rehabilitation (Anthony 1980; Bachrach 1980; Liberman et al. 1984). Training manuals also have been developed to improve the expertise of rehabilitation professionals (see Liberman et al. 1975, 1984; Anthony, Cohen, and Pierce 1980; Anthony, Pierce, and Cohen 1980*a*, 1980*b*; Cohen et al. 1980*a*, 1980*b*; Pierce et al. 1980*a*, 1980*b*; Cohen and Nemec 1985). However, innovation in psychiatric rehabilitation has been limited by the recency of constituent knowledge and skills, and the complexity of the task of dissemination (Zand and Sorenson 1975; Haveliwala 1979; Liberman et al. 1982; Wolkon, Peterson, and Gougla 1982; Marlowe, Spector, and Bedell 1983). Backer, Liberman, and Kuehnel (1986) analyzed three examples of effective innovation in psychosocial rehabilitation and concluded that the following factors were critical: interpersonal contact with credible professional peers; outside consultation for the adoption process; organizational support for the innovation; persistent championship of the innovation; adaptability of the innovation; and credible evidence of success for the innovative program.

Use of the methods of psychiatric rehabilitation may become increasingly feasible because of advances in the specificity and hence teachability of this technology. Methods inherent in the psychiatric rehabilitation approach have been refined, and components of assessment, treatment, and staff functions are now well defined (Beard, Probst, and Malamud 1982; Anthony, Cohen, and Cohen 1983).

The focus of this study is the psychiatric rehabilitation approach, which has been described by Anthony, Cohen, and Cohen (1983). Using this approach, a practitioner guides the client through a three-phase rehabilitation process of

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diagnosis, planning, and intervention. In the diagnostic phase, functional and resource assessments are made of the particular environments in which the client is to live, learn, and work. In the next phase, a plan is developed which prioritizes skills to teach clients the resource goals, the interventions for achieving these goals, and the time frames and personnel responsible for implementing the interventions. In the final phase, interventions for building skills and developing resources are implemented.

This study was designed to promote the use of psychiatric rehabilitation technology, emphasizing what practitioners *do* rather than simply what they *know*. A method of "training trainers" was used because it was hoped that training existing agency staff to become "in-house" trainers would increase the chances of rehabilitation methods becoming established in the organization, instead of disappearing when the outside consultants leave (Glaser 1981). The agency trainers also become intimately involved in the change process and have an ongoing capability to adopt the innovation. Table 1 delineates the principles of dissemination and utilization of innovations upon which this study was based.

Methods

Agency Recruitment and Selection. This project began with a letter sent by the Center for Psychiatric Rehabilitation to 132 mental health agencies that had previous contact with the Center. Of the 132 letters sent, 85 agencies called the Center to indicate interest. After learning the goals of the project, 23 decided they were not able to participate or were not interested. The remaining 62 were sent application packets to facilitate the next level of screening. Forty sites returned completed applications.

The application packet requested detailed information from each agency about the following: clients served; staffing (numbers and type of education/experience); type of training and supervision methods used; previous research and program evaluation; and administrative information. The application process was guided by the literature on adoption of innovations which suggests that the readiness and ability of an agency to adopt an innovation can and should be assessed before embarking on such a complex process. The selection process provided information about how compatible the philosophy and structure of each agency was with the proposed training; how motivated each agency was to implement the innovation; and whether each agency had the resources to support the innovation. (Anthony et al. 1985).

The final selection of training sites was made from scores based on the following:

- Assessment of written applications provided by the recruited sites using a standardized assessment format.
- Elimination of sites that did not meet minimal criteria for successful innovation (e.g., client/staff ratio adequate to implement the approach; agency control over the treatment process).
- Assessment of State support for the psychiatric rehabilitation training technology on a 5-point scale.
- Assessment of the level of interest and motivation of site personnel and validation of application data through site visits.

The intensive nature of the project allowed the Center to choose only three sites for the first year and seven for the second year. One site dropped out of the project during the second year because of manpower shortages.

Training. Each site selected at least two "trainer-apprentices" for the training. Several sites designated more than two. A total of 30 trainer-apprentices began the training and, of those, 29 completed the training.

Training focused on teaching the core skills of psychiatric rehabilitation: rehabilitation diagnosis, planning, and intervention. The trainer-apprentices were also taught how to train and supervise practitioners (i.e., their trainees) in the use of these skills at their own sites. The training process consisted of five steps.

Step 1: Training in psychiatric rehabilitation practice at the Center. The trainer-apprentices from all selected sites came to the Center for Psychiatric Rehabilitation at Boston University for 5 days of training in the skills of psychiatric rehabilitation (see table 2). The following training methods were used: didactic presentation, videotaped demonstrations, live demonstrations, discussions, case presentations, role-playing exercises, paper-and-pencil exercises, keeping a personal learning journal, and homework assignments. The objectives of the first 5 days of training were to increase the readiness, awareness, and ability to perform the skills of psychiatric rehabilitation of the trainer-apprentices. The training also provided an opportunity to exchange ideas and develop networks with

Table 1. Principles of utilization

Product principle

1. Utilization is enhanced when the product is characterized by credibility, observability, relevance, relative advantage, ease in understanding and installation, compatibility, and ability to experiment

Process principles

2. Utilization involves an ongoing process which includes: (1) the identification of a relevant need, (2) the development or adoption of an innovation, (3) the development of an implementation strategy, and (4) the stabilization of innovation. The need identification process must include a feasibility assessment related to personal and organizational readiness for adoption

3. Utilization is enhanced by an action orientation by disseminators. An action orientation assumes a mutual sharing of responsibility for implementation by the disseminator and the user

4. Successful utilization depends on the early and active support of potential users. Potential users need to be involved in the process of planning, implementing, and evaluating an innovation. This is especially true when the innovation involves behavioral changes on the part of the user

5. Utilization is enhanced through the establishment of a "linking role" by the disseminator. This linking role would include (1) establishing effective communication, (2) direct teaching, (3) problem solving, and (4) support and encouragement of users

6. The point of implementation is especially crucial in the utilization process. Local change agents and users need continuing support and encouragement from the disseminator to ensure adoption will be consistent with the spirit and intent of the innovation, and to overcome the resistances inherent in any change situation. This is especially true when the innovation requires skill/behavioral changes on the part of the user

7. Utilization is enhanced when the adequacy and effectiveness of the innovation are actively monitored and evaluated

Context principles

8. Successful utilization depends on the active support of the host agency. Critical factors include readiness for change, capacity for change, and the availability of resources to support change

9. Utilization is enhanced when individuals are motivated to learn, open to change, flexible, and possess an adequate level of self-integration

10. Utilization is enhanced when a "felt need" exists for the innovation. Potential users must recognize the relevance and benefit of the innovation to their concerns

11. Utilization is enhanced through the establishment of a "linker role" within the utilization site. This "linking person" would (1) coordinate the site effort, (2) maintain communications, (3) teach, (4) solve problems, and (5) provide local support and encouragement

12. Utilization is enhanced by an interpersonal environment within the host agency characterized by trust, clarity of communication, willingness to entertain challenge, and effective problem solving

13. Utilization is enhanced when the social/cultural environment is actively considered. This may take the form of establishing open communications and being sensitive to expressed needs and concerns

14. The linking person in an agency needs to provide continuing support during the implementation process to ensure adoption will be consistent with the spirit and intent of the innovation, and, to overcome the resistances inherent in any change situation. This is especially true when the innovation requires skill/behavioral changes on the part of the adopter

15. Utilization is enhanced when the philosophy and values of the site are consistent with the spirit and intent of the innovation

Table 2. Skills of psychiatric rehabilitation taught to trainer-apprentices and trainees

Skill domain	Specific components
Rehabilitation diagnosis	Defining an overall rehabilitation goal Defining rehabilitation problems Defining goals in relation to specific environmental situations Identifying skill strengths and deficits in relation to the rehabilitation goal Defining skill strengths and deficits in observable, measurable terms Identifying present and needed levels of functioning Involving the client in the process of rehabilitation Responding to the client's feelings and problems Sharing one's own perspective
Planning	Assigning priorities to client's problems and goals Translating skill strengths and deficits into goal statements Identifying time lines for goals Developing goals for learning and applying skills Linking goals to the overall rehabilitation goal
Intervention	Identifying client steps that will lead to achievement of skill goals Behaviorally stating, measuring, and sequencing skill steps Developing teaching plans to teach skills to clients Explaining and demonstrating skill steps to clients Providing opportunities for clients to practice and receive feedback on skills Providing followup to ensure skill use

other professionals working in the field of psychiatric rehabilitation.

Step 2: Practice at their sites. Trainer-apprentices returned to their agencies to practice the skills of psychiatric rehabilitation as a means of increasing mastery. They conducted a minimum of four client interviews in which they practiced their skills. The interviews were audiotaped and mailed to project mentors at the Center. The Center mentors used telephone contact to provide feedback and remediation based upon their evaluation of the four taped sessions. Telephone contact throughout the training period was extensive, averaging a minimum of two 30-minute calls per month to each site. Sites were encouraged to contact the Center mentors whenever necessary for support, remediation, and advice.

Step 3: Training in "how to teach psychiatric rehabilitation" at the Center. The second week of training, approximately 7 weeks after the first week at Boston University, focused on teaching techniques (table 3). Twenty-four staff members from Massachusetts mental health agencies volunteered to act as "trainees" to be taught by the trainer-apprentices. Thus, apprentices had the opportunity to teach a group of professionals similar to their own staff while being observed, taped, and critiqued by Center mentors.

Step 4: Practice at sites. Apprentice trainers were required to practice their training skills by developing curricula and conducting "practice" sessions with their fellow trainer-apprentices and/or colleagues outside their agencies. Practice was conducted in small groups and in 1:1 sessions with trainees. The taped training sessions were reviewed by Center mentors and feedback was provided.

Step 5: Develop training programs used at sites. The trainer-apprentices developed plans to conduct on-site training of their staff in the skills of psychiatric rehabilitation. A total of 168 trainees (i.e., practitioners trained on-site by the trainer-apprentices) participated in some portion of the study. The training of staff at the sites involved a series of

Table 3. Teaching skills and techniques

Skill domain	Specific components
Planning the training	Outlining the content Developing the curriculum
Involving the trainees	Gathering information Encouraging participation Modifying curricula
Following up the training	Developing study groups Developing monitoring plans Providing feedback Revising the followup strategy

2-day workshops followed by weekly study groups where the staff reviewed their attempts to use their newly acquired skills with agency clients. The workshops were monitored by Center mentors during each of two planned visits to every site that were made in the 4 months following the Center training. These site visits gave the Center mentors an opportunity to observe the on-site training effort, to provide feedback and remedial training, and to plan needed changes. The study groups were taped to allow review by both the agency trainer-apprentices and Center mentors.

Evaluation Design. The goal of this project was to promote the use of our psychiatric rehabilitation approach in the participating agencies. Evaluations of the project's effectiveness were based on measures of:

- Acquisition of psychiatric rehabilitation skills.
- Application of psychiatric rehabilitation skills.
- Utilization of psychiatric rehabilitation skills.

Instruments. Skill acquisition was assessed with a paper-and-pencil instrument that was administered to all trainer-apprentices and trainees before and after training. Skill application (i.e., the extent to which a practitioner faithfully applied the acquired skills *with a client*) was assessed using audiotapes. Because of resistance on the part of the practitioners to providing audiotapes, a large amount of posttraining data were not collected. Even with these difficulties, however, the nine sites mailed in 375 pretraining audiotapes and 191 posttraining audiotapes.

Both the skill acquisition and skill application measures were developed by Center mentors who were experts in psychiatric rehabilitation. They were asked to generate, revise, and refine the content considered critical to all components of the skills being taught. This process of generation and refinement took place over a 4-month period during the first phase of the project. Both the skill acquisition and skill application measures were evaluated by raters trained at the Center to an acceptable level of interrater reliability.

Slight changes in the project between years 1 and 2 affected skill-acquisition comparisons. That is, changes in the instruments used to assess skill acquisition were made just before year 2. As a result, the trainer-apprentices from year 2 could not be compared to other trainer-apprentices and trainees. Comparisons were made, however, from pretraining to posttraining for year 2 trainer-apprentices.

Skill utilization, or the extent to which practitioners and agencies incorporated psychiatric rehabilitation into the ongoing treatment process, was measured three ways: (1) a self-report paper-and-pencil questionnaire at posttest only; (2) an analysis of clinical records before and after the training; (3) an assessment of the programs, staff, clients, and treatment methods before and after the training.

The self-report of skill utilization was made with a 50-item questionnaire administered at posttest only. The participants were asked to indicate the extent to which they needed to learn the 21 distinct psychiatric rehabilitation skills and the extent to which they were using these skills. They were also asked to describe the administrative support provided them to adopt and integrate the newly learned psychiatric rehabilitation skills, a key variable in successful adoption of innovations (Backer, Liberman, and Kuehnel 1986).

The content analysis of clinical recordkeeping was performed by rating samples of records using a formalized scale, which was developed and refined by Center staff based upon psychiatric rehabilitation principles. It was assumed that an innovation that was "embedded" into an organization, and was not merely a superficial alteration, would be evident in the organization's recordkeeping system.

Trainer-apprentices from each site were asked to provide two to four random samples of records for themselves and for each trainee who participated in the project. If the job duties of the trainer-apprentices did not require them to keep clinical records (e.g., a supervisor with no client caseload), then no records were provided. Records were requested that would sample and reflect each major component of psychiatric rehabilitation skills (i.e., rehabilitation diagnosis, planning, and intervention). The review of clinical records was used to determine how congruent they were with the psychiatric rehabilitation approach taught in the project (see table 4 for criteria used).

The final measure of utilization was the same instrument that was previously administered for site

Table 4. Clinical record review criteria

I. Rehabilitation diagnosis

1. There is a statement of an *Overall rehabilitation* or *treatment goal* (i.e., a statement of the specific environment which the client is attempting to maintain or move)
2. Timelines for goal are given
3. The entire problems and goal section is written in language likely to be understood by the client
4. There is evidence of client involvement in the goal (e.g., client signature or place for signature)
5. There is evidence of a *functional assessment*, where client's skill strengths and deficits are defined in relation to the rehabilitation goal
6. Skill strengths and deficits are listed in relation to specific environments in which client is attempting to maintain him/herself or move
7. Skill strengths and deficits are operationally defined (i.e., described in an observable, measurable manner)
8. Deficit behaviors are stated so that the present and needed levels of functioning are indicated
9. Skill strengths and deficits are stated in words likely to be understood by client
10. The entire assessment is written in language likely to be understood by the client

II. Planning

1. There is written *identification of interventions to modify client's skills and environmental resources in order to achieve overall rehabilitation goal*
2. There is a statement of the overall rehabilitation goal written on the plan
3. Skill and resource strengths and deficits are translated into goal statements
4. Goals are prioritized
5. Interventions for each of high priority goals are stated (e.g., skill programming, skill teaching, community resource coordination)
6. Times for interventions are stated (e.g., when intervention begins or ends)
7. A specific person is assigned responsibility for each high priority intervention
8. The entire plan is written in language likely to be understood by the client
9. There is evidence of client involvement in the planning (e.g., signature on plan or place for signature)

III. Interventions

1. There is evidence of *rehabilitation programming* (i.e., a step-by-step plan to achieve goals—client is the one taking active steps)
2. There is evidence of *skill teaching* (i.e., a step-by-step procedure that enables client to learn new skills)

selection purposes. This instrument was used to provide pre-post data about the effect of the training on the agency's programs and structure. The following areas were surveyed:

- Client information.
- Staff information (e.g., client/staff ratio, staff turnover).
- Program information (e.g., assessment procedures, client participation in the assessment, and program goals).
- Training/supervision information.
- Administrative information (e.g., administrative support for the psychiatric rehabilitation approach).

A system was developed to rate the above information. Points were given depending on how closely the agency matched the characteristics of an ideal psychiatric rehabilitation setting. For example, a "positive" answer to the staff/client ratio was a ratio of less than 1:10, while a "neutral" answer was a ratio of 1:11 to 1:16, and a "negative" answer was a ratio of more than 1:16. This assessment was performed before the training and again approximately 2-3 months after the intervention was completed.

Training of Raters. Five individuals (Master's students in the Boston University Psychiatric Rehabilitation Program) were trained to use specified criteria for the ratings of the trainer-apprentices and trainees. Each rater was knowledgeable in the principles and skills of psychiatric rehabilitation and received 20 hours of training specific to the criteria. Samples of mental health workers' skill application, skill acquisition, and clinical records were rated by an expert trainer, and those ratings were used as criteria for the training of raters. Checks after each training session were performed to determine how close to criterion the raters were performing.

Interrater agreement was calculated on the measures of skill acquisition and application by determining the percentage of agreement of each rater with the "expert" rater. Agreements were calculated as follows: agreements/(agreements plus disagreements). Twelve comparisons were made on measures of skill acquisition; seven of these comparisons yielded agreements of 75 percent or more, and five yielded agreements between 64 and 74 percent. Sixteen comparisons were made of interrater agreement on skill application scores; eleven of these comparisons yielded percentages of greater than 75 percent, and five yielded agreements of between 50 and 65 percent.

The clinical review scoring was also examined for interrater agreement. Pearson correlation coefficients were computed using four samples of clinical records. The ratings of each of the three raters used for the records reviews were compared to the expert. Of the 12 correlation coefficients, seven ratings were between $r = .87$ and 1.00; two were between $r = .49$ and .62; and two were between $r = .30$ and .35.

The raters were unaware of the order of all assessments and the record reviews; i.e., they didn't know whether the data derived from pretraining or posttraining periods.

Results

Acquisition of Psychiatric Rehabilitation Skills. For the trainer-apprentices of year 1 and trainees of year 2, significant improvement in skill acquisition was made from pretraining to posttraining performance in the areas of rehabilitation diagnosis ($t = 18.12$, $df = 94$, $p < .0001$), planning ($t = 10.58$, $df = 100$, $p < .0001$), and intervention ($t = 14.56$, $df = 99$, $p < .0001$). Trainer-apprentices from year 2 made significant gains from pretraining to posttraining in all areas except planning. They improved in their ability to perform a rehabilitation diagnosis, and in two measures of intervention ($t = 5.07$, $df = 7$, $p < .001$; $t = 7.79$, $df = 5$, $p < .001$; $t = 3.61$, $df = 7$, $p < .009$, respectively). In planning, significant gains were not noted ($t = 0.74$, $df = 7$, $p < .48$). One additional measure was used for year 2 apprentice-trainers, that of their ability to formulate an overall rehabilitation goal. They improved significantly in that skill from pretraining to posttraining ($t = 4.21$, $df = 7$, $p < .004$).

Application of Psychiatric Rehabilitation Skills. As a total group, trainer-apprentices and their trainees demonstrated significant improvement from pretraining to posttraining for skill application in each of the three areas of rehabilitation diagnosis, planning, and intervention ($t = 7.98$, $df = 62$, $p < .0001$; $t = 10.71$, $df = 48$, $p < .0001$; $t = 7.38$, $df = 50$, $p < .0001$).

Use of Psychiatric Rehabilitation Skills. For the self-report of utilization, trainer-apprentices and trainees were asked to indicate the extent to which they felt they *needed* to learn each of 21 psychiatric rehabilitation skills. One hundred utilization questionnaires were completed. Level of need was rated on a 5-point scale ranging from no need (1.0) to great need (5.0). The individual skills (and their mean level of need) viewed as most "needed" were:

- Identifying skill strengths and deficits in relation to a rehabilitation goal ($M = 4.0$).
- Defining goals in relation to specific environmental situations ($M = 3.91$).
- Defining skill strengths and deficits in observable, measurable terms ($M = 3.91$).
- Developing teaching plans ($M = 3.88$).
- Defining an overall rehabilitation goal ($M = 3.87$).

In response to the question about the extent to which the trainees were using psychiatric rehabilitation skills, the following skills had the highest mean score:

- Involving the client in the process of rehabilitation ($M = 4.61$).
- Responding to the client's feeling and problems ($M = 4.52$).
- Identifying skill strengths and deficits in relation to a rehabilitation goal ($M = 4.31$).
- Defining goals in relation to specific environmental situations ($M = 4.30$).
- Providing feedback opportunities ($M = 4.25$).

There was some convergence in the skills perceived as needed and as being used. Two of the five skills perceived as most needed were also rated as being used to the greatest extent. Curiously, the skill of "developing teaching plans" ranked high in perceived need but was not perceived as being greatly used.

To the remaining questions on use of skills, the trainees and trainers-apprentices were asked to indicate to what extent they perceived readiness, support, and a strategy to adopt the psychiatric rehabilitation approach in their agency. Mean scores to these nine items fell into the "some extent" to "moderate extent" categories. The lowest mean responses were to questions of whether it was "manageable to adopt the new skills" and "the extent to which the agency had adopted the principles and practices of psychiatric rehabilitation."

In the second measure of utilization, a total of 307 samples of clinical records were received by the Center and were sufficiently complete for rating. The samples of records were obtained before the training and again 1-1½ years after the training ended. The Center allowed 1½ years to elapse after the training for the posttraining record samples because clinical records were expected to be the last medium to reflect the adoption of an innovation. Due to the large amount of staff turnover between the pretraining and posttraining record reviews, however, statistical analysis comparing the two sets of ratings could not be undertaken. Therefore, a percentage of gain score for each component of the record review was computed using the following formula:

$$\frac{\text{Post Mean Rating} - \text{Pre Mean Rating}}{\text{Pre Mean Rating}}$$

The rehabilitation diagnosis record review had a mean of .58 on pretraining ($n = 85$) and a mean of 1.19 ($n = 43$) at posttraining (range = 0.0–2.4) for a gain score of 105 percent. The planning record review had a mean of .69 before ($n = 127$) and a mean of 1.60 after training (range = 0.0–2.2) for a gain score of 132 percent. Finally, the intervention portion of the record review had a mean on pretraining of 1.95 ($n = 10$) and a mean of 2.54 ($n = 19$) on posttraining (range = 0.0–3.0) for a gain score of 30 percent. For the record samples of trainer-apprentices and trainees who remained in the agency, paired t tests were conducted on rehabilitation diagnosis and planning samples. Significant improvements were noted in both record reviews ($t = 2.72$, $df = 12$, $p < .02$; $t = 8.55$, $df = 32$, $p < .001$). No pretraining-posttraining samples of the intervention samples were available for analysis.

Impact of Training on Program Structure. Comparing the sites to an "ideal" program structure yielded a mean of 16.75 at pretraining and a mean at posttest of 22.75, out of a possible 40 points. A paired t test suggested that significant changes occurred in agency structure ($t = 1.97$, $df = 7$, $p < .05$).

Discussion

The major conclusion from this study is that psychiatric rehabilitation skills can be effectively transferred via a "training of trainers" approach. That is, staff from carefully selected agencies can learn psychiatric rehabilitation skills and then learn how to teach them to their own staff. Our experience indicates, however, that intensive consultation is required to help agencies adopt this innovation. The amount and duration of followup assistance required to implement psychiatric rehabilitation in these nine sites was more extensive than originally expected. Analysis of the utilization questionnaire suggested that the trainer-apprentices and trainees also perceived the process of innovation as a difficult one.

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Our experience corroborated what was suggested by the utilization literature—namely, that innovations such as this are complex and can change the very fabric of an organization. Change must occur at all levels of an organization if an innovation is to be permanent and not just superficial. The principles of utilization listed in table 1 provide an accurate and comprehensive picture of what an agency and a change agent face during the planning and implementation of any meaningful innovation. All of the variables outlined by Backer, Liberman, and Kuehnel (1986) were critical to the innovation process, especially the need for organizational support and persistent championship of the innovation.

Additional findings which emerged from this 3-year project can be summarized as follows:

1. The number of inquiries and applications to the project indicated a growing interest in psychiatric rehabilitation as a treatment modality for individuals who are severely psychiatrically disabled (Mosher 1986).

2. An analysis of applicant data indicated an ongoing need for awareness about psychiatric rehabilitation as a treatment approach (Dincin 1981; Lamb 1982; Kelner 1984).

3. The rigorous site selection process contributed to the Center's understanding of site readiness for use of psychiatric rehabilitation skills. The philosophy of an agency, its rate of staff turnover, its programmatic sophistication, and the value it places on staff development are critical to implementing psychiatric rehabilitation approaches. A selection process must take these factors into account.

4. Accurate selection of agencies for "training the trainers" is not possible without a site visit where one can observe and assess the organization, and potential trainers and trainees (Farkas, Cohen, and Nemec 1986). While the written site selection application provided helpful information, it was not sufficient to judge the motivation and capacity of the personnel.

5. The initial 5 days of training in psychiatric rehabilitation skills at Boston University served a readiness function, but failed to make the trainers-apprentices totally proficient in the skills. Most trainer-apprentices did become sufficiently motivated to acquire the skills by practice at their agency with feedback from the Center's trainers.

6. Learning how to teach the psychiatric rehabilitation skills allowed the trainer-apprentices to gain substantially in their understanding of psychiatric rehabilitation. It seems as if the adage is true: "The best way to learn is by teaching."

7. Training by the trainer-apprentices at their sites proved to be the most difficult task. The Center assumed that knowing the psychiatric rehabilitation skills and knowing how to teach them would be sufficient. However, because of resistance to innovation, the trainer-apprentices also had to learn how to mobilize the political system in their agency.

8. The most effective part of the training at the sites was the weekly "study group" concept carried out at each site. The most difficult part of the training appeared to be the intervention skills. The agencies seemed to adopt the rehabilitation diagnosis and planning phases of the approach more readily than the intervention of teaching skills directly to clients. This intervention may be particularly difficult because it requires the greatest shift by practitioners from their typical roles and practices.

9. Without question, the training at the sites raised the energy level of the agencies but also produced many crises in the staff (e.g., "What if I don't want to do rehabilitation?").

10. Staff turnover and resistance to research projects greatly affect the data collection process (Marlowe, Spector, and Bedell 1983). It is unclear whether staff turnover rates changed because of the project, but the literature suggests that major innovations often provoke staff turnover early in the implementation phase.

11. Audiotapes appear to be more useful in determining if skills can be applied than paper-and-pencil questionnaires; that is, trainees may demonstrate acquisition of the skills on paper and still not be able to apply them.

By conducting this utilization project with its comprehensive evaluation, the Center gained insight into how to implement a psychiatric rehabilitation approach and how to refine the psychiatric rehabilitation skills curricula. This project stimulated the production of a series of multimedia training packages designed to teach practitioner skills (Cohen, Farkas, and Cohen 1985).

In addition, the extensive development and testing of personnel and program instruments has made it possible to evaluate the use of training interventions and program consultation. Course work, workshops, and technical assistance in psychiatric rehabilitation can now be assessed by whether people are *doing* things differently as a result of the intervention.

Using audiotapes for feedback and as measures of outcome has become an integral part of the Center's training technology. Individuals who wish to be certified trainers or practitioners in psychiatric rehabilitation must submit audiotapes as evidence of their ability to *apply* skills. An off-campus Master's degree program in psychiatric rehabilitation at Boston University relies extensively on audiotapes of client-practitioner sessions as data for feedback and evaluation. The use of audiotapes in psychotherapy outcome research also has received wider recognition (Cooke and Kipnis 1986). In conclusion, this project demonstrated the effectiveness of a "training of trainers" strategy for promoting the use of psychiatric rehabilitation skills in nine different mental health agencies.

References

Anthony, W.A., ed. Rehabilitating the person with a psychiatric disability: The state of the art. *Rehabilitation Counseling Bulletin,* (Whole Special Issue), 1980.

Anthony, W.A.; Cohen, M.; and Cohen, B. The philosophy, treatment process and principles of the psychiatric rehabilitation approach. *New Directions in Mental Health Services,* 17:67–69, 1983.

Anthony, W.A.; Cohen, M.; and Pierce, R. *Instructor's Guide to the Psychiatric Rehabilitation Practice Series.* Baltimore, MD: University Park Press, 1980.

Anthony, W.A.; Cohen, B.F.; Rogers, E.S.; and Danley, K. *A Project to Demonstrate and Evaluate a Dissemination Strategy for a Psychiatric Rehabilitation Technology: Final Report of NIMH Manpower Research and Demonstration Grant MH-14502-06.* Boston: Center for Psychiatric Rehabilitation, Boston University, 1985.

Anthony, W.A.; Pierce, R.; and Cohen, M. *The Skills of Diagnostic Planning. Psychiatric Rehabilitation Practice Series: Book 1.* Baltimore, MD: University Park Press, 1980*a.*

Anthony, W.A.; Pierce, R.; and Cohen, M. *The Skills of Rehabilitation Programming. Psychiatric Rehabilitation Practice Series: Book 2.* Baltimore, MD: University Park Press, 1980*b.*

Astrachan, B.M., and Tischler, G.L. A systems approach to the management of psychiatric facilities. *Administration in Mental Health,* 8:225–236, 1981.

Bachrach, L. Overview: Model programs for chronic mental patients. *American Journal of Psychiatry,* 137:1023–1031, 1980.

Backer, T.E.; Liberman, R.P.; and Kuehnel, T.G. Dissemination and adoption of innovative psychosocial interventions. *Journal of Consulting and Clinical Psychology,* 54:111–118, 1986.

Beard, J.H.; Probst, R.N.; and Malamud, T.J. The Fountain House model of psychiatric rehabilitation. *Psychosocial Rehabilitation Journal,* 5:47–53, 1982.

Boje, D.M.; Fedor, D.B.; and Rowland, K.M. Myth-making: A qualitative step in OD interventions. *Journal of Abnormal Psychology,* 18:17–28, 1982.

Carolyn, M., and Stoffelmayer, B. Diversity of goals in a State psychiatric hospital. *Administration in Mental Health,* 9:57–66, 1981.

Cohen, M.; Anthony, W.A.; Pierce, R.; and Spaniol, L. *The Skills of Professional Evaluation. Psychiatric Rehabilitation Practice Series: Book 3.* Baltimore, MD: University Park Press, 1980*a.*

Cohen, M.; Anthony, W.A.; Pierce, R.; and Vitalo, R. *The Skills of Community Coordination. Psychiatric Rehabilitation Practice Series: Book 6.* Baltimore, MD: University Park Press, 1980*b.*

Cohen, M.; Farkas, M.; and Cohen, B. *Psychiatric Rehabilitation Trainer Packages: Functional Assessment.* Boston, MA: The Center for Rehabilitation Research and Training in Mental Health, 1985.

Cohen, M., and Nemec, P. *Psychiatric Rehabilitation Trainer Packages: Trainer Orientation.* Boston, MA: The Center for Rehabilitation Research and Training in Mental Health, 1985.

Cooke, M., and Kipnis, D. Influence tactics in psychotherapy. *Journal of Consulting and Clinical Psychology,* 54:22–26, 1986.

Dincin, J. A community agency model. In: Talbott, J.A., ed. *The Chronically Mentally Ill.* New York: Human Sciences Press, 1981. pp. 212–226.

Farkas, M.; Cohen, M.; and Nemec, P. *Psychiatric Rehabilitation Programs: The Gap Between Orientation and Implementation.* Boston, MA: Boston University, Center for Psychiatric Rehabilitation, 1986.

Glaser, E.M. Durability of innovations in human service organizations. *Knowledge,* 3:167–185, 1981.

Glaser, E.M. Preliminary report on a quality of work life (QWL) improvement effort at a community mental health center and a State hospital. *Consultation,* 1:51–56, 1982.

Glaser, E.M., and Backer, T.E. Organization development in mental health services. *Administration in Mental Health,* 6:192–215, 1979.

Goldman, W. Change in a State Department of Mental Health: A view from within. *Administration in Mental Health,* 4:2–9, 1976.

Haveliwala, Y.A. Changing a mental hospital. *Administration in Mental Health,* 7:112–119, 1979.

Jansson, B.S. Policy dissonance and executive preferences: Implications for reform. *Administration in Mental Health,* 7:179–186, 1980.

Kelner, F. A rehabilitation approach to program diagnosis in technical assistance consultation. *Psychosocial Rehabilitation Journal,* 7(3):32–43, 1984.

Lamb, H.R. *Treating the Long-term Mentally Ill.* San Francisco: Jossey-Bass, 1982.

Liberman, R.P.; King, L.W.; DeRisi, W.J.; and McCann, W. *Personal Effectiveness.* Champaign, IL: Research Press, 1975.

Liberman, R.P.; Eckman, T.; Kuehnel, T.; Rosenstein, J.; and Kuehnel, J. Dissemination of new behavior therapy programs to community mental health centers. *American Journal of Psychiatry,* 139:224–226, 1982.

Liberman, R.P.; Kuehnel, T.G.; Phipps, C.C.; and Cardin, V.A. *Resource Book for Psychiatric Rehabilitation.* Washington, DC: National Institute of Handicapped Research, 1984.

Marlowe, H.A.; Spector, P.E.; and Bedell, J.R. Implementing a psychosocial rehabilitation program in a State mental hospital: A case study of organizational change. *Psychosocial Rehabilitation Journal,* 6(3):2–11, 1983.

Mosher, L. The current status of the Community Support Program: A personal assessment. *Psychosocial Rehabilitation Journal,* 9(3):3–14, 1986.

Passmore, W.A., and King, D.C. Understanding organizational change: A comparative study of multifaceted interventions. *Journal of Abnormal Psychology,* 14:455–468, 1978.

Pierce, R.; Cohen, M.; Anthony, W.A.; and Cohen, B. *The Skills of Career Counseling. Psychiatric Rehabilitation Practice Series: Book 4.* Baltimore, MD: University Park Press, 1980*a*.

Pierce, R.; Cohen, M.; Anthony, W.A.; and Cohen, B. *The Skills of Career Placement. Psychiatric Rehabilitation Practice Series: Book 5.* Baltimore, MD: University Park Press, 1980*b*.

Stern, R., and Minkhoff, K. Paradoxes in programming for chronic patients in a community clinic. *Hospital & Community Psychiatry,* 30:613–617, 1979.

Weinman, M.L.; Grimes, R.M.; Hsi, B.P.; Justice, B.; and Schoolar, J.C. Organizational structure and effectiveness in general hospital psychiatry departments. *Administration in Mental Health,* 7:32–42, 1979.

White, P.E. Resources as determinants of organizational behavior. *Administrative Science Quarterly,* 19:119–125, 1974.

Wolkon, G.H.; Peterson, C.L.; and Gougla, P. University-based continuing education and mental health system change. *American Psychologist,* 37:966–970, 1982.

Zand, D.E., and Sorenson, R. Theory of change and the effective use of management science. *Administrative Science Quarterly,* 20:532–545, 1975.

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Discussion: What Does Rehabilitation Accomplish?

by John S. Strauss

Abstract

It is often assumed that rehabilitation is limited to helping schizophrenic patients compensate for deficits that are either immutable or altered only by specific medical treatment. However, recent advances in knowledge require that this basic, constricting, and poorly founded assumption about the limitations of rehabilitation be challenged. Data now exist to suggest that for severe mental disorders like schizophrenia rehabilitation may not be just palliative or help compensate for deficits, but may contribute in important ways to basic recovery processes.

Having just viewed the television telling us again that schizophrenia is a brain disease, and having just attended several conferences where that message was also promulgated, I think back to Adolf Meyer's response to Kraepelin's similar view. Meyer (1948) stated, in essence, if dementia praecox (schizophrenia) is organic, where is the evidence? Although important progress has been made in tracing possible organic—and psychological and social—aspects of schizophrenia, the level of evidence does not begin to support many of the assumptions made.

Is this polemic relevant to a *Schizophrenia Bulletin* issue on rehabilitation? Clearly, I think so. The impressive advances in the rehabilitation field documented in this issue and the growing respect for those advances and their importance require an increasingly close look at what rehabilitation accomplishes. This in turn requires careful inspection of some of the major assumptions made about the nature of mental disorders, their course, and how this course is altered. Is rehabilitation just "what to do until the doctor comes" (or discovers a cure), or does rehabilitation actually contribute to the healing process? The articles in this issue focus especially on clarifying approaches to rehabilitation and developing improved research. These are important goals, but they also require a third goal—an understanding of what rehabilitation accomplishes. It is this goal on which I would like to focus.

The "vulnerability-stress-coping-competence" model described by Anthony and Liberman (1986) supplies a valuable beginning for such a consideration. The model has the major role of providing broader categories for thinking about disorder and recovery than can a narrow version of a disease model. But the very breadth of these categories requires an even more basic and more specific empirically based conceptualization. The problem, of course, is to come up with such a conceptualization that also recognizes our considerable ignorance in the field. No one needs just another premature "answer"—whether organic, psychoanalytic, or social (to alienate members of a wide range of schools)—building a conceptual solution on extremely limited data.

Some more specific aspects of a conceptualization about schizophrenia can be suggested by information now available on course and outcome. There are by now copious data indicating that schizophrenia is an extremely complex disorder with a complex course. Kraepelin's (1915) hypothesis that people with schizophrenia almost

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invariably deteriorate continues to be accepted as accurate by many in the field, although there are no systematic data supporting this assumption. There is, however, a growing body of information showing that, over extended periods of time, significant percentages of people with schizophrenia improve markedly or recover completely (Bleuler 1974; Ciompi 1980; Harding et al., in press). There is also ample evidence to show that the disorder, instead of having entirely endogenous mechanisms, interacts in important ways with the person's social functioning and the environment. Premorbid social function continues to be one of the most robust predictors of outcome (Phillips 1953; Strauss et al. 1977). And some of the most solid data in the schizophrenia field relate to the environmental impacts of family environment (Brown, Birley, and Wing 1972). There is also research and clinical evidence to suggest that work, a range of social supports, and perhaps stressful life events also play a major part in the course and outcome of schizophrenic disorders (Kraepelin 1915; Day 1981; Strauss et al. 1981).

Citing the impact of these various areas of function and environmental characteristics on the course of schizophrenia is not merely for the purpose of substantiating that this is a complex disorder and that psychological and social factors are involved. These complexities are major clues to the nature of the disorder itself, clues that are ignored at great risk to those who have schizophrenia, want to understand it, or hope to treat it.

But it is difficult to proceed further toward a more precise conceptualization. The data, although copious and complex, do not fit easily into any detailed coherent explanation, certainly not any unitary explanation, such as viewing schizophrenia as totally organic, as totally a function of psychological development, or as totally a result of social labeling or learning processes. Our knowledge and its shortcomings do, however, have important implications for what conclusions we should and should not reach about the accomplishments and supposed limitations of rehabilitation.

The rehabilitation approach to severe mental disorders including schizophrenia has been an extremely important movement in the field and just now is becoming recognized for the tremendous contributions it can make. But the rehabilitation model may also emphasize its limits more than is warranted. There has been a tendency to understand the rehabilitation of severe mental disorders in a very restricted way: "Rehabilitation is not treatment but an approach to helping people compensate for impairments." The rehabilitation field often seems to be saying that rehabilitation professionals are not (usually) physicians, but people who work with a range of severe physical and mental illnesses helping patients function to the degree to which they are able.

But in the rehabilitation paradigm for mental disorders, as with so many conceptualizations for which there are limited data, implicit metaphor is used to cover gaps in our knowledge, ambiguities, and a wide range of uncertainty (Wynne, personal communication; Lakoff and Johnson 1980). The implicit metaphor here is that schizophrenia is a disease, and that rehabilitation doesn't treat disease but helps people function optimally with the deficiencies they have. However, in schizophrenia and in other major mental disorders as well, we do not know that rehabilitation is limited in this way. Perhaps it is in some instances, and perhaps it isn't in others. In some instances rehabilitation may even be treatment. Perhaps it helps people recover from the disorder itself.

It is now clear that some people with schizophrenia do improve significantly and that others recover. What is not clear is the nature and origins of these processes of improvement. Just as the rehabilitation models and the disease models, when interpreted in certain ways, are metaphors for what we think may be happening, so too the metaphor of healing may also be useful in schizophrenia. The question would then become what facilitates the healing processes.

At least two directions for answering this question are possible. One direction is that suggested by the article by Spaulding et al. in this issue. In their inquiry into processes underlying various areas of dysfunction in schizophrenia, Spaulding et al. usefully attempt to get beyond and below the more superficial behavioral characteristics. This is one valuable approach. Although over the years it has been extremely difficult to identify what such basic processes might be, this problem may in part have been a result of our limits in methodology and the fact that we may be dealing with heterogeneous types of dysfunction and heterogeneous disorders. Nevertheless, as Spaulding et al. state, recent psychophysiological research may be introducing useful advances toward clarifying basic processes in schizophrenia.

The other direction for understanding the processes underlying improvement when it occurs in schizophrenia is of particular relevance to clarifying the accomplishments of rehabilitation.

That direction is the pursuit of clues from clinical experiences and from broadly framed clinical studies of patients. Data from such sources suggest two kinds of processes that may be important factors in recovery, central to understanding the accomplishments of rehabilitation, and all but neglected in most systematic research. These two processes focus on the personal side of schizophrenia—the efforts that the person makes on his or her own behalf, the role of meanings, and the sense of identity the person has.

There is growing evidence that the person, as a person, is an important part of the course and outcome of schizophrenia. Regarding the issue of the person's acting on his or her own behalf, there are data to suggest that people with schizophrenia may play an important role in controlling their own symptoms (Liberman 1982; Breier and Strauss 1983; McCandless-Glimchen et al. 1986), that they modulate and select the kind of help they get from other people (Breier and Strauss 1984), and, of course, choose to collaborate with treatment, seek out or avoid social supports and stressful situations, and withdraw or take risks to improve their functioning.

Regarding the role of personal meaning and the person's sense of his or her identity, there is clinical evidence, but almost no systematic study that suggests that these factors are also crucial. Even in television programs about schizophrenia, there is an occasional interview that goes beyond showing how bizarre people with schizophrenia can be, to an interview in which a patient describes how he or she used to be and how now there are still longings, a sense of self, and fears that have a massive impact on whether and how the person attempts to improve or even function. The neglect of such areas by the field is reflected, for example, in the observations by Lukoff et al. in this journal in their valuable report on sex education for people with schizophrenia. Sexual functioning in schizophrenia has been almost entirely neglected by professionals who may even give a "collective gulp" when explicit sexual questions that can be asked of such patients are enumerated. This is only one area of dealing with individuals who have schizophrenia as people that tends to be overlooked in mental health practice and research.

In coping with schizophrenia, we are working with people who have often lost their sense of self. Kraepelin (1915) noted their lack of "will" and Bleuler (1911/1950) the ways in which their goal-directed thoughts, desires, and feelings got disconnected as part of the loosening in associations. Many patients with schizophrenia, if given the opportunity, describe their efforts to get themselves "together" again. Perhaps one of the accomplishments of rehabilitation when it is effective is that it helps the person do just that. Rehabilitation, with its potential for helping the person be goal directed and develop a socially acceptable identity, at least as much as other treatment approaches, may contribute to such integration.

Thus, rehabilitation, with its increasingly valuable role in the field, may have given in too soon to the sense that "we are only doing rehabilitation." In view of the current considerable ignorance about the underlying nature and factors influencing the course of schizophrenia, the rehabilitation field needs to pursue actively the possibility that it can help with the basic process of reintegration and that this process may be a major aspect of healing and perhaps even recovery.

These thoughts are not intended to be harshly critical of those interested in rehabilitation—a group that has helped people with schizophrenia live meaningful, productive, and happier lives, and whose progress is represented in this issue. Instead of devaluing the crucial work of rehabilitation, I would like to praise but encourage, motivate, and even spread some guilt to push further those involved in rehabilitation research and practice to integrate their work and training more completely with the notion of the person as a person and with an inquiring mind about the still unknown nature of mental disorders. This might be accomplished by the use of a dynamic orientation—not by any means necessarily psychoanalytic—focusing on the process of change. This focus would involve attention to simple, functional roles and behaviors but would also include the role of functioning in promoting the healing of psychological abnormalities including unembarrassed attention to meanings and identities. One does not have to be soft-headed to look at the possible role of functioning, personal meaning, and identity in the recovery process. In fact, it is probably better not to be too soft-headed about them or too credulous.

But clues to the rôle functioning serves in recovery from mental disorders and to the importance of identity and meaning are present for those who have direct contact with patients to see. We need to move toward these and learn from them, exploring more empirically and with an open mind ways in which rehabilitation can be treatment as well as helping people to compensate for deficits. And specifically, it is important to consider ways in which rehabilitation may help the person in the basic process of reintegration which has been, as most theorists including Kraepelin, Bleuler, and Freud agree, one of the basic problems of even such a severe disorder as schizophrenia.

References

Anthony, W.A., and Liberman, R.P. The practice of psychiatric rehabilitation: Historical, conceptual, and research base. *Schizophrenia Bulletin*, 12:542–559, 1986.

Bleuler, E. *Dementia Praecox or the Group of Schizophrenias.* (1911) Translated by J. Zinkin. New York: International Universities Press, 1950.

Bleuler, M. The long-term course of the schizophrenic psychoses. *Psychological Medicine*, 4:244–254, 1974.

Breier, A., and Strauss, J.S. Self-control in psychiatric disorders. *Archives of General Psychiatry*, 40:1141–1145, 1983.

Breier, A., and Strauss, J.S. Social relationships in the recovery from psychotic disorder. *American Journal of Psychiatry*, 141:949–955, 1984.

Brown, G.W.; Birley, J.L.T.; and Wing, J.K. Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, 121:241–258, 1972.

Ciompi, L. The natural history of schizophrenia in the long term. *British Journal of Psychiatry*, 136:413–420, 1980.

Day, R. Life events and schizophrenia: The "triggering" hypothesis. *Acta Psychiatrica Scandinavica*, 64:97–122, 1981.

Harding, C.; Brooks, G.W.; Ashikaga, T.; Strauss, J.S.; and Breier, A. The Vermont longitudinal study: II. Long-term outcome for *DSM-III* schizophrenia. *American Journal of Psychiatry*, in press.

Kraepelin, E. *Clinical Psychiatry: A Textbook for Students and Physicians.* 7th ed. Translated by A.R. Defendorf. New York: MacMillan, 1915.

Lakoff, G., and Johnson, M. *Metaphors We Live By.* Chicago: University of Chicago Press, 1980.

Liberman, R.P. What is schizophrenia? *Schizophrenia Bulletin*, 8:435–437, 1982.

Lukoff, D.; Gioia-Hasick, D.; Sullivan, G.; Golden, J.S.; and Nuechterlein, K.H. Sex education and rehabilitation with schizophrenic male outpatients. *Schizophrenia Bulletin*, 12:669–677, 1986.

McCandless-Glimchen, L.; McKnight, S.; Hamera, E.; Smith, B.L.; Peterson, K.A.; and Plumlee, A.A. Use of symptoms by schizophrenics to monitor and regulate their illness. *Hospital and Community Psychiatry*, 37:929–933, 1986.

Meyer, A. Fundamental conceptions of dementia praecox. In: Lief, A., ed. *The Common Sense Psychiatry of Adolf Meyer.* New York: McGraw-Hill, Inc., 1948. pp. 184–192.

Phillips, L. Case history data and prognosis in schizophrenia. *Journal of Nervous and Mental Disease*, 117:515–525, 1953.

Spaulding, W.; Storms, L.; Goodrich, V.; and Sullivan, M. Applications of experimental psychopathology in psychiatric rehabilitation. *Schizophrenia Bulletin*, 12:560–577, 1986.

Strauss, J.S.; Kokes, R.F.; Klorman, R.; and Sacksteder, J. Premorbid adjustment in schizophrenia. *Schizophrenia Bulletin*, 3:182–244, 1977.

Strauss, J.S.; Loevsky, L.; Glazer, W.; and Leaf, P. Organizing the complexities of schizophrenia. *Journal of Nervous and Mental Disease*, 169:120–126, 1981.

Wynne, F. Personal communication, November 3, 1985.

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Autism and Genetic Disorders

by Allan L. Reiss, Carl Feinstein, and Kenneth N. Rosenbaum

Abstract

The syndrome of autism has been documented as occurring in association with a wide variety of genetic conditions. Autistic patients with a coexistent genetic condition, however, are not behaviorally or developmentally distinct from autistic patients for whom there is no known etiology or associated organic condition. This report reviews the literature linking autistic behavior with genetic conditions. Genetic, neurodevelopmental, and neuropathological findings in three genetic conditions which frequently give rise to autism are presented in detail. On the basis of this review, two hypotheses are supported: (1) autism is a behaviorally defined phenotype which arises from diverse causes of central nervous system (CNS) damage, and (2) the autistic phenotype represents only one point along a continuum of psychological dysfunction resulting from CNS damage. Current theories of genetic influences on brain development are reviewed, with emphasis on the relationships among qualitative, quantitative, and temporal abnormalities of CNS maturation and behavioral dysfunction. A hypothesis of abnormal brain development resulting from dysfunctional myelination is proposed as a potential etiologic factor in autism.

It is generally accepted that autism is a chronic debilitating mental illness resulting from central nervous system (CNS) dysfunction. Essential features of the syndrome include disturbances of language, cognition, perception, relatedness, motility, and development (Ornitz and Ritvo 1976). It is unsettled whether autism is a unitary disease with a single etiology or a behavioral phenotype with multiple, heterogeneous causes. The latter hypothesis is supported by the diversity of organic conditions that appear to be causally related to the development of autistic behavior. These include infections such as encephalitis or congenital rubella (Chess 1971; DeLong, Bean, and Brown 1981), seizure disorders, genetic conditions like fragile X syndrome or tuberous sclerosis (Mansheim 1979; Meryash, Szymanski, and Park 1982), metabolic disorders such as phenylketonuria or histidinemia (Friedman 1969; Kotsopoulous and Kutty 1979), and a variety of other conditions which affect brain development and function (Coleman 1976; Ornitz 1983).

Some authors believe that even though autistic patients with organic conditions demonstrate classical autistic behavioral patterns, they should be distinguished from autistic individuals for whom there is no known etiology or associated condition (Rimland 1971; Eisenberg 1972). Autistic patients with a coexisting organic condition or neurological symptoms are not, however, behaviorally or developmentally distinct from autistic patients without such features (Knobloch and Passamanick 1975; Ornitz, Guthrie, and Farley 1977; Garreau, Barthelemy, and Sauvage 1984).

The syndrome of autism may represent only one point on a continuum of cognitive, perceptual, and linguistic dysfunction resulting from specific types of genetically and/or environmentally induced

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brain damage. Alternate manifestations of such damage could include specific developmental disorders, uncomplicated mental retardation, hyperactivity, linguistic deficits, a "partial" autistic syndrome, or nonautistic psychosis. These conditions are present in many individuals with organic syndromes which frequently produce autistic behavior such as the fragile X syndrome (Hagerman, McBogg, and Hagerman 1983) or congenital rubella (Chess 1971). They are also present in relatives of patients with idiopathic autism. Siblings of these children manifest cognitive and linguistic disorders with much greater frequency than the general population (Rutter, Bartak, and Newman 1971; August, Stewart, and Tsai 1981; Minton et al. 1982). In their study of twins of autistic children, Folstein and Rutter (1977) reported a 38 percent concordance rate for autism in monozygotic pairs. This figure was increased to 82 percent when the definition of concordance was broadened to include any form of cognitive or language disorder (including autism). The authors concluded that the "genetic factor" in their patient population gave rise to a vulnerability to cognitive/linguistic dysfunction with autism representing only one possible manifestation.

On the basis of the preceding observations, two hypotheses are suggested: (1) autism is a behaviorally defined phenotype which arises from diverse causes of central nervous system (CNS) damage as opposed to a homogeneous, distinct disease entity, and (2) the phenotype represents only one point along a continuum of psychological dysfunction resulting from CNS damage instead of a separate category of psychopathology. If the first hypothesis is valid, any endeavor to uncover a *common* genetic determinant from a population of autistic patients would be an impossible task. To date, such attempts have yielded inconsistent or negative results (Hanson and Gottesman 1976; Spence 1976). Further inquiry into the genetic and neurobiological determinants of autism, then, requires an alternate route of investigation. We, therefore, chose to review the literature on known genetic disorders reported to be associated with autism. This approach was chosen in the hope that the biochemical, neurodevelopmental, and pathophysiological data from previous studies of the genetic conditions would yield common neurobiological denominators relevant to autism, and help to elucidate the pathway from gene to behavior.

Reports of genetic conditions in which autistic behavior has been described are presented in tabular form. We have included those reports in the literature in which the diagnosis of autism was made with reference to an accepted diagnostic instrument, or where enough details of the case were provided for the *DSM-III* diagnosis (American Psychiatric Association 1980) to be made post hoc by the authors. The designation of "probable autism" is used to describe cases in which neither of these criteria are satisfied fully, but the diagnosis is strongly suspected from the information available. To concentrate on conditions in which relevant genetic and neurobiological information may be available, we limited this review to autistic patients with chromosomal abnormalities, single gene disorders, and syndromes of unknown etiology that are strongly suspected of having a genetic basis.

Literature Review

Chromosomal Disorders (table 1). The overwhelming majority of autistic children with chromosomal abnormalities reported in the literature have sex chromosome anomalies, particularly the fragile X syndrome. Autistic behavior occurs infrequently in autosomal disorders and is not consistently associated with any single chromosome or chromosome group.

The specific chromosome abnormality in the fragile X syndrome is detected by visualizing a fragile site on the X chromosome when cells are grown in folate-deficient or other specially modified media (Turner and Jacobs 1983). The frequency of the fragile X syndrome in the general population has been reported to be as high as 0.9 per 1,000 live male births (Herbst and Miller 1980), making it at least the second leading cause of mental retardation due to a chromosomal abnormality after Down's syndrome. Studies investigating psychological abnormalities in *populations* of patients with the fragile X syndrome have described from 4 to 60 percent of affected males as autistic (Turner, Daniel, and Frost 1980; Brown et al. 1982; Levitas et al. 1983; Nielson 1983; Fryns et al. 1984) Although there may be some common characteristic physical features associated with this syndrome such as large ears or macroorchidism (Hagerman, McBogg, and Hagerman 1983), many patients may not have any distinguishing features except for their behavioral dysfunction. One study has reported on the presence of the fragile X chromosome in a large autistic population (Watson et al. 1984). Out of 76 autistic males, four (5.4 percent) demonstrated the fragile X marker.

Table 1. Chromosomal disorders

Genetic condition	Study	Autistic patients (*n*)	Specific features of patients identified
XYY	Abrams & Pergament (1971)	1, case report	Hypertelorism, high arched palate
	Nelson, Christensen & Friedrich (1973)	1, identified from 22 patients with XYY	
	Mallin & Walker (1972)	1, case report	Patient and brother (XY) had congenital adrenal hyperplasia
XXYY	Ornitz, Guthrie & Farley (1977)	1, identified from 74 autistic patients	Neonatal distress, hypotonia at birth
XXY	Campbell, Wolman & Brewer (1972)	1, case report	Self-injurious behavior
XXX	Wolraich et al. (1970)	1, identified from 25 autistic patients	Probable autism
Fragile X syndrome	Levitas et al. (1983)	6, identified from 10 fragile X patients	
	Brown et al. (1982)	5, identified from 22 fragile X patients	
	Turner, Daniel & Frost (1980)	1, identified from 25 fragile X patients	Probable autism
	Nielson (1983)	1, identified from 27 fragile X patients	Probable autism
	Fryns et al. (1984)	3, identified from 21 fragile X patients	Probable autism
	Watson et al. (1984)	4, identified from 76 autistic males	
	August (1983)	2, case reports	
	Gillberg (1983)	3, case reports	Patients are identical triplets
	Meryash, Szymanski & Park (1982)	3, case reports	
	Reiss (1984, unpublished data)	2, case reports	
"Large" Y chromosome	Hoshino et al. (1979)	9, identified from 32 autistic patients	Patients with large Y had less severe autistic behavior and language disturbance compared to patients with normal Y
	Judd & Mandell (1968)	3, identified from 8 autistic patients	

Table 1. Chromosomal disorders—*Continued*

Genetic condition	Study	Autistic patients (*n*)	Specific features of patients identified
Trisomy 21	Wakabayashi (1979)	1, case report	
	Maltz (1979)	1, case report	
	Knobloch & Passamanick (1975)	1, case report	
Trisomy 22	Turner & Jennings (1961)	1, case report	Probable autism
t(22,13) (translocation)	Hansen et al. (1977)	1, case report	Severe mental retardation, seizures, abnormal electroencephalogram, ataxia, hypotonic
Gp + (increased chromosomal material from a G chromosome)	Crandall, Carrel & Sparkes (1972)	1, identified from 700 outpatients referred to child psychiatry	IQ = 45, chromosome abnormality is probable normal variant
3p − (deletion of material from chromosome ^{3})	Hagerman (1984, unpublished data)	1, case report	Prominent ears, pectus excarotum, probable autism
5p +	Hagerman (1984, unpublished data)	1, case report	Hyperreflexia, tremor, probable autism
8p −	Hagerman (1984, unpublished data)	1, case report	Multiple dysmorphic features, probable autism
17p −	Hagerman (1984, unpublished data)	1, case report	Hypertelorism, low-set ears, supernumamerary digit
Increased chromosome breakage	Siva San Kar (1970)	Population of 31 autistic patients	

Two studies (Judd and Mandell 1968; Hoshino et al. 1979) have found an association between large size of the Y chromosome and autism. Large chromosome size is correlated with the presence of increased repetitive DNA sequences (McKay, Bobrow, and Cooke 1978). Increased size of the Y chromosome is particularly of interest because of the high male-to-female ratio seen in autism and the association of autism with the XYY karyotype (Abrams and Pergament 1971; Mallin and Walker 1972; Nelson, Christensen, and Friedrich 1973). Considerable variation in the size of the Y chromosome within populations is a normal finding, however (Emery and Rimoin 1983).

Increased chromosome breakage in autistic children compared with other psychiatric populations (Siva San Kar 1970) is also of unknown significance. Increased chromosome breakage is seen in several genetic syndromes (Emery and Rimoin 1983), viral infections, and secondary to ionizing radiation and certain drugs (Jackson and Schimke 1979). In the single study reporting this finding (Siva San Kar 1970), detailed information pertaining to medical history, number of chromosome breakages per patient, or location of the breakage sites was not provided.

Single Gene Disorders (table 2). The list of single gene disorders associated with autism includes a diverse group of autosomal dominant and recessive conditions, each of which gives rise to a broad range of cognitive, language, and behavioral abnormalities in affected individuals. Most of the reported cases, though, come from the well-known association of autism with phenylketonuria (PKU). PKU is an autosomal recessive disorder caused by the inborn deficiency of the enzyme, phenylalanine hydroxylase. Patients with PKU are unable to metabolize normal amounts of phenylalanine in the diet, leading to an accumulation of this substance throughout the body. A number of factors resulting from the inability to metabolize phenylalanine are presumed to cause the psychiatric and neurological symptoms seen in untreated patients (Menkes 1980).

In his review of PKU and chromosomes, Friedman (1969) reported that most studies analyzing behavior of untreated patients described a large percentage of affected individuals as manifesting part or all of the autistic syndrome. In a separate report (Knobloch and Passamanick 1975), the incidence of PKU in an autistic population identified in a "pediatric developmental service" was 21.9 percent (14 out of 64). Lowe et al. (1980) screened 65 children with "pervasive developmental delays" for PKU, including 50 children with autism. Three cases were discovered, including one 2-year-old child who had reportedly had a negative test for PKU shortly after birth. Hackney et al. (1968) studied 46 children with documented PKU and reported that nine were autistic. These reports imply a much greater association between PKU and autism than would be expected if these two disorders were not causally related.

In a review of neuropathological findings in patients with PKU, Malamud (1966) emphasized abnormalities in myelination as the most consistent abnormality found in the brain. The pattern of CNS tissue damage described was one of white matter vacuolation, also called "status spongiosis," and frank areas of demyelination. Spongy white matter lesions, seen in all patients, consistently occurred in the central, subependymal, and periventricular areas of the cerebrum, the cerebellum, the optic tracts, and the fornix. Scattered lesions were also seen in the basal ganglia, brainstem, and spinal cord. Well-demarcated areas of demyelination were localized in lateral-central and adjacent gyral cerebral white matter and in the cerebellum, but were only seen in older patients in which the disease process had been ongoing for many years. The extent of demyelination was proportional to the age of the patient, indicating an ongoing pathological process.

Other reports have also described quantitative and qualitative abnormalities in myelin, or decreased amounts of lipid components of myelin in the brains of patients with PKU (Prensky, Carr, and Moser 1968; Shah, Peterson, and McKean 1972; Agrawal and Davison 1973). Recent studies examining animal models have suggested that actual formation of the myelin membrane may not be abnormal in PKU. Instead, damage to the developing brain may occur during the latter stages of neuronal differentiation. In one report (Huether, Kaus, and Neuhoff 1982), alterations in axonal outgrowth from specific neuronal populations, especially in the forebrain, were hypothesized to cause desynchronization in the temporal coordination of the myelination process. Another study (Cordero et al. 1983) found abnormalities in dendritic arborization and cell orientation of pyramidal cells in the rat cortex. In a post-mortem examination of an autistic patient with PKU (Williams et al. 1980), changes in pyramidal cell dendritic spine density were seen, a finding that was also discovered in the examination of an autistic patient without an identifiable organic condition (Williams et al. 1980).

An area of controversy in PKU research relates to the specific developmental stage or stages during which CNS tissue damage occurs. Examination of untreated patients with PKU and animal models indicate that qualitative or quantitative abnormalities in myelination of CNS white matter tracts, disturbed neuronal differentiation, or both arise shortly after birth. These lesions appear to correlate clinically with the development of the most severe psychiatric and neurological deficits in PKU, including autism (Lemire et al. 1975; Menkes 1980). There is also evidence of aberrations in brain myelin and other neurocellular abnormalities arising during prenatal CNS development. These abnormalities may be causally related to milder deficits, such as specific developmental disorders, seen in patients treated with dietary intervention from birth (Melnick, Michals, and Matalon 1981).

Because of similarities in observed pathophysiology, two of the three autosomal dominant disorders, neurofibromatosis and tuberous sclerosis, are considered related. Mental retardation is found in 10-20 percent of children with neurofibromatosis and at least 70 percent of patients with tuberous sclerosis

Table 2. Single gene disorders

Genetic condition	Study	Autistic patients (*n*)	Specific features of patients identified
Autosomnal recessive			
Phenylketonuria	Friedman (1969)	Over 40, described in literature review	Probable autism
	Knobloch & Passamanick (1975)	14, identified from 64 autistic patients	
	Lowe et al. (1980)	2, identified from 50 autistic patients	Original PKU screen negative in one patient
	Hackney et al. (1968)	9, identified from 46 patients with PKU	
	Williams et al. (1980)	1, case report	
Neurolipidosis	Creak (1963)	2, identified from 100 patients with "childhood psychosis"	Diagnosis made at autopsy
Hurler's syndrome	Knobloch & Passamanick (1975)	2, identified from 64 autistic patients	No longer autistic at followup
Histidinemia	Rutter & Bartak (1971)	1, case report	
	Kotsopoulous & Kutty (1979)	1, case report	
Oculocutaneous Albinism	Ornitz, Guthrie & Farley (1977)	1, identified from 74 autistic patients	
Autosomnal dominant			
Neurofibromatosis	Gillberg & Forsell (1984)	2, identified from 26 autistic patients	Both patients had perinatal complications
Tuberous sclerosis	Creak (1963)	1, identified from 100 patients with "childhood psychosis"	
	Lotter (1974)	1, case report	Probable autism, diagnosis at autopsy
	Mansheim (1979)	1, case report	34-week gestation, seizures
	Dick and Ziegler (1967)	1, case report	
	Taft and Cohen (1971)	2, case reports	
	O'Gorman (1970)	1, case report	Probable autism
Noonan syndrome	Paul, Cohen & Volkmar (1983)	1, case report	Probable autism

(Smith 1982). Although patients with tuberous sclerosis have frequently been reported to manifest autistic behavior (Creak 1963; Dick and Ziegler 1967; O'Gorman 1970; Taft and Cohen 1971; Lotter 1974; Mansheim 1979), there have been no systematic behavioral studies of a population of patients with this condition. Autism has also been described in association with infantile spasms (Riikonen and Amnell 1981), a severe form of epilepsy that is often the first presenting sign of tuberous sclerosis.

Neuropathological reports of patients with tuberous sclerosis are dominated by descriptions of the gross anatomy, histology, and ultrastructure of cortical tubers and supependymal nodules (Urich 1976; Bender and Yunis 1980; Trombley and Mirra 1981). The aberrant cell type most often detected in these lesions is considered to be of neuroglial origin (Urich 1976; Trombley and Mirra 1981), although some studies of cellular ultrastructure have found cells with neuronal markers (Bender and Yunis 1980). Neurologia, specifically oligodendrocytes, are the myelin-forming cells in the CNS. There is also evidence of disordered synaptogenesis within these lesions (Bender and Yunis 1980). These findings have been interpreted as being "most consistent with a defect, possibly metabolic, that affects CNS cells at an early stage and results in varying degrees of abnormal differentiation" (Bender and Yunis 1980). With involvement of neuroglial differentiation in tuberous sclerosis, it is not surprising that areas of deficient or disordered myelin are also seen in this disorder. These areas are often diffuse, involve large areas of white matter independent of tuberous or nodular lesions, and can be visualized by computed tomography (CT scan) (Garrick, Gomez, and Houser 1979). Evidence from studies of fetuses and neonates indicates that pathologic changes in the CNS of patients with tuberous sclerosis, like those in PKU, begin before birth (Thibault and Manueldis 1970).

The two cases of autism and one case of childhood psychosis with autistic features associated with neurofibromatosis (Gillberg and Forsell 1984) were discovered from a total of 51 cases of childhood psychoses of unknown etiology, one half of whom were autistic. The authors found the association of childhood psychoses with neurofibromatosis to be 120 times greater than the expected population incidence if these two disorders were unrelated.

Disorders of Unknown Etiology (table 3). Some cases of Cornelia de Lange syndrome (CDLS) have been associated with excess material from chromosome number 3 (Wilson, Hieber, and Schmickel 1978). Individuals with CDLS have a characteristic facial appearance, moderate to severe developmental

Table 3. Disorders of unknown etiology

Genetic condition	Study	Autistic patients (*n*)	Specific features of patients identified
Cornelia de Lange syndrome (CDLS)	Johnson et al. (1976)	7, identified from 9 patients with CDLS	Probable autism
	Nyhan (1972)	3, identified from 3 patients with CDLS	Probable autism
	Knobloch & Passamanick (1975)	1, identified from 64 autistic patients	
Williams syndrome	Reiss et al. (1985)	2, identified from 13 autistic patients	Both patients had elevated serotonin levels
Moebius syndrome	Ornitz, Guthrie & Farley (1977)	1, identified from 74 autistic patients	Club feet, unilateral absence of pectoralis major
Marshall-Smith syndrome	Keith et al. (1972)	1, identified from 4 patients with physical features similar to Marshall-Smith Syndrome	Probable autism

delays, and a wide range of associated physical anomalies (Smith 1982). Two reports have attempted to describe the behavioral phenotype associated with CDLS using systematic analysis of videotape recordings (Nyhan 1972; Johnson et al. 1976). Out of a total of 12 patients studied, 10 clearly met behavioral criteria for autism on all parameters except for age of onset of symptoms, which was not reported. Behaviors that were described included pervasive, qualitative disturbances in social relatedness, severe disturbance of verbal and nonverbal communication, a pattern of abnormal responsiveness to sensory stimuli (twirling and circling behavior, self-mutilation, preference for rotary stimulation), and perseverative, stereotypic motoric behaviors.

As in PKU, a common neuropathological abnormality in CDLS is the presence of deficits in myelination (Berg et al. 1970; Barr et al. 1971; Lemire et al. 1975). One study of a 17-month-old infant with this disorder demonstrated diffuse deficiency in brain myelination, especially prominent in the brainstem (Ptacek et al. 1963). In a postmortem examination of a 5-month-old infant with CDLS at our facility, deficient and delayed myelination was seen in the basal ganglia (unpublished data). Neuropathological reports have also described more severe developmental damage including abnormalities of gyral and sulcal size (Ptacek et al. 1963), microcephaly (Ptacek et al. 1963; Falek, Schmidt, and Jervis 1966), cytoarchitectural abnormalities of the cerebral cortex (Schlesinger et al. 1963; Hart, Jaslow, and Gomez 1965), and pituitary abnormalities (Ptacek et al. 1963; Falek, Schmidt, and Jervis 1966).

Williams syndrome is diagnosed by abnormalities in developmental history, physical appearance, and associated congenital anomalies (Smith 1982). Clinical reports indicate that most patients have abnormalities in social relating, language dysfunction, and cognitive deficits (Jones and Smith 1975).

Discussion

Autism as a Behavioral Phenotype. The heterogeneity of genetic conditions associated with autism supports the hypothesis that the syndrome represents a behavioral phenotype as opposed to a homogeneous disease entity. Many genetic conditions lead to widespread neurological damage and result in significant psychological and behavioral dysfunction (Smith 1982). Autism only occurs in a few of these conditions with a frequency much greater than would be predicted by chance. This suggests that particular genetic disorders are especially likely to cause a pattern of CNS damage that results in the manifestations of the autistic phenotype.

The premise that autism is a behavioral phenotype resulting from different etiologies is supported by studies of patients with idiopathic autism which indicate heterogeneity in this population, as well. Evidence of brain pathology is apparent in these patients. Many demonstrate "hard" signs of neurological dysfunction (Gubbay, Lobascher, and Kingerlee 1970; Maurer and Damasio 1982; Garreau, Barthelemy, and Sauvage 1984). CT scans have revealed increased ventricular size and other abnormalities in gross brain structure in a large percentage of patients (Damasio et al. 1980; Campbell et al. 1982; Gillberg and Svendson 1983; Rosenbloom et al. 1984). The CT abnormalities are quite diverse, however, and inconsistent from one patient to the next, suggesting that different pathological processes are operating in different patients.

These observations indicate that the autistic phenotype is the result of damage to one or more specific functional systems of the brain. This damage can result from a variety of organic or genetic etiologies, some of which remain unidentified. The presence of a recognizable organic condition or signs of CNS dysfunction such as mental retardation, seizures, neurological signs, or ventricular dilation may provide clues as to the etiology, timing, extent, and location of the damage that has occurred.

Autism as a Spectrum Disorder. The hypothesis that the autistic phenotype represents only one point on a continuum of psychological dysfunction is also supported by our review. With almost no exception, each of the genetic conditions we have presented gives rise to a spectrum of cognitive, linguistic, perceptual, and behavioral abnormalities in affected individuals. This is similar to Folstein and Rutter's (1977) twin data from patients with idiopathic autism. The spectrum of deficits can range from minimal or virtually no manifestations to severe, multiple handicapping conditions such as profound mental retardation and/or autism. Conditions such as the fragile X syndrome (Hagerman, McBogg, and Hagerman 1983) and CDLS syndrome (Johnson et al. 1976) have more individuals represented on the "severe" or autistic end of the spectrum, whereas sex chromosome aneuploidy tends to cause milder deficits in most affected individuals (Stewart et al. 1982; Stewart, Netley, and Park 1982).

Genetics, Brain Development, and Autistic Phenotype. The association of a single behavioral phenotype with varying genetic disorders suggests that a specific, functional brain system is vulnerable to damage through a variety of pathological mechanisms. Pathology in genetic disorders is ultimately derived from aberrant genes giving rise to qualitative or quantitative abnormalities in one or more proteins. The genetic disorders associated with autism are vastly different with respect to location and extent of abnormality in gene structure, protein deficiencies, and/or physical manifestations. This suggests that there is not a single protein abnormality that is responsible for producing the pattern of brain dysfunction underlying autistic behavior.

The concept of a single phenotype being associated with different genotypic conditions is well recognized in genetics and can best be illustrated by common physical malformations such as cleft lip or palate. Orofacial clefting is found in at least 154 different genetic conditions (Cohn 1978). It is thought to be caused by pathological disruption of facial morphogenesis before 36 days of gestation (Smith 1982).

It is likely that developing brain structures or CNS systems are also vulnerable to intrinsic or extrinsic disruption during specific, critical time periods. For example, the presence of steroid receptors during particular periods of fetal or early postnatal life is thought to affect neuronal cell survival in specific pathways or structures, cellular differentiation, and development of neurotransmitter systems (Gorski 1978; Kolata 1979). Minor changes in receptor structure, function, timing, or anatomical location could potentially cause specific deficits in functional neuronal connectivity with corresponding behavioral abnormalities (Barlow 1973). By a similar mechanism, disruption of CNS development during one or more critical stages involved in the establishment of perceptual, motoric, language, and related systems could produce the autistic phenotype.

There is indirect evidence which suggests that developmental disruption of the CNS has occurred in at least some autistic children. For example, compared to normal children, autistic patients have a significantly increased incidence of minor physical anomalies (Firestone and Peters 1983). Minor physical anomalies are thought to arise from biological stresses disrupting morphogenesis at specific stages of development. If such anomalies are present, it can be assumed that the developing brain has also been subject to potential developmental interference.

It is likely that many factors influence the severity of psychological deficits that arise in conjunction with particular genetic abnormalities (Gilbert and Opitz 1982). Varying severity of psychological impairment (i.e., a continuum of dysfunction) may be related to variable expressivity of the genetic disorder itself. An example of variable expressivity of physical anomalies is the range of dermatological abnormalities seen in neurofibromatosis. Manifestations range from minor skin lesions to widespread, disfiguring soft tissue tumors. Variable expressivity in females with X-linked conditions such as the fragile X syndrome may be influenced by differential lyonization (X chromosome inactivation) of cells in the CNS. Expressivity can also be affected by the degree of interaction between genetic influences and extrinsic factors.

Disruption of Brain Development in Autism: A Hypothesis. Ciaranello, Vandenberg, and Anders (1982) have reported that the majority of autistic children do not have demonstrable gross neuroanatomical structural abnormalities indicative of early CNS developmental stresses. They argue that the behavioral syndrome must, therefore, result from brain insult occurring in the later stages of neurodevelopment. These are the stages that are responsible for producing functional connections between neurons through the processes of dendritic and axonal elongation, synapse formation, and myelination. Abnormalities in gene function could alter the process of forming functional neuronal connections by affecting the delicate physical, temporal, or metabolic factors involved in one or more of these processes. The data we have presented from studies investigating the neuropathology of genetic conditions strongly associated with autism lend support to this hypothesis.

In his review of the two primary neuroanatomic theories of autism, Ornitz (1983) argues that most clinical and experimental data support a model of brainstem dysfunction as the basis for the constellation of autistic symptoms. This model implicates brainstem vestibular nuclei, and related nonspecific thalamic centers, in particular. Dysfunction in these areas is proposed to lead to abnormalities in processing and modulation of basic sensory input and motor output. This makes crucial information unavailable to processing by higher centers (particularly cortical) because of distortion of signals at lower levels. The other neuroanatomical model of autism supported by experimental and clinical data implicates dysfunction of higher cortical centers, particularly mesial frontal, temporal lobe, and neostriatal structures (Maurer and Damasio 1982).

Ornitz attempts to integrate these two hypotheses by noting that brainstem and cortical systems are mutually interdependent on transfer of sensory and motor information and regulatory modulation from one area to another. Autistic behavior is explained as a dysfunction in some aspect of this complex operational system responsible for multiple stages of sensory and motor processing.

Dysfunction of specific tracts resulting from abnormal or deficient myelination is an appropriate candidate to consider as one neuropathological mechanism that could lead to the autistic behavioral syndrome. In addition to PKU, CDLS, and tuberous sclerosis, abnormalities in brain myelination have been detected in patients with other genetic conditions associated with autism such as neurofibromatosis (Crome 1962), X-linked mental retardation (Dunn et al. 1962), and, commonly, in the CNS of patients with congenital rubella (Lemire et al. 1975), a disorder in which up to 12 percent of affected children manifest the autistic syndrome (Chess 1971; Desmond et al. 1970). Deficits or abnormalities in myelination in particular brain pathways during development could produce the disconnection within or between functional neuroanatomical processing levels that Ornitz describes.

Although of profound functional significance, subtle, qualitative abnormalities in myelin composition may be difficult to detect with conventional investigative techniques. The process of forming myelin is a complex, biochemical, and neurocellular task involving multiple interdependent stages that are completed in a precise, stepwise fashion (Lemire et al. 1975). The fact that temporal, spatial, and biochemical factors are all crucial variables throughout this entire process implies that there are multiple points and dimensions of vulnerability to disruption. For example, we have described how axonal outgrowth may be altered in specific areas of the brain of patients with PKU. If this occurred, it would cause a disturbance in the temporal relationship between axonal elongation and wrapping of the formed myelin sheath. The result would be inadequate myelination and corresponding functional deficits in neuronal conduction in those specific brain tracts.

Brain myelination occurs at a particularly rapid rate from the last trimester of gestation through the first 2–3 years of life. A hypothesis of dysfunctional myelination is therefore consistent with the suggestion that CNS damage in autism occurs in the latter stages of brain development. The period of extensive brain myelination also corresponds with the clinical observation that autistic behavior is usually first noted before 30 months of age. The critical stage of neurodevelopment we have referred to previously, then, may actually be one or more time frames during which myelination of specific pathways involved in sensory and motor modulation is occurring. For example, myelination of vestibular tracts is begun at approximately month 5 of gestation and is 50 percent completed by 2 years of age (Lemire et al. 1975). Myelination of reticular formation pathways proceeds from birth to at least 30 months of age (Lemire et al. 1975).

If disruption in myelination of specific tracts of the brain was responsible for the CNS dysfunction occurring in autistic children, variability in timing, extent, and location of disruption could potentially explain the inconsistent neurophysiological data obtained in studies of autistic patients to date. Disruption could take place within different levels and locations within the same *functional system* and outwardly still produce the same behavioral phenotype. For example, lesions in tracts within the brainstem would be likely to produce abnormal brainstem evoked responses (BSER). Although it may disrupt the same functional system and produce an identical behavioral phenotype, lesions in pathways above brainstem structures would not be expected to cause abnormalities on BSER.

It may be that myelination is only one of many potentially vulnerable processes during neurodevelopment that are etiologically relevant to autism. The findings of abnormal neuronal architecture and polarity, deficient axonal and dendritic growth, and disordered synaptogenesis in the genetic conditions previously described also reflect damage in the later stages of brain development. The means by which damage is produced in autism may not be nearly as important as the *timing* and the *location* within the intricate network of functioning systems that the human brain comprises.

The diversity of CNS abnormalities seen in the genetic conditions we have reviewed makes a thorough discussion of neuroanatomical location impossible at this time. From our review, a location which was commonly affected in the genetic disorders we have discussed at greater length is the periventricular area, particularly regions near the thalamus, subependymal lining, and basal ganglia. This area is of particular interest since limbic system structures are located in this region.

An abnormality in CNS serotonergic systems has been mentioned as a possible factor in the pathophysiology of autism (Young et al. 1982). One third of autistic patients have consistently been found to have elevated serotonin levels (Young et al. 1982). In contrast, decreased levels are more frequently seen in several genetic conditions we have reported to be associated with autism such as PKU, trisomy 21, histidinemia (Young et al. 1982), and CDLS (Greenberg and Coleman 1973).

Clinical Implications. The association of autism with genetic disorders clearly points out the need for comprehensive medical and genetic evaluation of all autistic children, including cytogenetic assessment. There are many potential benefits for detecting a genetic condition in autistic patients. Many children with genetic disorders associated with autism can have serious and potentially life-threatening medical problems as well as congenital anomalies that are secondary to their genetic condition. Examples are congenital heart disease in Williams Syndrome, epilepsy in tuberous sclerosis and CDLS, and metabolic defects in PKU. Once diagnosed, these problems are often responsive to specialized treatment. This can maximize benefits derived from nonmedical, academic, or psychological interventions as well as prevent medical and psychological deterioration from occurring. Like dietary management in PKU, actual treatments for many genetic conditions may soon be developed. The administration of supplemental folic acid to patients with the fragile X syndrome (Hagerman, McBogg, and Hagerman 1983), for example, may aid in the treatment of psychological symptoms associated with this disorder. Genetic counseling issues are also of utmost importance to the extended family with respect to disease prevention. This is especially pertinent with disorders in which prenatal diagnosis is possible such as the fragile X syndrome.

Conclusions

Our review has provided support for the hypothesis that the autistic syndrome is produced by damage to a developing, functional, neuroanatomical system in the brain. Since many different pathological processes can potentially produce this damage, it is not unexpected that within the entire autistic population, one will find a wide range of associated behavioral symptoms, neurological signs, psychophysiological abnormalities, and neuroanatomical lesions. Mental retardation, neurologic signs, and CT scan abnormalities are all frequently (although not always) seen in autistic patients with or without organic conditions. This suggests that CNS damage often occurs in more than one functional system.

Before concluding, we must emphasize that the neuropathological findings we have described in this article are from children with genetic conditions who have not had comprehensive psychiatric evaluations. Studies investigating behavioral, neuropathological, biochemical, and neurophysiological findings from children with genetic syndromes associated with autism are obviously indicated to obtain more precise information. These data would be particularly beneficial if obtained from patients both with and without autistic behavior to delineate neurobiological differences between these two groups. Magnetic resonance (MR) brain imaging, a more accurate, noninvasive method of identifying subtle white matter lesions, may also help confirm the presence and nature of the CNS dysfunction in patients with the autistic syndrome.

References

Abrams, N., and Pergament, E. Childhood psychosis combined with the XYY abnormality. *Journal of Genetic Psychology*, 118:13–16, 1971.

Agrawal, H.C., and Davison, A.N. Myelination and amino acid imbalance in the developing brain. In: Himwich, W.A., ed. *Biochemistry of the Developing Brain*. Vol. 1. New York: Marcel Dekker, Inc., 1973. pp. 143–186.

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: The Association, 1980.

August, G.J. A genetic marker associated with infantile autism. *American Journal of Psychiatry*, 140:6, 1983.

August, G.J.; Stewart, M.A.; and Tsai, L. The incidence of cognitive disabilities in the siblings of autistic children. *British Journal of Psychiatry*, 138:416–422, 1981.

Barlow, P. The influence of inactive chromosomes on human development. *Human Genetics*, 17:105–136, 1973.

Barr, A.N.; Grabow, J.D.; Matthews, C.G.; Grosse, F.R.; Motl, M.L.; and Opitz, J.M. Neurologic and psychometric findings in the Brachman-de Lange syndrome. *Neuropaediatrie*, 3:47–66, 1971.

Bender, B.L., and Yunis, E.J. Central nervous system pathology of tuberous sclerosis in children. *Ultra-*

structural Pathology, 1:287-299, 1980.

Berg, J.M.; McCreary, B.D.; Ridler, M.A.; and Smith G.F. *The de Lange Syndrome*. New York: Pergamon Press, 1970.

Brown, W.T.; Jenkens, E.C.; Friedman, E.; Brooks, J.; Wisniewski, K.; Ragathu, S.; and French, J. Autism is associated with the fragile X syndrome. *Journal of Autism and Developmental Disorders*, 12:303-308, 1982.

Campbell, M.; Rosenbloom, S.; Perry, R.; George, A.E.; Kricheff, I.I.; Anderson, L.; Small, A.M.; and Jennings, S.J. Computerized axial tomography in young autistic children. *American Journal of Psychiatry*, 139:510-512, 1982.

Campbell, M.; Wolman, S.R.; and Brewer, H. Klinefelter's syndrome in a three year old severely disturbed child. *Journal of Autism and Childhood Schizophrenia*, 2:34-48, 1972.

Chess, S. Autism in children with congenital rubella. *Journal of Autism and Childhood Schizophrenia*, 1:33-47, 1971.

Ciaranello, R.D.; Vandenberg, S.R.; and Anders, T. Intrinsic and extrinsic determinants of neuronal development: Relation to infantile autism. *Journal of Autism and Developmental Disorders*, 12:115-145, 1982.

Cohn, M.M. Syndromes with cleft lip and cleft palate. *Cleft Palate Journal*, 15:306-328, 1978.

Coleman, M. Introduction. In: Coleman, M., ed. *The Autistic Syndromes*. Amsterdam: North-Holland Publishing Co., 1976. pp. 1-10.

Cordero, M.E.; Trejo, M.; Colombo, M.; and Aranda, V. Histological maturation of the neocortex in phenylketonuric rats. *Early Human Development*, 8:157-173, 1983.

Crandall, B.F.; Carrel, R.E.; and Sparkes, R.S. Chromosome findings in 700 children referred to a psychiatric clinic. *Journal of Pediatrics*, 80:62-68, 1972.

Creak, E.M. Childhood psychosis. *British Journal of Psychiatry*, 109:84-89, 1963.

Crome, L. A case of central neurofibromatosis. *Archives of Disease in Childhood*, 37:640-651, 1962.

Damasio, H.; Maurer, R.G.; Damasio, A.R.; and Chui, H.C. Computerized tomographic scan findings in patients with autistic behavior. *Archives of Neurology*, 37:504-510, 1980.

DeLong, G.R.; Bean, C.; and Brown, F.R. Acquired reversible autistic syndrome in acute encephalopathic illness in children. *Archives of Neurology*, 38:191-194, 1981.

Desmond, M.M.; Wilson, G.S.; Verniaud, W.M.; Melnick, J.L.; and Rawls, W.E. The early growth and development of infants with congenital rubella. *Advances in Teratology*, 4:39-63, 1970.

Dick, A., and Ziegler, D. Tuberous sclerosis: A report of two cases. *Journal of the Kansas Medical Society*, 68:102-105, 1967.

Dunn, H.G.; Renpenning, H.; Gerrard, J.W.; Miller, J.R.; Tabata, T.; and Federoff, S. Mental retardation as a sex-linked defect. *American Journal of Mental Deficiency*, 67:827-848, 1962.

Eisenberg, L. The classification of childhood psychoses reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2:338-342, 1972.

Emery, A.E., and Rimoin, D.L. *Principles and Practice of Medical Genetics*. New York: Churchill Livingston, 1983.

Falek, A.; Schmidt, R.; and Jervis, G.A. Familial de Lange syndrome with chromosomal abnormalities. *Pediatrics*, 7:92-101, 1966.

Firestone, P., and Peters, S. Minor physical anomalies and behavior in children: A review. *Journal of Autism and Developmental Disorders*, 13:411-425, 1983.

Folstein, S., and Rutter, M. Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, 18:297-321, 1977.

Friedman, E. The "autistic syndrome" and phenylketonuria. *Schizophrenia*, 1:249-261, 1969.

Fryns, J.P.; Jacobs, J.; Kleczkowsha, A.; and Van den Berghe, H. The psychological profile of the fragile X syndrome. *Clinical Genetics*, 25:131-134, 1984.

Garreau, B.; Barthelemy, C.; and Sauvage, D. Comparison of autistic syndromes with and without associated neurological problems. *Journal of Autism and Developmental Disorders*, 14:105-111, 1984.

Garrick, R.; Gomez, M.R.; and Houser, O.W. Demyelination of the brain in tuberous sclerosis: Computed tomography evidence. *Mayo Clinic Proceedings*, 54:685-689, 1979.

Gilbert, E.F., and Opitz, J.M. Developmental and other pathologic changes in syndromes due to chromosome abnormalities. In: Rosenberg, H.S., and Bernstein, J., eds. *Perspectives in Pediatric Pathology*. Vol. 7. New York: Masson Publishing, USA, Inc., 1982. pp. 1-64.

Gillberg, C. Identical triplets with infantile autism and the fragile X syndrome. *British Journal of Psychiatry*, 143:256-260, 1983.

Gillberg, C., and Forsell, C. Childhood psychosis and neurofibromatosis—More than a coincidence.

Journal of Autism and Developmental Disorders, 14:1–9, 1984.

Gillberg, C., and Svendsen, P. Childhood psychosis and computed tomographic brain scan findings. *Journal of Autism and Developmental Disorders,* 13:19–32, 1983.

Gorski, R.A. Sexual differentiation of the brain. *Hospital Practice,* 13:55–62, 1978.

Greenberg, A., and Coleman, M. Depressed whole blood serotonin levels associated with behavioral abnormalities in the de Lange syndrome. *Pediatrics,* 51:720–724, 1973.

Gubbay, S.S.; Lobascher, M.; and Kingerlee, P. A neurological appraisal of autistic children: Results of a Western Australian survey. *Developmental Medicine and Child Neurology,* 12:422–429, 1970.

Hackney, I.M.; Hanley, W.B.; Davidson, W.; and Lindsao, L. Phenylketonuria: Mental development, behavior and termination of the low phenylalanine diet. *Journal of Pediatrics,* 72:646–655, 1968.

Hagerman, R.J.; McBogg, P.; and Hagerman, P.J. The fragile X syndrome: History, diagnosis and treatment. *Developmental and Behavioral Pediatrics,* 4:122–130, 1983.

Hansen, A.; Brask, B.H.; Nielson, J.; Rasmussen, K.; and Silesen, I. A case report of an autistic girl with an extra bisatellited marker chromosome. *Journal of Autism and Childhood Schizophrenia,* 7:263–267, 1977.

Hanson, D.R., and Gottesman, I.I. The genetics, if any, of infantile autism and childhood schizophrenia. *Journal of Autism and Childhood Schizophrenia,* 6:209–234, 1976.

Hart, Z.W.; Jaslow, R.L.; and Gomez, M.R. The de Lange syndrome. *American Journal of Diseases of Children,* 109:325–332, 1965.

Herbst, D., and Miller, J. Nonspecific X-linked mental retardation: II. The frequency in British Columbia. *American Journal of Medical Genetics,* 7:461–469, 1980.

Hoshino, Y.; Yashima, Y.; Tachibana, R.; Koneko, M.; Watanabe, M.; and Kumashiro, H. Sex chromosome abnormalities in autistic children: Long Y chromosome. *Fukushima Journal of Medical Science,* 26:31–42, 1979.

Huether, G.; Kaus, R.; and Neuhoff, V. Brain development in experimental hyperphenylalaninemia: Myelination. *Neuropediatrics,* 13:177–182, 1982.

Jackson, L.G., and Schimke, R.N. *Clinical Genetics: A Source Book for Physicians.* New York: John Wiley & Sons, Inc., 1979.

Johnson, H.G.; Ekman, P.; Friesen, W.; Nyhan, W.L.; and Shear, C. A behavioral phenotype in the de Lange syndrome. *Pediatric Research,* 10:843–850, 1976.

Jones, K.L., and Smith, D.W. The Williams elfin facies syndrome. *Journal of Pediatrics,* 86:718–723, 1975.

Judd, L.L., and Mandell, A.J. Chromosome studies in early infantile autism. *Archives of General Psychiatry,* 18:450–457, 1968.

Keith, C.G.; Dobbs, R.H.; Shaw, D.G.; and Cottrall, K. Abnormal facies, myopia and short stature. *Archives of Disease in Childhood,* 47:787–793, 1972.

Knobloch, H., and Passamanick, B. Some etiologic and prognostic factors in early infantile autism and psychosis. *Pediatrics,* 55:182–191, 1975.

Kolata, G.B. Sex hormones and brain development. *Science,* 205:985–986, 1979.

Kotsopoulous, S., and Kutty, K.M. Histidinemia and infantile autism. *Journal of Autism and Developmental Disorders,* 9:55–60, 1979.

Lemire, J.L.; Loeser, J.D.; Leech, R.W.; and Alvord, E.C. *Normal and Abnormal Development of the Human Nervous System.* Hagerstown: Harper and Row, Inc., 1975.

Levitas, A.; Hagerman, R.J.; Braden, M.; Rimland, B.; McBogg, P.; and Matus, I. Autism and the fragile X syndrome. *Developmental and Behavioral Pediatrics,* 4:151–158, 1983.

Lotter, V. Factors related to outcome in autistic children. *Journal of Autism and Childhood Schizophrenia,* 4:263–277, 1974.

Lowe, T.L.; Tanaka, K.; Seashore, M.R.; Young, J.G.; and Cohen, D.J. Detection of phenylketonuria in autistic and psychotic children. *Journal of the American Medical Association,* 243:126–128, 1980.

Malamud, N. Neuropathology of phenylketonuria. *Journal of Neuropathology and Experimental Neurology,* 25:254–268, 1966.

Mallin, S.R., and Walker, F.A. Effects of the XYY karyotype in one of the two brothers with congenital adrenal hyperplasia. *Clinical Genetics,* 3:490–494, 1972.

Maltz, A.D. Down syndrome and early infantile autism: Diagnostic confusion. *Journal of Autism and Developmental Disorders,* 9:453–454, 1979.

Mansheim, P. Tuberous sclerosis and autistic behavior. *Journal of Clinical Psychiatry,* 40:97–98, 1979.

Maurer, R.G., and Damasio, A.R. Childhood autism from the point of view of behavioral neurology. *Journal of Autism and Developmental Disorders,* 12:195–205, 1982.

McKay, R.D.; Bobrow, G.M.; and Cooke, J.H. The identification of a repeated DNA sequence involved in the human karyotype polymorphism of the Y chromosome. *Cytogenetic and Cellular Genetics,* 21:19–32, 1978.

Melnick, C.R.; Michals, K.K.; and Matalon, R. Linguistic development of children with phenylketonuria and normal intelligence. *Journal of Pediatrics,* 98:269–272, 1981.

Menkes, J.H. *Textbook of Child Neurology.* 2nd ed. Philadelphia: Lea and Febiger, 1980.

Meryash, D.L.; Szymanski, L.S.; and Park, S.G. Infantile autism associated with the fragile X syndrome. *Journal of Autism and Developmental Disorders,* 12:295–301, 1982.

Minton, J.; Campbell, M.; Green, W.H.; Jennings, S.; and Samit, C. Cognitive assessment of siblings of autistic children. *Journal of the American Academy of Child Psychiatry,* 21:256–261, 1982.

Nelson, J.; Christensen, K.R.; and Friedrich, V. Childhood of males with the XYY syndrome. *Journal of Autism and Childhood Schizophrenia,* 3:5–26, 1973.

Nielson, K.B. Diagnosis of the fragile X syndrome (Martin-Bell syndrome): Clinical findings in 27 males with fragile site at Xq28. *Journal of Mental Deficiency Research,* 27:211–266, 1983.

Nyhan, W.L. Behavioral phenotypes in organic genetic disease. *Pediatric Research,* 6:1–9, 1972.

O'Gorman, G. *The Nature of Childhood Autism.* 2nd ed. London, Butterworth and Co., 1970.

Ornitz, E.M. The functional neuroanatomy of infantile autism. *International Journal of Neuroscience,* 19:85–124, 1983.

Ornitz, E.M.; Guthrie, D.; and Farley, A.H. The early development of autistic children. *Journal of Autism and Childhood Schizophrenia,* 7:207–229, 1977.

Ornitz, E.M., and Ritvo, E.R. The syndrome of autism: A critical review. *American Journal of Psychiatry,* 133:609–621, 1976.

Paul, R.; Cohen, D.J.; and Volkmar, F.R. Autistic behaviors in a boy with Noonan syndrome. *Journal of Autism and Developmental Disorders,* 13:433–434, 1983.

Prensky, A.L.; Carr, S.; and Moser, H.W. Development of myelin in inherited disorders of amino acid metabolism. *Archives of Neurology,* 19:552–558, 1968.

Ptacek, L.J.; Opitz, J.M.; Smith, D.W.; Gerritsen, T.; and Waisman, H.A. The Cornelia de Lange syndrome. *Journal of Pediatrics,* 63:1000–1020, 1963.

Reiss, A.L.; Feinstein, C.; Rosenbaum, K.N.; Borengasser-Caruso, M.A.; and Goldsmith, B. Autism associated with the Williams syndrome. *Journal of Pediatrics,* 106:247–249, 1985.

Riikonen, R., and Amnell, G. Psychiatric disorders in children with earlier infantile spasms. *Developmental Medicine and Child Neurology,* 23:747–760, 1981.

Rimland, B. The differentiation of childhood psychoses: An analysis of checklists for 2,218 psychotic children. *Journal of Autism and Childhood Schizophrenia,* 1:161–174, 1971.

Rosenbloom, S.; Campbell, M.; George, A.E.; Kricheff, I.I.; Taleparos, E.; Anderson, L.; Reuben, R.N.; and Korein, J. High resolution CT scanning in infantile autism: A quantitative approach. *Journal of the American Academy of Child Psychiatry,* 23:72–77, 1984.

Rutter, M., and Bartak, L. Causes of infantile autism: Some considerations from recent research. *Journal of Autism and Childhood Schizophrenia,* 1:20–32, 1971.

Rutter, M.; Bartak, L.; and Newman, S. Autism—A central disorder of cognition and language. In: Rutter, M., ed. *Infantile Autism: Concepts, Characteristics and Treatment.* London: Churchill-Livingston, 1971. pp. 148–173.

Schlesinger, B.; Clayton, B.; Bodian, M.; and Jones, K.V. Typus degenerativus amstelodamensis. *Archives of Diseases in Childhood,* 38:349–357, 1963.

Shah, S.N.; Peterson, N.A.; and Mckean, A.N. Lipid composition of human cerebral white matter and myelin in phenylketonuria. *Journal of Neurochemistry,* 19:2369–2376, 1972.

Siva San Kar, D.V. Chromosome breakage in infantile autism. *Developmental Medicine and Child Neurology,* 12:572–575, 1970.

Smith, D.W. *Recognizable Patterns of Human Malformation.* 3rd ed. Philadelphia; W.B. Saunders, 1982.

Spence, M.A. *Genetic Studies.* In: Ritvo, E.R., ed. *Autism: Diagnosis, Current Research and Management.* New York: Spectrum Publications, 1976. pp. 169–174.

Stewart, D.A.; Bailey, J.D.; Netley, C.T.; Rovet, J.; Park, E.; Cripps, M.; and Curtis, J.A. Growth and development of children with X and Y chromosome aneuploidy from infancy to pubertal age: The Toronto study. In: Stewart, D.A., ed. *Children With Sex Chromosome*

Aneuploidy: Follow-up Studies. New York: Alan R. Liss, Inc., 1982. pp. 99-154.

Stewart, D.A.; Netley, C.J.; and Park, E. Summary of clinical findings of children with 47XXY, 47XYY and 47XXX karyotypes. In: Stewart, D.A., ed. *Children With Sex Chromosome Aneuploidy: Follow-up Studies.* New York: Alan R. Liss, Inc., 1982. pp. 1-5.

Taft, L., and Cohen, H. Hypsarrythmia and infantile autism: A clinical report. *Journal of Autism and Childhood Schizophrenia,* 1:337-349, 1971.

Thibault, J.H., and Manueldis, E.E. Tuberous sclerosis in a premature infant: Report of a case and review of the literature. *Neurology,* 20:139-146, 1970.

Trombley, I.K., and Mirra, S.S. Ultrastructure of tuberous sclerosis: Cortical tuber and subependymal tumor. *Annals of Neurology,* 9:174-181, 1981.

Turner, B., and Jennings, A.N. Trisomy for chromosome 22. *Lancet,* I:49-50, 1961.

Turner, G.; Daniel, A.; and Frost, M. X-linked mental retardation, macro-orchidism and the Xq27 fragile site. *Journal of Pediatrics,* 96:837-841, 1980.

Turner, G., and Jacobs, P. Marker (X)-linked mental retardation. *Advances in Human Genetics,* 13:83-112, 1983.

Urich, H. Malformation of the nervous system, perinatal damage and related conditions in early life. In: Blackwood, W., and Corsellis, J.H., eds. *Greenfield's Neuropathology.* Chicago: Year Book Medical Publishers, Inc., 1976. pp. 361-469.

Wakabayashi, S. A case of infantile autism associated with Down syndrome. *Journal of Autism and Developmental Disorders,* 9:31-36, 1979.

Watson, M.S.; Leckman, J.F.; Annex, B.; Breg, W.R.; Boles, D.; Volkmar, F.R.; and Cohen, D.J. Fragile X in a survey of 75 autistic males. *New England Journal of Medicine,* 310:1462, 1984.

Williams, R.S.; Hauser, S.L.; Purpura, D.P.; DeLong, R.; and Swisher, C.N. Autism and mental retardation: Neuropathologic studies performed in four persons with autistic behavior. *Archives of Neurology,* 37:749-753, 1980.

Wilson, G.N.; Hieber, V.C.; and Schmickel, R.D. The association of chromosome 3 duplication and Cornelia de Lange syndrome. *Journal of Pediatrics,* 5:783-788, 1978.

Wolraich, M.; Bzostek, B.; Neu, R.L.; and Gardner, L.I. Lack of chromosome aberrations in autism. *New England Journal of Medicine,* 38:1231, 1970.

Young, J.G.; Kavanaugh, M.E.; Anderson, G.M.; Shaywitz, B.A.; and Cohen, D.J. Clinical neurochemistry of autism and associated disorders. *Journal of Autism and Developmental Disorders,* 12:147-165, 1982.

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Polyuria Among Patients With Psychosis

by W. Victor R. Vieweg, Joseph J. David, Joseph L. Glick, Wilma T. Rowe, Randell T. Curnow, Mark D. Lawrence, Joseph J. Yazel, and Wilford W. Spradlin

Abstract

Using the criterion of early morning urinary specific gravity (SPGR) of 1.008 or less to define the presence of polyuria, we identified 26 of 72 male (36 percent) and 14 of 31 female (45 percent) institutionalized chronically psychotic patients as polyuric during a comprehensive survey of one of the chronic care units at a State mental hospital. Factors including diagnosis, sex, age, weight, and serum sodium did not distinguish the polyuric from the nonpolyuric patients. For men, administration of lithium was associated with polyuria. Urinary creatinine concentration (UCR) correlated well with SPGR, and UCR may provide an alternate index to separate polyuric from nonpolyuric patients. The clinical implications of our findings are discussed.

Hoskins and Sleeper (1933) were the first investigators to evaluate chronically psychotic patients for polyuria. Using frequent catheterizations, they found that male patients with schizophrenic disorders excreted about twice as much urine per day (2,602 ml) as normal male controls (1,328 ml). In this study, 55 percent of their patients excreted more than 2,000 ml of urine daily in contrast to only 19 percent of controls who exceeded this amount (Sleeper and Jellinek 1936). In 1979, Jos and Perez-Cruet reported a 6.6 percent prevalence of "compulsive water drinking" among State mental hospital patients, with one half of them experiencing symptoms of "water intoxication." More recently, Blum and Friedland (1983) found that 17.5 percent of male psychiatric patients in a large Veterans Administration hospital had a urinary specific gravity of 1.008 or less, and this criterion was used to establish a diagnosis of "chronic psychogenic polydipsia."

More recently, Lawson, Karson, and Bigelow (1985) attempted to replicate, in the drug era, the work of Hoskins and Sleeper (1933). The mean daily urine volume for patients with schizophrenic disorders was reported as 2,319 ml compared with 1,054 ml for nonschizophrenic patients and 1,265 ml for a normal population. If the daily urine volume was more than 600 ml or if the nursing staff judged the volumes to be complete, the data were used in their report. This article seems methodologically flawed because of the very low measurement of daily urinary creatinine of 857 ± SD 375 mg for 28 male and 7 female patients with schizophrenic disorders. The point to be made is that even careful investigators may underestimate daily urine volume among chronically psychotic patients despite the use of currently acceptable techniques to measure daily urine volume.

Our recent interest in patients with psychosis, intermittent hyponatremia, and polydipsia (PIP syndrome) (Vieweg et al. 1984*a*, 1984*b*, 1985*a*, 1985*b*, 1985*c*) has stimulated us to estimate the prevalence of polyuria among patients with chronic psychosis, most of whom have a schizophrenic disorder. Using the criterion of early morning urinary specific gravity (SPGR) of 1.008 or less to define polyuria, we found that 36 percent of male and 45 percent of female institutionalized chronically psychotic patients were polyuric.

Methods

Seventy-two men (mean age 37.6 ± SD 11.7 years) and 31 women (mean age 47.0 ± SD 10.3 years) residing

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in one of the chronic care units at Western State Hospital made up the study population. Psychosis was present in all patients, and *DSM-III* criteria were used to establish specific diagnoses (American Psychiatric Association 1980).

Determinations of age, sex, weight, diagnosis, drugs received, serum sodium, urinary creatinine concentration, and specific gravity were made for each patient. Weight, blood, and urine samples were obtained together in the early morning. Polyuria was considered present if the early morning urinary specific gravity was 1.008 or less.

Differences between group means were examined using unpaired t statistics. Correlations among age, weight, serum sodium, urinary creatinine concentration, and specific gravity determinations were sought using the Pearson product-moment correlation coefficient. The frequencies of sex, polyuria, diagnoses, and drugs used among the various groups were compared with χ^2 tests.

There were 15 unpaired t statistics and 7 χ^2 analyses performed on the same data base. We used the Bonferroni correction technique, which allows for changing the significance level by dividing the conventional level (.05) by the number of statistical operations used: the reader is invited to use a significance level of 0.003 for unpaired t statistics and 0.007 for χ^2 analysis.

Results

We performed χ^2 tests initially to see if sex alone might predispose the patient to polyuria and thus vitiate further analysis. Using a 2 × 2 contingency table involving men and women and those patients with specific gravity (SPGR) $\leq$ 1.008 and SPGR $>$ 1.008 (table 1), we found a χ^2 of 0.41 ($p = 0.527$). These observations were further substantiated when we found the mean values for SPGR among men (1.0116 ± SD .0067) and women (1.0102 ± SD .0057) to be similar ($t = 1.002$, $p = .320$).

Male patients were younger than female patients ($t = 3.871$, $p = .000$) (table 1). Both serum sodium ($t = .845$, $p > .317$) and urinary creatinine concentrations ($t = 1.025$, $p = .309$) were similar among the men and women. Differences in weight between men and women did not quite approach statistical significance ($t = 2.117$, $p = .035$). Diagnoses were similarly distributed among men ($\chi^2 = 3.13$, $p = .208$) and women ($\chi^2 = 9.53$, $p = .049$) for patients with and without polyuria (table 1).

Among the men, the distribution of drugs was different ($\chi^2 = 11.39$, $p = .004$) for patients with and without polyuria. This difference disappeared ($\chi^2 = .89$, $p = .349$) when lithium was removed from the contingency table. For the women, there was no difference ($\chi^2 = 2.01$, $p = .367$) between those patients with and without polyuria. Comparing men and women, we found little difference ($\chi^2 = 5.88$, $p = .0519$) in the distribution of drugs. Since all patients were receiving antipsychotic drugs, this factor was not included in the above calculations.

Thirty-six percent of men and 45 percent of women were polyuric (early morning SPGR $\leq$ 1.008). For men, SPGR ($t = 9.55$, $p = .000$) and urinary creatinine concentration (UCR) ($t = 6.955$, $p = .000$) differed between polyuric and nonpolyuric patients. This difference could not be accounted for by age ($t = 1.431$, $p = .153$), weight ($t = 1.105$, $p = .272$), or serum sodium ($t = .839$, $p > .317$) determinations. Also, for women, SPGR ($t = 7.243$, $p = .000$) and UCR ($t = 6.669$, $p = .000$) differed between polyuric and nonpolyuric patients. This difference could not be accounted for by age ($t = .429$, $p > .500$), weight ($t = 1.828$, $p = .075$), or serum sodium ($t = 1.494$, $p > .500$) determinations. Correlations among age, weight, serum sodium, UCR, and SPGR were sought among the polyuric and nonpolyuric men and women (table 1). The only consistent correlation was the relationship between UCR and SPGR (r between .754 and .921, $p < .001$).

Discussion

Hyposthenuria is a semiquantitative index of polyuria, and early morning urinary specific gravities of 1.008 or less have been used to establish the presence of polyuria (Blum and Friedland 1983). Using this index of polyuria, we identified 26 of 72 male (36 percent) and 14 of 31 female (45 percent) institutionalized chronically psychotic patients as polyuric during a comprehensive survey of one of the chronic care units at a State mental hospital. Our prevalence is distinctly higher than that reported either by Jos and Perez-Cruet (1979) (6.6 percent) or Blum and Friedland (1983) (17.5 percent). Our observations approach Sleeper and Jellinek's (1936) finding that 55 percent of chronically psychotic patients are polyuric.

Factors including diagnosis, sex, age, weight, and serum sodium did not distinguish the polyuric from the nonpolyuric patients. For men, administration of lithium was associated with polyuria. This finding is consistent with the well-known fact that lithium may induce nephrogenic diabetes insipidus in up

Table 1. Values for sex, age, weight (kg), schizophrenic disorder (SZ), schizoaffective disorder (SA), organic mental disorder (OB), major depressive disorder (DP), bipolar disorder (BP), serum sodium (SOD) (mEq/l), urinary creatinine concentration (UCR) (mg/dl), urinary specific gravity (SPGR), antipsychotic drugs (AP), lithium (LI), carbamazepine (CARB), and phenytoin (PHY) for 103 institutionalized, chronically psychotic men and women evaluated for polyuria

Polyuria index	Subjects	Sex	Age	Weight	SZ	SA	OB	DP	BP	SOD	UCR	SPGR	AP	LI	CARB	PHY
A. SPGR >1.008	46	M	39.1 ± 12.7	77.2 ± 14.8	27	3	16	0	0	138.9 ± 3.6	108.3 ± 56.0	1.0154 ± 0.0054	46	8	11	15
B. SPGR ≤1.008	26	M	35.0 ± 9.4	73.5 ± 10.9	20	2	4	0	0	139.6 ± 3.1	30.2 ± 15.2	1.0049 ± 0.0021	26	14	5	2
C. All men	72	M	37.6 ± 11.7	75.8 ± 13.6	47	5	20	0	0	139.1 ± 3.4	80.1 ± 59.1	1.0116 ± 0.0067	72	22	16	17
D. SPGR >1.008	17	F	47.8 ± 12.4	73.4 ± 12.1	9	3	3	2	0	140.7 ± 3.4	98.1 ± 35.8	1.0143 ± 0.0041	17	3	1	1
E. SPGR ≤1.008	14	F	46.1 ± 7.3 ±	65.4 ± 12.2	13	0	0	0	1	138.6 ± 4.3	31.6 ± 11.0	1.0053 ± 0.0024	14	7	0	1
F. All women	31	F	47.0 ± 10.3	69.8 ± 12.6	22	3	3	2	1	139.8 ± 3.9	68.0 ± 43.2	1.0102 ± 0.0057	31	10	1	2

Correlation matrixes

A.	Weight	SOD	UCR	SPGR
Age	0.013	−0.199	−0.274	−0.020
Weight		0.008	0.122	0.162
SOD			0.048	−0.004
UCR				0.800**

B.	Weight	SOD	UCR	SPGR
Age	−0.209	0.122	0.105	0.185
Weight		−0.072	0.183	0.102
SOD			0.239	0.077
UCR				0.754**

C.	Weight	SOD	UCR	SPGR
Age	−0.015	−0.131	−0.063	0.132
Weight		−0.026	0.177	0.197
SOD			−0.014	−0.070
UCR				0.884**

D.	Weight	SOD	UCR	SPGR
Age	−0.340	0.183	−0.161	−0.032
Weight		0.091	0.007	−0.179
SOD			−0.168	−0.204
UCR				0.806**

E.	Weight	SOD	UCR	SPGR
Age	0.095	−0.003	−0.404	−0.534*
Weight		−0.277	−0.070	−0.089
SOD			0.591*	0.615*
UCR				0.852**

F.	Weight	SOD	UCR	SPGR
Age	−0.156	0.123	0.056	−0.021
Weight		−0.001	0.245	0.176
SOD			0.215	0.272
UCR				0.921**

Where mean values appear ± 1 standard deviation is recorded. For correlation matrixes * = $p < 0.05$ and ** = $p < .001$.

to 20 percent of patients receiving this drug. Interestingly, lithium was not predictive in identifying polyuric patients among women.

Expectedly, the mean SPGR was lower among polyuric compared with nonpolyuric patients of both sexes. There was an excellent correlation between SPGR and UCR in this study, and one might choose UCR as an alternate index to separate polyuric from nonpolyuric patients. In Hoskins and Sleeper's study (1933) of polyuria among 57 male patients with schizophrenic disorders, the mean UCR was 49.55 mg/dl (actually creatinine nitrogen was reported, but this measurement can be converted to creatinine by multiplying creatinine nitrogen by 2.69 and then UCR obtained by dividing creatinine by urinary volume). This mean value of UCR of 49.55 mg/dl was associated with a mean daily urinary volume of 2,496 ± SD 1,707 ml. Using a method previously described by us (Vieweg et al. 1985*a*, 1985*b*), we found the mean daily urinary volume of our 26 male polyuric patients to be 3,935 ± SD 4,361 ml, compared to 2,987 ± SD 1,410 ml in our 14 female polyuric patients. Thus, our polyuric patients excreted larger volumes than those reported by Hoskins and Sleeper (1933). The difference may reflect the fact that Hoskins and Sleeper did not divide their population into polyuric and nonpolyuric patients. Jos and Perez-Cruet (1979) and Blum and Friedland (1983) did not report daily urinary volumes. The recent report by Lawson, Karson, and Bigelow (1985) appears to have underestimated daily urine volumes among psychotic patients.

The clinical implications of our findings remain unclear. Few of our patients manifested hyponatremia during this survey (although one patient had a serum sodium determination of 126 mEq/l). We did not perform a chart review, as did Jos and Perez-Cruet (1979), looking for any episodes of symptomatic hyponatremia during the course of the patients' hospitalization. However, on the basis of our own clinical experience and the experience recorded in the literature, a large number of institutionalized chronically psychotic patients appear to be at risk to suffer the complications of "water intoxication." We have suggested a simple method to identify those patients who may be at greatest risk. Further studies are indicated.

It appears to us that the disturbances in the limbic system associated with psychosis through anatomical and biochemical pathways bring about changes in the supraoptic and paraventricular nuclei of the hypothalamus where thirst and osmotic regulation take place. Presumably, in the absence of hyponatremia, our polydipsic patients have altered thirst regulation. When hyponatremia appears, there is likely superimposed osmotic dysregulation. These mechanisms have been reviewed by us in greater detail recently (Vieweg et al. 1985*a*).

References

American Psychiatric Association. *DSM-III: Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed. Washington, DC: The Association, 1980.

Blum, A., and Friedland, G.W. Urinary tract abnormalities due to chronic psychogenic polydipsia. *American Journal of Psychiatry,* 140:915–916, 1983.

Hoskins, R.G., and Sleeper, F.H. Organic functions in schizophrenia. *Archives of Neurology and Psychiatry,* 30:123–140, 1933.

Jos, C.J., and Perez-Cruet, J. Incidence and morbidity of self-induced water intoxication in State mental hospital patients. *American Journal of Psychiatry,* 136:221–222, 1979.

Lawson, W.B.; Karson, C.N.; and Bigelow, L.B. Increased urine volume in chronic schizophrenic patients. *Psychiatry Research,* 14:323–331, 1985.

Sleeper, F.H., and Jellinek, E.M. A comparative, physiologic, and psychiatric study of polyuric and non-polyuric schizophrenic patients. *Journal of Nervous and Mental Disease,* 83:557–563, 1936.

Vieweg, W.V.R.; Rowe, W.T.; David, J.J.; Sutker, L.H.; and Spradlin, W.W. Evaluation of patients with self-induced water intoxication and schizophrenic disorders (SIWIS). *Journal of Nervous and Mental Disease,* 172:552–555, 1984*b*.

Vieweg, W.V.R.; Rowe, W.; David, J.; and Spradlin, W. Hyposthenuria as a marker for self-induced water intoxication and schizophrenic disorders. *American Journal of Psychiatry,* 141:1258–1260, 1984*a*.

Vieweg, W.V.R.; David, J.J.; Rowe, W.T.; Wampler, G.J.; Burns, W.J.; and Spradlin, W.W. Death from self-induced water intoxication among patients with schizophrenic disorders. *Journal of Nervous and Mental Disease,* 173:161–165, 1985*b*.

Vieweg, W.V.R.; Rowe, W.T.; David, J.J.; Curnow, R.T.; and Spradlin, W.W. Patterns of urinary excretion among patients with self-induced water intoxication and psychosis. *Psychiatry Research,* 15:71–79, 1985*c*.

Vieweg, W.V.R.; David, J.J.; Rowe, W.T.; Peach, M.J.; Veldhuis, J.D.; Kaiser, D.L.; and Spradlin, W.W. Psychogenic polydipsia and water intoxication—Concepts that have failed. *Biological Psychiatry*, 20:1308–1320, 1985*a*.

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Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work, in cooperation with several community and mental health organizations, has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside."

For more information about the rental or purchase of these videotapes, please contact: **Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.**

First Person Account: Three Generations of Schizophrenia

by Lucy Fuchs

The article that follows is part of the *Schizophrenia Bulletin's* ongoing *First Person Accounts* series. We hope that mental health professionals—the *Bulletin's* primary audience—will take this opportunity to learn about the issues and difficulties confronted by consumers of mental health care. In addition, we hope that these accounts will give patients and families a better sense of not being alone in confronting the problems that can be anticipated by persons with serious emotional difficulties. We welcome other contributions from patients, ex-patients, or family members. Our major editorial requirement is that such contributions be clearly written and organized, and that a novel or unique aspect of schizophrenia be described, with special emphasis on points that will be important for professionals. Clinicians who see articulate patients, with experiences they believe should be shared, might encourage these patients to submit their articles to *First Person Accounts*, Schizophrenia Research Branch, NIMH, 5600 Fishers Lane, Rm. 10C-06, Rockville, MD 20857.—The Editors.

Abstract

The author gives an account of three generations of schizophrenia from the changing perspective of childhood through adulthood, as niece, sister, and mother. She describes the impact on the family in this 50-year span and discusses changing societal attitudes to mental illness as well as changes in treatment and professional thinking.

Ibsen treated it tragically in "Ghosts" and Gilbert and Sullivan humorously in "Ruddigore." I am referring to the theme of a family curse in the form of insanity passed on from one generation to another. I disliked "Ghosts" because of its morbid outlook and enjoyed "Ruddigore" because of its lighthearted foolishness. In neither case did I think that the idea of inherited mental illness was realistic. Years later, I recognize that during all that time my family was enacting its own tragedy affecting several generations. Although I never chose it, a small part was thrust upon me as a child and I was inexorably drawn into a larger role as the tragedy progressed. I am referring to schizophrenia.

Early in my life I became aware of the visits my father made to his brother. The trip was an exhausting 3-hour train and bus ride to a sad and mysterious place. So sad that my mother, whom I knew to be kind, was unwilling to go. She did, however, spend the previous day cooking the meal my father was to share with my uncle. Early in the morning on the day of the trip, I watched my mother and father pack additional delicacies, mended underwear, and miscellaneous items like magazines and drawing supplies chosen to entice my uncle into activity. The day after was given to a review. "What did the doctor say?" made sense to me. "How did Aaron behave?" was disquieting as it implied misbehavior and childishness in an adult. As I grew older, I observed my father become more exhausted from his trips and more and more disappointed in his brother's failure to improve. The saddest visits were when my father had to report that my uncle's behavior led to being moved to a "bad ward." At the end of the 1940s, my parents deliberated about a proposed lobotomy. They finally

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agreed, hoping for the best. The best never happened. My uncle died in Pilgrim State Hospital in his eighties. He had been there for 60 years.

I did not see my uncle until I was an adult, but his self-portrait hung in our living room. His handsome, brooding presence made me feel a loss, for though I had other uncles, no other was an artist or so young.

And what caused this sad illness? Without knowing the name of my uncle's disorder, we all seemed to have accepted psychoanalytic thinking. My grandmother, my uncle's mother, was a dignified but cold woman so that the belief that schizophrenia was due to maternal rejection grew on fertile ground.

My uncle's condition had a name and came on suddenly. More tragic was my brother's, which had no name and grew insidiously. A bright, handsome child, 5 years my senior, he was loved by my parents and was a delight to his teachers. He was not, however, interested in children. As he grew older, my parents became increasingly anxious about his preference for adults. I shared their anxiety and, 40 years later, I remember our enthusiasm when he spoke of two boys whom he might invite home. Although they never came, I remember their names still. So, in subtle ways, the natural order was reversed, and I was asked to understand and make allowances for my older brother.

Lacking his own friends, he bossed and teased me. Although he played with me rarely, he resented my play with other children and disrupted our games or was embarrassingly disagreeable when I brought new friends home. Hardest for me were his sudden changes of mood. I risked his anger when I did not follow his direction and my own self-respect when I bowed to pressure. These choices were all the more painful because I loved him. As he reached adolescence, he was also becoming more critical and angry toward my parents. The three of us became adept at avoiding confrontations. Things reached a climax when he was 16. He started attending a local college and began having trouble studying. Now I know one reason was the schizophrenic "loss of thought." My parents arranged for a psychiatrist to see him, but after one session he refused to return.

Despite these problems, my brother must have made heroic efforts. Not only was he graduated from college, but he even served in the army for a few years. He never saw active duty and was taken under the wing of his superior officers in an Intelligence Unit. They appreciated his dependability and strict and religious outlook. Despite warm and lengthy letters, he was again difficult to live with on his return. He completely disowned me because he disapproved of my engagement and did not speak to me until my mother's terminal illness 15 years later. During this time, he structured a life with which he could cope. He never married, became intensely religious, and spent much time and energy in helping our aging parents. He worked as an elementary school teacher in an inner city school and won some acclaim as a strict but fair teacher to children who needed rules and structure. He finally found friends within a strict religious community, and this was some comfort to my parents who had by then resigned themselves to his not marrying. They derived some satisfaction in knowing that he was involved in his work and at long last had friends. I am detailing my brother's long-range adjustment because I believe attention must be paid to other "mild schizophrenics" who are unhappy and cause pain to their loved ones but still manage a life outside treatment and without an official diagnosis. Eventually my brother diagnosed himself. His anger abated as his delusional thinking loosened its hold. He no longer sees me or my husband as evil people. He divulged his self-diagnosis when my daughter became psychotic.

My brother wanted to be helpful and told me that he noticed his own thinking became less difficult when he was taking medication for another illness. He shared with me how he talked back to his voice when it caused trouble. "If you can't say something wise, shut-up!" Before this revelation, I had not formed a definite opinion of his problem. I considered schizophrenia as a diagnosis when he was unreasonably accusatory and punishing toward me, but leaned toward obsessive-compulsive disorder when his accusations had some basis in reality. At any rate, in the light of his own experience, he encouraged my husband and me to continue our sympathetic support of our delusional daughter, and stressed the need to be hopeful and encouraging. So, family commitment has survived years of pain, madness, and confusion and the years to come hold some promise of affording simple family contacts and pleasures.

If diagnosis eluded me for so long, so did causality. Though sympathetic toward my parents, I was also influenced by the thinking of the time and in my heart I blamed them. Now I realize that living with a mentally disturbed son had made these naturally gentle and cautious people increasingly anxious. At the time, it was easy to credit neighbors' and relatives' comments that my brother's problems were caused by my parents' insecurity and their foolish capitulation to his angry outbursts. Today, some family therapists lean dangerously close to this attitude with their theory of the "identified patient" who acts out the pathology of the rest of the family. True in particular cases, perhaps, this theory unfeelingly ignores the inevitable changes that occur in families who live with a mentally ill member. At any rate, when I was young, I resolved that as a parent I would neither neglect one child nor indulge the other, and I was determined to live life with more joy. Good resolutions—but no proof against biological or genetic catastrophe.

Considering my early life, it is clear why I would be drawn to working with troubled people and why I would also look forward to having a happy, healthy family. Things went well, and my husband and I were confident about our children's futures in their early years. I knew, of course, that there was no perfect child and that there was a certain amount of pain and struggle in every life. For this reason, when my second child, Susan, turned out to be an unusually quiet baby and later a shy toddler, I valued these qualities as part of her unique nature. If people were less attentive to her than to her more outgoing older brother, I wondered at them and cherished her all the more. Looking back, I can see that very early I tried to fill the gap between her and the rest of the world by becoming more attentive, playful, and protective of her. So, slowly and quite naturally, I accustomed myself to her ways and to feeling responsible for her happiness. Despite her early reserve, by 7 or 8, she had become an energetic and interested child. She excelled in school, was an avid reader, and did well in many athletic activities. With all this, she still did not find making friends easy. She was shy, easily hurt, yet critical and argumentative. We thought she would eventually find a special friend or two, and we minimized her legalistic quarrels by thinking she had strong opinions and was independent. Jokingly, we said she was surely cut out to be a lawyer.

By high school Susan was more outgoing. She attracted potential friends because of her intelligence and her dynamic interest in ideas and activities. She was still easily hurt, however, and her relationships were uneven and stormy. It was a period of confusion and stress for me as well. Although I was the one she came to in tears for comfort, I was becoming aware that there was a lack of warmth and reciprocity. We no longer had the good times together that we had had earlier. There was no balance, and living with my daughter became emotionally draining. Still, I accepted the common wisdom that Susan was merely going through adolescence and that I was an overly concerned mother. When Susan reached age 15, we finally sought professional help because of the appearance of a new symptom as she began to have trouble completing written assignments. Once or twice she said she thought she was going crazy, because she couldn't think. That was outside the realm of my imagination, and I assured her that most adolescents have these fears. It was apparently outside the range of possibilities for the psychiatrist as he indicated Susan had some problems in self-esteem and suggested my distancing myself more. I followed this advice as well as scrupulously keeping out of the treatment process with him and the psychiatrist who saw her in college. In college, Susan continued the same up and down life: strong commitments, painful disappointment, and trouble with written assignments. Looking back, I think that whatever good both psychiatrists did came not from their insight-oriented therapy, but as a result of their relationship and support. Susan dropped out of college in her third year and not until her first psychotic breakdown 2 years later did we realize she was schizophrenic. In looking back, I think I can see many earlier clues. Now, I question the ease with which pronouncements about "normal adolescence" are made. I also question the theory that early identification of schizophrenia is bad and necessarily leads to self-fulfilling prophecies. What about the wrong diagnosis of neuroses? In what other field is ignorance considered an advantage? A case could just as easily be made that early identification of children-at-risk could lead to early and more appropriate help. I particularly regret that I followed professional advice and distanced myself in her adolescence. Schizophrenics already feel too distant.

There are many other dynamics to explore. For example, was the bossy and argumentative nature of my brother and daughter a marker? When combined with shyness, could it be understood as first an attempt to get others to share their atypical perceptions, followed by hurt and angry withdrawal when they do not succeed?

Susan became catatonic at age 23, while living a nomadic life. She was hospitalized following the intervention of a policeman but released against the advice of the hospital and as a result of being informed of her rights by a patient advocate. We breathed a sigh of relief for the 3 days she was in the hospital, where we thought she would get help. We were informed of the diagnosis of schizophrenia at the same time she was released and were naturally shocked at the precipitate release.

Susan wandered around various cities for another year and a half, living on the street and in shelters or crashing with friends. She was in touch every few weeks, sometimes phoning with fantastic accusations and threats. When she periodically came home, we tried to be caring and helpful and suggested she seek psychiatric help, but were afraid to push too hard. The one occasion on which we took a strong stand led to a violent outburst and flight. Her second catatonic episode mercifully came during a visit home so we were prepared. This time we arranged for her commitment to a hospital nearby and had already chosen a psychiatrist. Medication got her moving again and helped her delusional thinking. She was more warm and outgoing than I had seen her in years. Susan went to a halfway house and enjoyed the group therapy and the contacts with the staff. We began to hope again. However, although she put up with the side effects of pacing for a month, she finally stopped her medication abruptly and lost all her gains. She took up a vagabond life again. Still we hoped we were ahead and that she would voluntarily seek help because of her positive experiences. We had not yet understood the awful grip of disordered thinking. Instead, we were on the same merry-go-round with Susan: another involuntary commitment, another precipitous release with the "help" of a patient advocate, more self-destructive behavior, cries for help, and chaotic visits home.

We do not know if she will ever accept help or have a viable life. Yet, not knowing, we must somehow get on with our own lives. We do this for ourselves, for each other, and for our son. As it is, he will carry an extra responsibility for the rest of his life and we do not want to add to it. For my healthy son, who has had only the usual amount of human pain, and for my daughter, who lives in almost constant pain, I feel I must go on living as well as I can. One must search for choices and even pleasures if only on the margins and borders of the most pervasive tragedy. As I write, I realize I have been saying this to myself as well and have done so since as a child I witnessed my father's anguish about his schizophrenic brother and son.

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Bibliography on Schizophrenia

The bibliography that appears below is drawn from the Mental Health Abstracts data base, produced by IFI/Plenum Data Company, 3202 Kirkwood Highway, Wilmington, DE 19808. The titles were selected for inclusion here on the basis of their direct relevance to schizophrenia. Judgments about the quality of the article or book have been left to the reader. Due to space limitations, the *Schizophrenia Bulletin* has temporarily discontinued publishing the abstracts themselves; however, we hope to reinstitute this portion in the near future.

The articles listed below are not available from the Schizophrenia Research Branch, but are available for access through Dialog Information Retrieval Systems in reference departments of major university libraries and public libraries.

Attention, Perception, Cognition

6887. **Asarnow, R.F., and MacCrimmon, D.J.** (Division of Child Psychiatry/Mental Retardation, Dept. of Psychiatry, University of California at Los Angeles, Los Angeles, CA 90024) Attention/information processing, neuropsychological functioning, and thought disorder during the acute and partial recovery phases of schizophrenia: A longitudinal study. *Psychiatry Research*, 7:309–319, 1982.

6888. **Calev, A., and Monk, A.F.** (No address given) Verbal memory tasks showing no deficit in schizophrenia—Fact or artifact? *British Journal of Psychiatry*, 141:528–530, 1982.

6889. **Carter, L.; Alpert, M.; and Stewart, S.M.** (New York University School of Medicine, New York, NY 10016) Schizophrenic children's utilization of images and words in performance of cognitive tasks. *Journal of Autism and Developmental Disorders*, 12:279–293, 1982.

6890. **Frith, C.D.; Crow, T.J.; Johnstone, E.C.; Owens, D.G.C.; and Stevens, M.** (Division of Psychiatry, MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex, HA1 3UJ, England) Integration of schematic faces and other complex objects in schizophrenia. *Journal of Nervous and Mental Disease*, 171:34–39, 1983.

6891. **Harvey, P.D.** (No address given) Speech competence in manic and schizophrenic psychoses: The association between clinically rated thought disorder and cohesion and reference performance. *Journal of Abnormal Psychology*, 92:368–377, 1983.

6892. **Iacono, W., and Koenig, W.G.R.** (Dept. of Psychology, University of British Columbia, Vancouver, British Columbia, Canada) Features that distinguish the smooth-pursuit eye-tracking performance of schizophrenic, affective-disorder, and normal individuals. *Journal of Abnormal Psychology*, 92:29–40, 1983.

6893. **Levin, S.; Holzman, P.S.; Jones, A.; Merrin, E.L.; and Stark, L.** (Dept. of Physiological Optics, Neurology Laboratory, University of California, Berkeley, CA 94720) Identification of abnormal patterns in eye movements of schizophrenic patients. *Archives of General Psychiatry*, 39:1125–1130, 1982.

6894. **Levin, S.; Holzman, P.S.; Jones, A.; Merrin, E.L.; and Stark, L.** (Dept. of Physiological Optics, Neurology Laboratory, University of California, Berkeley, CA 94720) Saccadic eye movements of schizophrenic patients measured by reflected light technique. *Biological Psychiatry*, 17:1277–1287, 1982.

6895. **Magaro, P.A., and Page, J.** (Ohio State University, Columbus, OH 43210) Brain disconnection, schizophrenia, and paranoia. *Journal of Nervous and Mental Disease,* 171:133–139, 1983.

6896. **Pishkin, V., and Williams, W.V.** (University of Oklahoma, Norman, OK 73069) Cognitive deficit in schizophrenia subvocal mediation, rigidity, and complexity parameters. *Journal of Nervous and Mental Disease,* 171:24–29, 1983.

6897. **Silverstein, M.L., and Harrow, M.** (University of Chicago, Chicago, IL 60637) Goal-directed thinking in schizophrenics' associations. *Journal of Personality Assessment,* 47:161–166, 1983.

6898. **Stuss, D.T.; Benson, D.F.; Ferrill, D.; Kaplan, E.F.; Liberman, I.; Naeser, M.A.; and Weir, W.S.** (University of Ottawa, Dept. of Medicine, Ottawa General Hospital, 501 Smyth Rd., Ottawa, Ontario, Canada K1H 8L6) The involvement of orbitofrontal cerebrum in cognitive tasks. *Neuropsychologia,* 21:235–248, 1983.

6899. **Walker, E., and Green, M.** (Cornell University, Ithaca, NY 14853) Motor proficiency and attentional-task performance by psychotic patients. *Journal of Abnormal Psychology,* 91:261–268, 1982.

6900. **Wolf, M.E.; Mosnaim, A.D.; and Ryan, J.J.** (Veterans Administration Medical Center, North Chicago, IL 60612) Cognitive functions in tardive dyskinesia. *Psychological Medicine,* 13:671–674, 1983.

Biology

6901. **Alfredsson, G.; Lindberg, M.; and Sedvall, G.** (Karolinska Hospital, Stockholm, Sweden) The presence of 7-hydroxychlorpromazine in CSF of chlorpromazine-treated patients. *Psychopharmacology,* 77:376–378, 1982.

6902. **Arato, M.; Bagdy, G.; and Perenyi, A.** (National Institute for Nervous and Mental Diseases, Budapest, Hungary) Reduced serum dopamine-beta-hydroxylase activity in paranoid schizophrenia. *Biological Psychiatry,* 17:853–854, 1982.

6903. **Ariel, R.N.; Berg, R.A.; Dirksen, J.W.; Forsell, T.; Golden, C.J.; Graber, B.; Quaife, M.A.; and Wilson, J.** (No address given) Regional cerebral blood flow in schizophrenics tested using the Xenon (Xe 133) inhalation method. *Archives of General Psychiatry,* 40:258–263, 1983.

6904. **Baker, M.F.; Bloxham, C.A.; Crow, T.J.; Kidley, R.M.; Parry, R.P.; and Tyrrell, D.A.J.** (Divisions of Psychiatry and Communicable Diseases, MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex, HA1 3UJ England) An investigation of the effects of intracerebral injection in the marmoset of cytopathic cerebrospinal fluid from patients with schizophrenia or neurological disease. *Psychological Medicine,* 13:499–511, 1983.

6905. **Baron, M.; Gruen, R.; Kane, J.; and Levitt, M.** (New York State Psychiatric Institute, New York, NY 10032) Neuroleptic drugs and erythrocyte catechol-*O*-methyltransferase in schizophrenia. *Biological Psychiatry,* 17:1339–1342, 1982.

6906. **Benson, K.L., and Zarcone, V.P., Jr.** (Dept. of Psychiatry, Palo Alto VA Medical Center, Palo Alto, CA 94304) Middle ear muscle activity during REM sleep in schizophrenic, schizoaffective, and depressed patients. *American Journal of Psychiatry,* 139:1474–1476, 1982.

6907. **Bodis-Wollner, I.; Mylin, L.; Thornton, J.; and Yahr, M.D.** (Mount Sinai School of Medicine, New York, NY 10029) Dopaminergic deficiency and delayed visual evoked potentials in humans. *Annals of Neurology,* 11:478–483, 1982.

6908. **Cleghorn, J.M.; Brown, G.M.; Brown, P.J.; Kaplan, R.D.; and Mitton, J.** (Dept. of Psychiatry, McMaster University, Hamilton, Ontario, Canada) Longitudinal instability of hormone responses in schizophrenia. *Progress in Neuro-psychopharmacology,* 7:545–551, 1983.

6909. **Del Vecchio, M.; D'Ambrosio, A.; Kemali, D.; and Maj, M.** (Clinica Psichiatrica, Prima Facoltá di Medicina e Chirurgia, Universitá di Napoli, Piazza Miraglia 2, 1-80138 Naples, Italy) Low platelet MAO activity in chronic schizophrenics: A long-term effect of neuroleptic treatment? *Psychopharmacology,* 79:177–179, 1983.

6910. **Dewan, N.J.; Boucher, M.L.; Levy, B.F.; Major, L.F.; and Pandurangi, A.K.** (Dept. of Psychiatry, Upstate Medical Center of the State University of New York, Syracuse, NY 13201) Abnormal dexamethasone suppression test results in chronic schizophrenic patients. *American Journal of Psychiatry,* 139:1501–1503, 1982.

6911. **Dudley, J.; Hawes, E.M.; Keegan, D.L.; Midha, K.K.; and Rauw, G.** (Dept. of Psychiatry, University of Saskatchewan, Saskatoon, Canada) Correlation of fluphenazine plasma levels versus clinical response in patients: A pilot study. *Progress in Neuro-psychopharmacology,* 7:791–795, 1983.

6912. **Eotstein, E.; Barone, D.; Mishra, R.K.; and Singal, D.P.** (Hamilton Psychiatric Hospital, Hamilton, Ontario, Canada) Lymphocyte ^{3}H-spiroperiodol binding in schizophrenia: Preliminary findings. *Progress in Neuro-psychopharmacology,* 7:729–732, 1983.

6913. Ferrier, I.N.; Adrian, T.E.; Crow, T.J.; Johnstone, E.C.; Lee, Y.C.; O'Shaughnessy, D.; Owens, D.G.C.; and Roberts, G.W. (Division of Psychiatry, MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex, HA1 3UJ, England) Reduced cholecystokinin-like and somatostatin-like immunoreactivity in limbic lobe is associated with symptoms in schizophrenia. *Life Sciences,* 33:475–482, 1983.

6914. Ferrier, I.N.; Cotes, P.M.; Crow, T.J.; and Johnstone, E.C. (Division of Psychiatry, MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex HA1 3UJ, England) Gonadotrophin secretion abnormalities in chronic schizophrenia. *Psychological Medicine,* 12:263–273, 1982.

6915. Gambill, J.D.; Karabelas, A.N.; Koebele, K.R.; Kornetsky, C.; and Mendes, R.W. (Boston University, 755 Commonwealth Ave., Boston, MA 02215) Skin-testing with neurotransmitters in schizophrenic patients. *Journal of Clinical Psychiatry,* 44:321–325, 1983.

6916. Gruzelier, J., and Manchanda, R. (Dept. of Psychiatry, Charing Cross Hospital Medical School, University of London, London W6 8RP, England) The syndrome of schizophrenia: Relations between electrodermal response, lateral asymmetries and clinical ratings. *British Journal of Psychiatry,* 141:488–495, 1982.

6917. Kafka, M.S., and van Kammen, D.P. (No address given) Alpha-adrenergic receptor function in schizophrenia: Receptor number, cyclic adenosine monophosphate production, adenylate cyclase activity, and effect of drugs. *Archives of General Psychiatry,* 40:264–270, 1983.

6918. Kato, G., and Ban, T. (Biochemical Pharmacology, Battelle-Geneva Research Centres, Geneva, Switzerland) Central nervous system receptors in neuropsychiatric disorders. *Progress in Neuro-psychopharmacology,* 6:207–222, 1982.

6919. Johnstone, E.C.; Bourne, R.C.; Crow, T.J.; Ferrier, I.N.; Frith, C.D.; Gamble, S.J.; and Owens, D.G.C. (Division of Psychiatry, MRC Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex, HA1 3UJ England) Adverse effects of anticholinergic medication on positive schizophrenic symptoms. *Psychological Medicine,* 13:513–527, 1983.

6920. Jorgensen, A.; Andersen, J.; Bjorndal, N.; Dencker, S.J.; Lundin, L.; and Malm, U. (H. Lundbeck and Co., Copenhagen-Valby, Denmark) Serum concentrations of cis(Z)-flupenthixol and prolactin in chronic schizophrenic patients treated with flupenthixol and cis(Z)-flupenthixol decanoate. *Psychopharmacology,* 77:58–65, 1982.

6921. Linnoila, M.; Cowdry, R.W.; Ninan, P.T.; Rubinow, D.R.; Waters, R.N.; and Whorton, R. (National Institute of Mental Health, 9000 Rockville Pike, Bethesda, MD 20892) CSF prostaglandin levels in depressed and schizophrenic patients. *Archives of General Psychiatry,* 40:405–406, 1983.

6922. Lippmann, S.; Baldwin, H.; Manshadi, M.; and Wagemaker, H. (University of Louisville, 2301 S. 3rd St., Louisville, KY 40208) Using pulse rate to assess the severity of psychosis. *Hospital & Community Psychiatry,* 34:739–741, 1983.

6923. Luchins, D.J.; Weinberger, D.R.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, National Institute of Mental Health, St. Elizabeths Hospital, Washington, DC 20032) Schizophrenia and cerebral asymmetry detected by computed tomography. *American Journal of Psychiatry,* 139:753–757, 1982.

6924. Maas, S.; Beckmann, H.; and Emrich, H.M. (Zentralinstitut für Seelische Gesundheit, Mannheim, Federal Republic of Germany) Analgesic and euphoric effects of high dose diazepam in schizophrenia. *Neuropsychobiology,* 8:123–128, 1982.

6925. Mackay, A.V.P.; Arregui, A.; Bird, E.; Creese, I.; Iversen, L.L.; Rossor, M.; Snyder, S.H.; and Spokes, E. (No address given) Increased brain dopamine and dopamine receptors in schizophrenia. *Archives of General Psychiatry,* 39:991–997, 1982.

6926. Mathew, R.J.; Barr, D.L.; Duncan, G.C.; and Weinman, M.L. (Dept. of Psychiatry, Vanderbilt University School of Medicine, Nashville, TN 37232) Regional cerebral blood flow in schizophrenia. *Archives of General Psychiatry,* 39:1121–1124, 1982.

6927. Nasrallah, H.A.; McCalley-Whitters, M.; and Jacoby, C.G. (VA Medical Center, Iowa City, IA 52240) Cortical atrophy in schizophrenia and mania: A comparative CT study. *Journal of Clinical Psychiatry,* 43:439–441, 1982.

6928. Nielsen, E.; Ellison, G.; and Lyon, M. (No address given) Apparent hallucinations in monkeys during around-the-clock amphetamine for seven to fourteen days: Possible relevance to amphetamine psychosis. *Journal of Nervous and Mental Disease,* 171:222–232, 1983.

6929. Pickar, D.; Cohen, M.R.; Cohen, R.M.; and Naber, D. (Clinical Neuroscience Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD 20892) Clinical studies of the endogenous opioid system. *Biological Psychiatry*, 17:1243–1276, 1982.

6930. Piran, N.; Bigler, E.D.; and Cohen, D. (Dept. of Psychology, University of Texas, Austin, TX 78712) Motoric laterality and eye dominance suggest unique pattern of cerebral organization in schizophrenia. *Archives of General Psychiatry*, 39:1006–1010, 1982.

6931. Potkin, S.G.; Agarwal, R.P.; Gillin, J.C.; Henkin, R.I.; Shore, D.; Torrey, E.F.; Weinberger, D.R.; and Wyatt, R.J. (Neuropsychiatry Branch, Intramural Research Program, National Institute of Mental Health, Saint Elizabeths Hospital, Washington, DC 20032) Cerebrospinal fluid zinc concentrations in ex-heroin addicts and patients with schizophrenia: Some preliminary observations. *Biological Psychiatry*, 17:1315–1322, 1982.

6932. Potkin, S.G.; Weinberger, D.R.; Linnoila, M.; and Wyatt, R.J. (University of California, Irvine, Medical Center, Dept. of Psychiatry and Human Behavior, 101 City Drive S., Orange, CA 92668) Low CSF 5-hydroxyindoleacetic acid in schizophrenic patients with enlarged cerebral ventricles. *American Journal of Psychiatry*, 140:21–24, 1983.

6933. Rieger, W.; Skolnick, B.E.; Younkin, D.; Gur, R.E.; Caroff, S.; Gur, R.C.; Obrist, W.D.; and Reivich, M. (University of Pennsylvania, Philadelphia, PA 19104) Brain function in psychiatric disorders. *Archives of General Psychiatry*, 40:1250–1254, 1983.

6934. Rippere, V. (Dept. of Psychology, Institute of Psychiatry, DeCrespigny Park, Denmark Hill, London SE5 8AF, England) Schizophrenia and antibodies to food. *Biological Psychiatry*, 17:955–956, 1982.

6935. Rotman, A.; Munitz, H.; Wijsenbeek, H.; and Zemishlany, Z. (Weizmann Institute of Science, Rehovot, Israel) The active uptake of serotonin by platelets of schizophrenic patients and their families: Possibility of a genetic marker. *Psychopharmacology*, 77:171–174, 1982.

6936. Schneider, S.J. (Franklin Delano Roosevelt VA Medical Center, Montrose, NY 10548) Multiple measures of hemispheric dysfunction in schizophrenia and depression. *Psychological Medicine*, 13:287–297, 1983.

6937. Shah, N.S.; Burch, E.A., Jr.; Chestnut, E.; Donald, A.G.; Pressley, L.C.; and Rausch, J.L. (Ensor Foundation Research Laboratory, William S. Hall Psychiatric Institute, Columbia, SC 29203) Platelet uptake of dopamine in patients with schizophrenia. *Research Communications in Psychology, Psychiatry and Behavior*, 7:489–492, 1982.

6938. Shaw, J.C.; Colter, N.; and Resek, G. (Graylingwell Hospital, Chichester, West Sussex, England) EEG coherence, lateral preference and schizophrenia. *Psychological Medicine*, 13:299–306, 1983.

6939. Siever, L.J.; Buchsbaum, M.S.; Coursey, R.D.; Haier, R.J.; Holzman, P.S.; Murphy, D.L.; and Sostek, A.J. (Clinical Neuropharmacology Branch, National Institute of Mental Health, Bldg. 10, Room 3D41, 9000 Rockville Pike, Bethesda, MD 20892) Smooth pursuit eye tracking impairment relation to other "markers" of schizophrenia and psychologic correlates. *Archives of General Psychiatry*, 39:1001–1005, 1982.

6940. Simpson, J.C. (Section of Psychiatry and Human Behavior, Brown University, Providence, RI 02912) Amino acid levels in schizophrenia and celiac disease: Another look. *Biological Psychiatry*, 17:1353–1357, 1982.

6941. Stahl, S.M.; Berger, P.A.; Ciaranello, R.D.; Mefford, I.N.; and Woo, D.J. (No address given) Hyperserotonemia and platelet serotonin uptake and release in schizophrenia and affective disorders. *American Journal of Psychiatry*, 140:26–29, 1983.

6942. Targum, S.D., and Capodanno, A.E. (Psychiatric Institute, 2141 K St., NW, Suite 704, Washington DC 20037) The dexamethasone suppression test in adolescent psychiatric inpatients. *American Journal of Psychiatry*, 140:589–590, 1983.

6943. Tomer, R.; Mintz, M.; and Myslobodsky, M.S. (Tel-Aviv University, Ramat-Aviv 69978, Israel) Left hemisphere hyperactivity in schizophrenia: Abnormality inherent to psychosis or neuroleptic side effects? *Psychopharmacology*, 77:168–170, 1982.

6944. Walker, E., and McGuire, M. (Cornell University, Ithaca, NY 14850) Bilateral processing of visual and auditory stimuli by psychotic patients: A study of the nature and extent of performance asymmetries. *Research Communications in Psychology, Psychiatry, and Behavior*, 7:441–461, 1982.

6945. Yesavage, J.A. (VA Medical Center, Palo Alto, CA 94304) Inpatient violence and the schizophrenic patient: An inverse correlation between danger-related events and neuroleptic levels. *Biological Psychiatry*, 17:1331–1337, 1982.

Case Studies

6946. Awad, A.G. (University of Toronto, Toronto, Ontario, Canada) Schizophrenia and multiple sclerosis. *Journal of Nervous and Mental Disease*, 171:323–324, 1983.

6947. Bark, N.M. (Information Science Division, Rockland Research Institute, Orangeburg, NY 10962) Heatstroke in psychiatric patients: Two cases and a review. *Journal of Clinical Psychiatry*, 43:377–380, 1982.

6948. Campbell, R.; Schaffer, C.B.; and Tupin, J. (University of California, Davis, CA 95606) Catatonia associated with glutethimide withdrawal. *Journal of Clinical Psychiatry*, 44:32–33, 1983.

6949. Fishbain, D.A., and Rotondo, D.J. (University of Miami, Coral Gables, FL 33124) Foreign body ingestion associated with delusional beliefs. *Journal of Nervous and Mental Disease*, 171:321–322, 1983.

6950. Hoffman, B.F. (Clarke Institute of Psychiatry, 250 College St., Toronto M5T 1R8, Ontario, Canada) Reversible neurosyphilis presenting as chronic mania. *Journal of Clinical Psychiatry*, 43:338–339, 1982.

6951. Nash, J.L. (Vanderbilt University School of Medicine, Nashville, TN 37203) Delusions of innocence and antagonistic delusory ideas. *Journal of Clinical Psychiatry*, 43:336–337, 1982.

6952. Read, S.L. (University of California, Los Angeles, CA 90024) Catatonia in thrombotic thrombocytopenic purpura: Case report. *Journal of Clinical Psychiatry*, 44:343–344, 1983.

6953. Roberts, J.K.A.; Robertson, M.; and Trimble, M.R. (Dept. of Psychological Medicine, National Hospital for Nervous Diseases, London, England) Schizophrenic psychosis associated with aqueduct stenosis in adults. *Journal of Neurology, Neurosurgery and Psychiatry*, 46:892–898, 1983.

6954. Uhde, T.W.; Kleber, H.D.; and Redmond, D.E., Jr. (National Institute of Mental Health, 9000 Rockville Pike, Bethesda, MD 20892) Psychosis in the opioid addicted patient: Assessment and treatment. *Journal of Clinical Psychiatry*, 43:240–247, 1982.

Childhood Psychosis

6955. Janes, C.; Weeks, D.; and Worland, J. (Washington University School of Medicine, 4940 Audubon, St. Louis, MO 63110) School behavior in adolescent children of parents with mental disorder. *Journal of Nervous and Mental Disease*, 171:234–239, 1983.

Cross-Cultural Studies

6956. Cooklin, R.S.; Carney, M.W.P.; and Ravindran, A. (Dept. of Mental Health, University College, London, England) The patterns of mental disorder in Jewish and non-Jewish admissions to a district general hospital psychiatric unit: Is manic-depressive illness a typically Jewish disorder? *Psychological Medicine*, 13:209–212, 1983.

6957. Krassoievitch, M.; Perez-Rincon, H.; and Suarez, P. (No address given) Correlations between visual and auditory hallucinations in a population of Mexican schizophrenics. *Confrontations Psychiatriques*, 21:149–162, 1982.

Description

6958. Arana, G.W.; Barreira, P.J.; Cohen, B.M.; Fogelson, D.; and Lipinski, J.F. (McLean Hospital, 115 Mill St., Belmont, MA 02178) The dexamethasone suppression test in psychotic disorders. *American Journal of Psychiatry*, 140:1521–1523, 1983.

6959. Barquero, J.L.V. (No address given) Social class and social mobility as factors in development of mental illness. *Revista de Psiquiatria y Psicologia Medica de Europa y America Latinas*, 15:375–386, 1982.

6960. Bliss, E.; Larson, E.; and Nakashima, S.R. (University of Utah, Salt Lake City, UT 84112) Auditory hallucinations and schizophrenia. *Journal of Nervous and Mental Disease*, 171:30–33, 1983.

6961. Bogdan, A.N. (No address given) Marital status of patients with paranoid schizophrenia of various progression. *Journal of Neuropathology*, 83:1376–1383, 1983.

6962. Briere, J.; Downes, A.; and Spensley, V. (University of Manitoba, Winnipeg, Manitoba, Canada) Summer in the city: Urban weather conditions and psychiatric emergency-room visits. *Journal of Abnormal Psychology*, 92:77–80, 1983.

6963. Cancro, R. (Dept. of Psychiatry, New York University Medical Center, 550 First Ave., New York, NY 10016) Office management of the schizophrenic disorders. *Psychiatric Annals*, 12:583–586, 588–589, 1982.

6964. Carpenter, B.N., and Chapman, L.J. (University of Tulsa, Tulsa, OK 74104) Premorbid status in schizophrenia and abstract, literal, or autistic proverb interpretation.

Journal of Abnormal Psychology, 91:151-156, 1982.

6965. Chaika, E. (Providence College, Providence, RI 02918) A unified explanation for the diverse structural deviations reported for adult schizophrenics with disrupted speech. *Journal of Communication Disorders,* 15:167-189, 1982.

6966. Croughan, J.L.; Miller, J.P.; Wagelin, D.; and Whitman, B.Y. (Washington University School of Medicine, 4940 Audubon, St. Louis, MO 63110) Psychiatric illness in male and female narcotic addicts. *Journal of Clinical Psychiatry,* 43:225-228, 1982.

6967. Crow, T.J. (Division of Psychiatry, MRC Clinical Research Center, Northwick Park Hospital, Harrow, Middlesex HA1 3UJ, England) Two syndromes in schizophrenia? *Trends in Neurosciences,* 5:351-354, 1982.

6968. Dougher, M.J. (University of New Mexico, Albuquerque, NM 87106) Clinical effects of response deprivation and response satiation procedures. *Behavior Therapy;* 14:286-298, 1983.

6969. Friedman, S.; David, O.J.; Kesselman, M.; and Margolis, R. (Downstate Medical Center, Brooklyn, NY 11203) Predicting psychiatric admission from an emergency room: Psychiatric, psychosocial, and methodological factors. *Journal of Nervous and Mental Disease,* 171:155-157, 1983.

6970. Friedman, R.C.; Aronoff, M.S.; Clarkin, J.; Corn, R.; and Hurt, S.W. (Dept. of Psychiatry, New York Hospital/Cornell Medical Center, New York, NY 10021) Sexual histories and premenstrual affective syndrome in psychiatric inpatients. *American Journal of Psychiatry,* 159:1484-1486, 1982.

6971. Galdi, J. (Behavioral Neurology Program, New Hampshire Hospital, Concord, NH 03301) The causality of depression in schizophrenia. *British Journal of Psychiatry,* 142:621-625, 1983.

6972. Greenberg, R.P., and Dattore, P.J. (No address given) Do alexithymic traits predict illness? *Journal of Nervous and Mental Disease,* 171:276-278, 1983.

6973. Herrman, H.E.; Baldwin, J.A.; and Christie, D. (Unit of Clinical Epidemiology, University of Oxford, London, England) A record-linkage study of mortality and general hospital discharge in patients diagnosed as schizophrenic. *Psychological Medicine,* 13:581-593, 1983.

6974. Jackson, J. (No address given) Effects of sublimination of oneness fantasies on manifest pathology in male vs. female schizophrenics. *Journal of Nervous and Mental Disease,* 171:280-288, 1983.

6975. Kornilov, A.P. (No address given) Mental problem-solving in schizophrenia and epilepsy. *Soviet Psychology,* 14:25-41, 1982.

6976. Lebedinskii, V.V. (No address given) The role of asynchronic development in the evolution of psychopathological symptoms in early childhood schizophrenia. *Soviet Neurology and Psychiatry,* 14:3-24, 1982.

6977. Morozova, V.P. (No address given) Some regular features characteristic of the duration of paroxysms and remissions in patients with schizophrenia and manic-depressive psychoses with monopolar depressions. *Journal of Neuropathology,* 83:1358-1366, 1983.

6978. Neugebaver, R. (Columbia University Faculty of Medicine, New York, NY 10027) Reliability of life-event interviews with outpatient schizophrenics. *Archives of General Psychiatry,* 40:378-387, 1983.

6979. Ozerova, N.I. (No address given) A comparative study of the clinical features of the schizophrenic process in parents and children with regard to their sex. *Journal of Neuropathology,* 83:1371-1376, 1983.

6980. Roberts, R.E., and Vernon, S.W. (University of Texas Medical School, P.O. Box 20708, Houston, TX 77025) The Center for Epidemiologic Studies Depression Scale: Its use in a community sample. *American Journal of Psychiatry,* 140:41-46, 1983.

6981. Rotenberg, V.S., and Tsetlin, M.G. (No address given) Clinicophysiological analysis of sleep disorders in patients with borderline states. *Journal of Neuropathology,* 83:1212-1218, 1983.

6982. Roy A. (Clarke Institute of Psychiatry, 250 College St., Toronto M5T 1R8, Ontario, Canada) Risk factors for suicide in psychiatric patients. *Archives of General Psychiatry,* 39:1089-1095, 1982.

6983. Sheinin, L.M. (No address given) Some characteristic features of progressive paroxysmal schizophrenia combined with alcoholism. *Journal of Neuropathology,* 83:1383-1389, 1983.

6984. Summers, F.; Harrow, M.; and Westermeyer, J. (Northwestern University Medical School, Chicago IL 60611) Neurotic symptoms in the postacute phase of schizophrenia. *Journal of Nervous and Mental Disease,* 171:216-220, 1983.

6985. Tolkmitt, F.; Helfrich, H.; Scherer, K.R.; and Standke, R. (No address given) Vocal indicators of psychiatric treatment effects in depressives and schizophrenics.

Journal of Communication Disorders, 15:209–222, 1982.

6986. Tsuang, M.T.; Kronfol, Z.; and Simpson, J.C. (Dept. of Psychiatry, University of Iowa College of Medicine, 500 Newton Rd., Iowa City, IA 52242) Subtypes of drug abuse with psychosis: Demographic characteristics, clinical features, and family history. *Archives of General Psychiatry,* 39:141–147, 1982.

6987. Volovnik, V.M., and Sheinina, N.S. (No address given) Early dysfunctional states in premorbid anamnesis of patients with borderline states. *Journal of Neuropathology,* 83:1205–1211, 1983.

6988. Weiner, H. (St. Elizabeths Hospital, Washington, DC 20032) Histories of response omission and human operant behavior under a fixed-ratio schedule of reinforcement. *Psychiatry Research,* 32:409–434, 1982.

6989. Zigler, E., and Levine, J. (Yale University, 34 Park St., New Haven, CT 06508) Hallucinations vs. delusions: A developmental approach. *Journal of Nervous and Mental Disease,* 171:141–145, 1983.

Diagnosis

6990. Brockington, I.F.; Meltzer, H.Y.; and Perris, C. (Dept. of Psychiatry, University Hospital of South Manchester, West Didsbury, Manchester M20 8LR, England) Cycloid psychoses: Diagnosis and heuristic value. *Journal of Nervous and Mental Disease,* 170:651–656, 1982.

6991. Cleary, P.D.; Goldberg, I.D.; Kessler, L.G.; and Nycz, G.R. (Rutgers University Graduate School of Social Work, New Brunswick, NJ 08903) Screening for mental disorder among primary care patients: Usefulness of the general health questionnaire. *Archives of General Psychiatry,* 39:837–840, 1982.

6992. Egeland, J.A.; Eshleman, S.K.; and Hostetter, A.M. (No address given) Amish study, III: The impact of cultural factors on diagnosis of bipolar illness. *American Journal of Psychiatry,* 140:67–71, 1983.

6993. Gammon, G.D.; John, K.; Mullen, K.; Rothblum, E.D.; Tischler, G.L.; and Weissman, M.M. (Yale University, 34 Park St., New Haven, CT 06508) Use of a structured diagnostic interview to identify bipolar disorder in adolescent inpatients: Frequency and manifestations of the disorder. *American Journal of Psychiatry,* 140:543–547, 1983.

6994. Goldstein, W.N. (Georgetown University School of Medicine Washington, DC 20057) *DSM-III* and the diagnosis of schizophrenia. *American Journal of Psychotherapy,* 37:168–180, 1983.

6995. Klein, D.N. (State University of New York at Buffalo, Buffalo, NY 14214) Activity-withdrawal in the differential diagnosis of schizophrenia and mania. *Journal of Abnormal Psychology,* 91:157–164, 1982.

6996. Lipkowitz, M.H., and Idupuganti, S. (No address given) Diagnosing schizophrenia in 1980: A survey of U.S. psychiatrists. *American Journal of Psychiatry,* 140:52–55, 1983.

6997. Mermelstein, J.J. (Hobart and William Smith Colleges, Geneva, NY 14456) A process approach to the Bender-Gestalt test and its use in differentiating schizophrenic, brain-damaged, and medical patients. *Journal of Clinical Psychology,* 39:173–181, 1983.

6998. Monroe, R.R. (Dept. of Psychiatry, Institute of Psychiatry and Human Behavior, University of Maryland School of Medicine, 645 West Redwood St., Baltimore, MD 21201) *DSM-III* style diagnoses of the episodic disorders. *Journal of Nervous and Mental Disease,* 170:664–669, 1982.

6999. Morice, R.D., and Ingram, J.C.L. (No address given) Language analysis in schizophrenia: Diagnostic implications. *Australian and New Zealand Journal of Psychiatry,* 16:11–21, 1982.

7000. Newmark, C.S.; Gentry, L.; and Whitt, J.K. (Dept. of Psychiatry, University of North Carolina Medical School, Chapel Hill, NC 27514) Utility of MMPI indices of schizophrenia with adolescents. *Journal of Clinical Psychology,* 39:170–171, 1983.

7001. Pope, H.G., Jr. (Harvard University, Cambridge, MA 02115) Distinguishing bipolar disorder from schizophrenia in clinical practice: Guidelines and case reports. *Hospital & Community Psychiatry,* 34:322–328, 1983.

7002. Shagass, C.; Roemer, R.A.; and Straumanis, J.J. (Dept. of Psychiatry, Temple University Medical School, Philadelphia, PA 19122) Relationships between psychiatric diagnosis and some quantitative EEG variables. *Archives of General Psychiatry,* 39:1423–1435, 1982.

7003. Wright, E.C. (St. Lawrence's Hospital, Caterham, Surrey, London, England) The presentation of mental illness in mentally retarded adults. *British Journal of Psychiatry,* 141:496–502, 1982.

7004. Zigmond, A.S., and Sims, A.C.P. (Professorial Psychiatric Unit, St. James University Hospital, Beckett Street, Leeds 9, England) The effect of the use of the International Classification of Diseases 9th revision upon hospital inpatient diagnoses. *British Journal of Psychiatry,* 142:409–413, 1983.

Epidemiology

7005. Craig, T.J. (Information Sciences Division, Rockland Research Institute, Orangeburg, NY 10962) An epidemiologic study of problems associated with violence among psychiatric inpatients. *American Journal of Psychiatry,* 139:1262–1266, 1982.

The Family

7006. El-Islam, M.F. (No address given) Rehabilitation of schizophrenics by the extended family. *Acta Psychiatrica Scandinavica,* 65:112–119, 1982.

7007. McFarlane, W.R. (No address given) *Family Therapy in Schizophrenia.* New York: Guilford Press, 1983. 350 pp.

7008. Miklowitz, D.J.; Falloon, I.R.H.; and Goldstein, M.J. (University of California, Los Angeles, CA 90024) Premorbid and symptomatic characteristics of schizophrenics from families with high and low levels of expressed emotion. *Journal of Abnormal Psychology,* 92:359–367, 1983.

Genetics

7009. Baron, M.; Asnis, L.; Gruen, R.; and Kane, J. (Columbia University, 121 W. 68th St., New York, NY 10023) Familial relatedness of schizophrenia and schizotypal states. *American Journal of Psychiatry,* 140:1437–1442, 1983.

7010. Baron, M.; Asnis, L.; Gruen, R.; and Levitt, M. (New York State Psychiatric Institute, New York, NY 10032) Plasma amine oxidase and genetic vulnerability to schizophrenia. *Archives of General Psychiatry,* 40:275–279, 1983.

7011. Kendler, K.S.; Gruenberg, A.M.; and Strauss, J.S. (Dept. of Psychiatry and Schizophrenia Biological Research Center, Bronx V.A. Medical Center, Mount Sinai School of Medicine, New York, NY 10029) An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia V. *Archives of General Psychiatry,* 39:1257–1261, 1982.

7012. Pope, H.G., Jr.; Cohen, B.M.; Jonas, J.M.; and Lipinski, J.F. (Harvard Medical School, Boston, MA 02115) Failure to find evidence of schizophrenia in first-degree relatives of schizophrenic probands. *American Journal of Psychiatry,* 139:826–828, 1982.

7013. Walker, E., and Shaye, J. (Dept. of Human Development and Family Studies, Cornell University, Ithaca, NY 14850) Familial schizophrenia: A predictor of neuromotor and attentional abnormalities in schizophrenia. *Archives of General Psychiatry,* 39:1153–1156, 1982.

High-Risk Studies

7014. John, R.S.; Mednick, S.A.; and Schulsinger, F. (Social Science Research Institute, University of Southern California, Columbia, CA 29208) Teacher reports as a predictor of schizophrenia and borderline schizophrenia: A Bayesia decision analysis. *Journal of Abnormal Psychology,* 91:399–413, 1982.

7015. Martin, E.M., and Chapman, L.J. (University of Wisconsin, Madison, WI 53706) Communication effectiveness in psychosis-prone college students. *Journal of Abnormal Psychology,* 91:420–425, 1982.

7016. Nuechterlein, K.H. (Neuropsychiatric Institute, 760 Westwood Plaza, Los Angeles, CA 90024) Signal detection in vigilance tasks and behavioral attributes among offspring of schizophrenic mothers and among hyperactive children. *Journal of Abnormal Psychology,* 92:4–25, 1983.

7017. Simons, R.F. (University of Delaware, Newark, DE 19711) Physical anhedonia and future psychopathology: An electrocortical continuity? *Psychophysiology,* 19:433–441, 1982.

Overview

7018. Deutsch, S.I., and Davis, K.L. (Bronx VA Medical Center, 130 W. Kingsbridge Rd., Bronx, NY 10468) Schizophrenia: A review of diagnostic and biological issues I. Diagnosis and prognosis. *Hospital & Community Psychiatry,* 34:313–322, 1983.

7019. Kendler, K.S. (Medical College of Virginia, Richmond, VA 23298) Overview: A current perspective on twin studies of schizophrenia. *American Journal of Psychiatry,* 140:1413–1425, 1983.

Prognosis

7020. Knesevich, J.W.; Clayton, P.J.; and Zalcman, S.J. (Washington University, 4940 Audubon, St. Louis, MO 63130) Six-year followup of patients with carefully diagnosed good- and poor-prognosis schizophrenia. *American Journal of Psychiatry,* 140:1507–1510, 1983.

7021. Linn, M.W.; Caffey, E.M., Jr.; and Klett, C.J. (VA Medical Center, Miami, FL 33125) Relapse of psychiatric patients in foster care. *American Journal of Psychiatry*, 139:778–783, 1982.

7022. Rosen, L.N.; Dunner, D.L.; Fieve, R.R.; and Rosenthal, N. (National Institute of Mental Health, 9000 Rockville Pike, Bethesda, MD 20892) Social outcome compared in psychotic and nonpsychotic bipolar I patients. *Journal of Nervous and Mental Disease*, 171:272–275, 1983.

7023. Shmaonova, L.M.; Kramela Shuili, V.V.; Lieberman, Y.I.; and Lobova, E.K. (No address given) Frequency of acute schizophrenic episodes ("attack") and probability of predictability. *Psychiatric Journal of the University of Ottawa*, 8:5–14, 1983.

7024. Tsuang, M.T. (University of Iowa, 500 Newton Rd., Iowa City, IA 52242) Long-term outcome in schizophrenia. *Trends in Neurosciences*, 5:203–207, 1982.

7025. Watt, D.C.; Katz, K.; and Shepherd, M. (No address given) The natural history of schizophrenia: A 5-year prospective followup of a representative sample of schizophrenics by means of a standardized clinical and social assessment. *Psychological Medicine*, 13:663–670, 1983.

7026. Zubin, J.; Magaziner, J.; and Steinhauer, S.R. (Biometrics Research, VA Medical Center, Pittsburgh, PA 15206) The metamorphosis of schizophrenia: From chronicity to vulnerability. *Psychological Medicine*, 13:551–571, 1983.

Psychological Theory

7027. Bowman, M.B., and Lewis, M.S. (Robert Wood Johnson, Jr. Rehabilitation Institute, John F. Kennedy Medical Center, Edison, NJ 08837) The copper hypothesis of schizophrenia: A review. *Neuroscience and Biobehavioral Reviews*, 6:321–328, 1982.

7028. Grand, S. (No address given) The body and its boundaries: A psychoanalytic view of cognitive process disturbances in schizophrenia. *International Review of Psycho-analysis*, 9:327–342, 1982.

7029. Ruttenberg, H.S. (No address given) Modern psychoanalysis as philosophy. *Modern Psychoanalysis*, 7:7–27, 1982.

Treatment

7030. Astigueta, C.D. (No address given) Integrating psychopharmacology and contemporary psychoanalytic group psychotherapy. *Small Group Behavior*, 14:261–268, 1983.

7031. Hartog, J. (No address given) Schizophreniform psychosis two months after diazepam withdrawal. *Journal of Operational Psychiatry*, 13:41–44, 1982.

7032. Kariya, T.; Itom, H.; Mori, A.; Murasaki, M.; Shimazono, Y.; Sugano, K.; Toru, M.; Yamashita, I. (Dept. of Neuropsychiatry, Faculty of Medicine, Tokyo Medical and Dental University, Tokyo, Japan) A comparison of the clinical effects of timiperone, a new butyrophenone derivative, and haloperidol on schizophrenia using a double-blind technique. *Journal of International Medical Research*, 11:66–77, 1983.

7033. Mandel, M.R.; Severe, J.B.; Schooler, N.R.; Gelenberg, A.J.; and Mieske, M. (Dept. of Psychiatry, Harvard Medical School, Massachusetts General Hospital, Cambridge, MA 02138) Development and prediction of postpsychotic depression in neuroleptic-treated schizophrenics. *Archives of General Psychiatry*, 39:197–203, 1982.

7034. Mann, S.J. (No address given) The integration of psychodrama and family therapy in the treatment of schizophrenia. *Family Therapy*, 9:215–225, 1982.

7035. Mattes, J. (No address given) Cost of evaluation and monitoring of medication. *Hospital & Community Psychiatry*, 34:307–308, 1983.

7036. Neppe, V.M. (Cornell University, Ithaca, NY 14853) Carbamazepine as adjunctive treatment in nonepileptic chronic inpatients with EEG temporal lobe abnormalities. *Journal of Clinical Psychiatry*, 44:326–331, 1983.

7037. Nishikawa, T.; Koga, I.; Tanaka, M.; Tsuda, A.; and Uchida, Y. (Kurume University School of Medicine, Kurume, Japan) Prophylactic effect of neuroleptics in symptom-free schizophrenics. *Psychopharmacology*, 77:301–304, 1982.

7038. Oberkirch, A. (Yale School of Medicine, 34 Park St., New Haven, CT 06510) Personal writings in psychotherapy. *American Journal of Psychotherapy*, 37:265–272, 1983.

7039. Possick, S. (Yale University, 34 Park St., New Haven, CT 06508) The therapist as mentor: An aspect of clinical work with schizophrenic outpatients. *Journal of Nervous and Mental Disease*, 171:314–317, 1983.

7040. Revere, U.L.; Bigelow, L.B.; Dawson, S.D.; and Rodeffer, C.J. (St. Elizabeths Hospital, Washington, DC 20032) Modifying psychotherapeutic techniques to meet the needs of chronic schizophrenics. *Hospital & Community Psychiatry*, 34:361–362, 1983.

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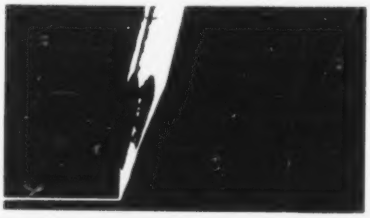

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